Canadian Pediatric Endocrine Group /

Groupe canadien d'endocrinologie pédiatrique

2011

Scientific Meeting

CPEG
GCEP

In cooperation with: UBC

Welcome

Dear Delegates,

Welcome to the 5th Annual Meeting of the Canadian Pediatric Endocrine Group (CPEG). After London, Vancouver, Ottawa and Calgary, we are meeting this year in Toronto.

Your Organizing Committee has been working hard over the last 12 months to prepare a high-quality meeting for pediatric endocrine nurses and endocrinologists that will cover novel concepts in endocrinology and diabetes, highlight the academic contributions of the host institution, and put emphasis on the scientific work by our fellows.

At the time of writing this welcome address, the local Organizing Committee in Toronto is finalizing the social program, an important part of our meeting that helps keep our group a tight one.

Finally, I want to thank our sponsors. Their support makes this important meeting possible.

I wish you a very successful meeting,

Bienvenue

Chers participants,

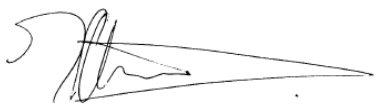
Bienvenue à cette 5 ème édition de la réunion annuelle du groupe canadien d'endocrinologie pédiatrique (GCEP). Après London, Vancouver, Ottawa et Calgary, nous voici cette année à Toronto.

Votre comité organisateur a travaillé dur pendant ces 12 derniers mois à la préparation d'une réunion de grande qualité pour les infirmières en endocrinologie pédiatrique et les endocrinologues pédiatres. Cette réunion couvrira des concepts nouveaux en endocrinologie et en diabète, mettra l'accent sur les contributions académiques de l'institution hôte et soulignera l'importance du travail scientifique de nos fellows.

Au moment d'écrire ce mot de bienvenue, le comité organisateur local à Toronto s'attache à finaliser le programme social qui représente une partie importante de notre réunion en aidant à promouvoir l'amitié au sein de notre groupe.

Pour terminer, je souhaite remercier nos sponsors dont le soutien contribue grandement à l'organisation de cette réunion.

Je vous souhaite une réunion couronnée de succès,



Jean-Pierre Chanoine, M.D.
President, CPEG, 2009-2011

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Fellowship Listing

The Canadian Pediatric Endocrine Group would like to acknowledge and thank the following organizations for their generous support in the form of fellowships:

1992 – 1993	Eli Lilly I: M. Lawson	
1993 – 1994	Eli Lilly I: M. Lawson	Eli Lilly II: A. Simone
	Novo Nordisk: S. Muirhead (Lawrence)	
1994 – 1995	Eli Lilly I: S. Taback	Eli Lilly II: A. Simone
	Novo Nordisk: S. Muirhead (Lawrence)	
1995 – 1996	Eli Lilly I: S. Taback	Eli Lilly II: B. Cummings
	Novo Nordisk: C. Vaz (50% - One Year)	
1996 – 1997	Eli Lilly I: J. Hamilton, E. Sellers	Eli Lilly II: B. Cummings
1997 – 1998	Eli Lilly I: J. Hamilton	Eli Lilly II: E. Sellers
	Serono: B. Cummings	
1998 – 1999	Eli Lilly I: J. Curtis	
	Serono: J. Hamilton	
1999 – 2000	Eli Lilly II: J. Curtis	
	Serono: J. Hamilton	
2000 – 2001	Eli Lilly I: C. Panagiotopoulos	
	Serono: C. Huang	
2001 – 2002	Eli Lilly II: C. Panagiotopoulos	
	Hoffmann La Roche: S. Stock	
2002 – 2003	Eli Lilly I: P. Krishnamoorthy	
	Hoffmann La Roche: R. McEachern	
	Serono: P. Zimakas	
2003 – 2004	Eli Lilly II: P. Krishnamoorthy	
	Hoffman La Roche: H. Bui	
2004 – 2005	Eli Lilly I: M. Nakhla	
	Hoffmann La Roche: J. Simoneau-Roy	
2005 – 2006	Eli Lilly II: M. Nakhla	
	Hoffmann La Roche: M. Jetha	
	Serono: I. Chapados	
2006 – 2007	Eli Lilly I: BA Wicklow	
	Hoffmann La Roche: S. Amed	
2007 – 2008	Eli Lilly II: BA Wicklow	
	Hoffmann La Roche: J. Deladoey	
	Serono: B. Babic, T. Pinto	
2008 – 2009	Eli Lilly I: P. Olivier	
	Hoffmann La Roche: T. Pinto	
	Novo Nordisk: AM Sbrocchi	
2009 – 2010	Eli Lilly II: P. Olivier	
	Hoffmann La Roche: S. Runge-Wildi	
	Novo Nordisk: R. Shulman	
	Sandoz: C. Saaman	
	Serono: T. Édouard	
2010 – 2011	Eli Lilly I: J. Wasserman	
	Hoffmann La Roche: Y. Yeshayahu	
	Novo Nordisk: E. Bassilious	
	Sandoz: S. Tsai	
2011 – 2012	Eli Lilly II: J. Wasserman	

Thursday, February 10, 2011

16:00 Registration Open (Vanity Fair Ballroom Foyer)

17:00 Welcome Reception & Exhibits Open (Palm Court/Pall Mall)

19:00 Adjourn

19:00 Satellite Symposium (please see information in the delegate packages)

Friday, February 11, 2011

07:00 Registration Open (Vanity Fair Ballroom – 2nd Floor)

07:15 Breakfast
(Served in the Vanity Fair Ballroom Foyer & Seating in Vanity Fair Ballroom B)

08:00 Opening Remarks & Welcome (Vanity Fair Ballroom - 2nd Floor)
Dr. Jean-Pierre Chanoine
Dr. Mark Palmert and Ms. Lina Moisan

08:15 CPEG Fellowship Awards
Presented by *Dr. Jean-Pierre Chanoine*

Theme I: Pediatric Pheo and Paraganglioma
Moderator: Dr. Stacy Urbach

08:30 Review of Pediatric Pheochromocytoma and Paraganglioma
Dr. Karel Pacak, Bethesda, MD (Including Q&A)

09:30 Refreshment Break & Exhibits Open
(Served in the exhibit area of Palm Court/Pall Mall foyer)

*10:00 Split Rooms, MD's: Theme II: Endocrine Cancer;
nurses to meet in the Knightsbridge Room, please see page 5 for the program

Theme II: Endocrine Cancer
Moderator: Dr. Guy Van Vliet

10:00 The RET Receptor in MEN 2 and Hirschsprung Disease: From Oncogenesis to Neurogenesis
Dr. Lois Mulligan, Queens, ON (Including Q&A)

10:40 New Insight into Histology and Cell Markers of Thyroid Cancer
Dr. Sylvia Asa, Toronto, ON (Including Q&A)

11:20 Thyroid Cancer Pediatric ATA Consensus Guidelines
Dr. Jill Hamilton, Toronto, ON (Including Q&A)

12:00 Lunch (Windsor Ballroom - Basement)

12:30 Dessert (Served in the exhibit area of Palm Court/Pall Mall)
& Posters (Conference Foyer)

13:30 Abstracts from CPEG Members and Fellow Presentations
(Please see Page 8 for detailed schedule)
Moderators: Dr. Andrea Haqq & Dr. Brandy Wicklow

15:30 Refreshment Break & Exhibits Open
(Served in the exhibit area of Palm Court/Pall Mall foyer)

16:00 Abstracts Continued

17:00 Adjourn

***Nursing Program for February 11th (in the Knightsbridge Room)**
Moderators: Ms. Irena Hozjan, NP-Paeds and Lina Moisan, RN

10:00 Round Table

11:00 AGM

12:00 Lunch (Windsor Ballroom - Basement)

12:30 Dessert (Served in the exhibit area of Palm Court/Pall Mall)
& Posters (Conference Foyer)

13:30 Strategies for the Nursing Care, Management and Support of Adolescents with PCOS
Phaedra Thomas, RN, Boston, MA

14:30 A Child Health Osteoporosis Project Comes to Life. Canadian Ambulatory Nursing Ratio Survey
Nicole Kirouac, RN, Winnipeg, MB

15:00 CPEG/CPEN Portal Update
Robert Preston, Toronto, ON

15:40 Refreshment Break & Exhibits Open
(Served in the exhibit area of Palm Court/Pall Mall foyer)

16:00 Short Stature Social Media Website Initiative
Susan Rybansky, RN, London, ON

16:30 Launch of the Centre for Healthy Active Living at CHEO
Brenda Fraser, RN and Sara Chang, RN, Ottawa, ON

17:00 Adjourn

Friday Night Dinner at Rosewater Room (19 Toronto Street)

19:00 Reception (At the Rosewater Room)

19:30 Dinner

Saturday, February 12, 2011

07:15 Breakfast (Served in the Palm Court/Pall Mall foyer)

08:00 Business Meeting

10:00 Refreshment Break & Exhibits Open
(Served in the exhibit area of Palm Court/Pall Mall foyer)

**10:15 Split Rooms; MD's: Theme III: Diabetes Complications;
nurses to meet in the Knightsbridge Room, please see page 7 for the program

Theme III: Diabetes Complications
Moderator: Dr. Denis Daneman

10:15 Use of Confocal Microscopy for Detection of Diabetic Neuropathy
Dr. Bruce Perkins, Toronto, ON (Including Q&A)

10:55 The Role of Hyperfiltration in Diabetic Nephropathy
Dr. David Cherney, Toronto, ON (Including Q&A)

11:35 Early Intervention for Microalbuminuria: Adolescent Type I Diabetes Cardio-Renal Intervention Trial (AdDIT)
Dr. Farid Mahmud, Toronto, ON (Including Q&A)

12:00 Lunch (Windsor Ballroom - Basement)

12:30 Dessert (Served in the exhibit area of Palm Court/Pall Mall)
& Posters (Conference Foyer)

13:25 Presentation of Dr. John Bailey Resident Research Award
Presented by *Dr. Jean-Pierre Chanoine*

Theme IV: Update on Neonatal Screening
Moderator: Dr. Shayne Taback

13:30 Economics of Newborn Screening: Assessing Costs and Benefits
Dr. Scott D Grosse, Atlanta, GA

14:00 Immunogenetic Prediction of Type 1 Diabetes in the General Pediatric Population
Dr. William A Hagopian, Seattle, WA

14:30 Update for Pediatric Endocrinologists on the Uses of MS/MS in Newborn Screening
Dr. Pranesh Chakraborty, Ottawa, ON

15:00 Panel Discussion & Questions

15:15 Refreshment Break & Exhibits Open
(Served in the exhibit area of Palm Court/Palm Mall foyer)

Theme V: Growth Curves
Moderator: Dr. Jean-Pierre Chanoine

15:35	Raising the Bar: A New Paradigm for Growth Standards for Infants and Young Children *Dr. Cutberto Garza, Chestnut Hill, MA*
16:05	Field Testing of the World Health Organization Growth Standards From Birth To 2 Years in Term and Preterm Infants: Assessment of Hospital Undernutrition and Overnutrition Rates, Usefulness of BMI and Association with Neurodevelopment *Dr. Deborah O'Connor, Toronto, ON*
16:25	WHO Growth Curves - A Clinician's Perspective *Dr. Elizabeth Cummings, Halifax, NS*
16:45	Panel Discussion and Questions
17:05	Closing Remarks & Evaluation Presented by *Dr. Jean-Pierre Chanoine*
17:15	Adjourn

****Nursing Program for February 12th (in the Knightsbridge Room)**
Moderators: Irena Hozjan, NP-Paeds and Lina Moisan, RN

10:15	When Chicken Soup Isn't Enough: How Assertive Nursing Saves Patients *Suzanne Gordon, Arlington, MA*
11:15	Closer to Home - BC Children's Hospital Endocrine Outreach Program *Susan Murphy, RN, Vancouver, BC*
11:45	ART's and TART's in CAH *Irena Hozjan, NP-Paeds, Toronto, ON*
12:00	Lunch (Windsor Ballroom - Basement)
12:30	Dessert (Served in the exhibit area of Palm Court/Pall Mall) & Posters (Conference Foyer)

Fellow Presentation Schedule: Friday, February 11, 2011

Time	Title	Oral Abstract Number	Page
13:30	Vitamin D Status in a Subgroup of Adolescents with Type 1 Diabetes	1	23
13:45	Identifying Hypertension in Children and Adolescents with Type 1 Diabetes: A Cross-Sectional Study	2	24
14:00	The Association Between Physical Activity, Fitness and Insulin Sensitivity in a Cohort of School-Aged Children with an Obese Parent	3	25
14:15	Cardiac Function During Exercise in Adolescents with Type 2 Diabetes (T2DM)	4	26
14:30	Adrenal Suppression and Fat Replacement of the Right Ventricle Myocardium in a Severely Obese Adolescent Girl	5	27
14:45	Prevalence and Function of p53 Mutations Among Children with Adrenocortical Carcinoma	6	28
15:00	Unusual Presentation of Combined Pheochromocytoma and Paraganglioma in a Teenage Girl	7	29
15:15	A Novel GermlineCDC73 (HRPT2) Mutation in An Adolescent with Atypical Parathyroid Adenoma: A Case Report	8	30
16:00	A Retrospective Review of Pituitary MRI Findings in Pediatric Patients on Growth Hormone Therapy	9	31
16:15	Can Recovery from Hypothalamic-Pituitary-Adrenal (HPA) Axis Suppression Following Supraphysiological Doses of Glucocorticoids be Predicted?	10	32
16:30	Expression of Lin28a and Lin28b Decreases Across the Pubertal Transition	11	33
16:45	Do Epigenetic Mechanisms Regulate the Timing of Puberty? Initial Evidence from Studies in Female Mice	12	34

Poster Abstract Listing

Title	Abstract Number	Page
A Case of Autoimmune Hyperthyroidism in a Child with End-stage Renal Disease	1	35
When is a pheo not a pheo? Depression in an adolescent leading to a pheocromocytoma-like biochemical profile	2	36
Suspected Antacid-Induced Rickets and Multiple Fractures in a Child with Rubinstein-Taybi Syndrome	3	37
Clitoromegaly in the Extremely Premature Infant – A Case Series of Four Infants	4	38
Relationship between body mass index and metabolic parameters in obese adolescents and birth size	5	39
Lowering thyrotropin cutoff for newborn screening: Additional cases of congenital hypothyroidism are identified, but what do they really have?	6	40

Program Organizing and Scientific Committee

Robert Barnes
Jean-Pierre Chanoine
Andrea Haqq
Irena Hozjan
Danièle Pacaud
Mark Palmert
Wendy Schwarz
Elizabeth Sellers
Jonathan Wasserman
Diane Wherrett

Dr. John Bailey Resident Research Award

The John Bailey Award is named after Dr. John D. Bailey. Dr. Bailey graduated from the University of Toronto Medical School in 1949, did his training in pediatrics at the Hospital for Sick Children, Toronto and the University of Manitoba in Winnipeg. By the mid-1950s he was firmly established as a major contributor to pediatric care and education at the Hospital for Sick Children and University of Toronto. His major area of interest was in understanding normal and abnormal growth patterns in childhood and adolescence. He was the first chief of the Endocrine Division at the Hospital for Sick Children and an important contributor to the development of the Canadian national growth hormone treatment program. He was the consummate academic: clinically fastidious, intellectually challenging and constantly learning and sharing.

Credits

This Conference has been approved for a maximum of **12 credit hours** under Section 1 of the Maintenance of Certification (MOC) program of the Royal College of Physicians and Surgeons of Canada

Learning Objectives

The overall learning objective of this meeting is to present the current state of knowledge of topics in pediatric endocrinology and diabetes.

Session Learning Objectives:

Review of Pediatric Pheochromocytoma and Paraganglioma
Karel Pacak, MD, PhD, DSc, Senior Investigator, Chief, Professor of Medicine, Section on Medical Neuroendocrinology, NICHD, NIH, Bethesda, MD

1. *To learn about new genetic approaches*
2. *To learn about new biochemical and imagery approaches*
3. *To learn about new/future therapeutic options*

The RET Receptor in MEN 2 and Hirschsprung Disease: From Oncogenesis to Neurogenesis
Lois Mulligan, Professor, Department of Pathology and Molecular Medicine, Division of Cancer Biology and Genetics, Cancer Research Institute at Queen's University, Kingston, ON

1. *To become familiar with the role of the RET proto-oncogene in both cancer and developmental disorders*
2. *To recognize the phenotypes and risks found in families with different types of RET mutations*
3. *To appreciate the diversity of phenotype and molecular effects associated with RET mutations and their genetic and clinical implications*

New Insight into Histology and Cell Markers of Thyroid Cancer
Sylvia Asa, PhD, MD, Pathologist-in-Chief and Medical Director, Laboratory medicine Program, University Health Network; Senior Scientist, Ontario Cancer Institute; Professor, Laboratory Medicine and Pathobiology, Univeresity of Toronto, Toronto, ON

1. *To understand the molecular and epigenetic changes that underlie thyroid cancer*
2. *To describe the value and limitations of diagnostic testing in the management of patients with thyroid cancer*
3. *To understand the clinicopathologic features that are important in determining therapeutic approaches to thyroid cancer*

Thyroid Cancer Pediatric ATA Consensus Guidelines
Jill Hamilton, MD, FRCPC, Staff Endocrinologist, Associate Professor, Department of Paediatrics, Hospital for Sick Children,, University of Toronto, Toronto, ON

1. *To understand the differences between adults and children in presentation and natural history of differentiated thyroid cancer*
2. *To review the ATA recommendations for approach to diagnosis, treatment and follow up of pediatric thyroid nodules*
3. *To review the ATA recommendations for approach to diagnosis, treatment and follow up of pediatric differentiated thyroid cancer*

Strategies for the Nursing Care, Management and Support of Adolescents with PCOS
Phaedra Thomas, RN, BSN, Co-Director and Nurse Educator, Center for Young Women's Health, Children's Hospital Boston, Boston, MA

1. *To identify 2 strategies that will help you care for your adolescent patient with PCOS*
2. *To describe various interventions and tools to help your adolescent patient manage her PCOS symptoms*
3. *To understand the concerns of adolescents with PCOS*

Use of Corneal Confocal Microscopy for Detection of Diabetic Neuropathy
Bruce A Perkins, MD, MPH, Endocrinology and Metabolism, University Health Network (Toronto General Hospital); Assistant Professor, University of Toronto, Toronto, ON

1. *To explain limitations in the clinical diagnosis of diabetic sensorimotor polyneuropathy*
2. *To describe the concepts of concurrent and predictive validity for diagnostic studies.*
3. *To appreciate the potential role of corneal nerve fiber length as a diagnostic tool for diabetic sensorimotor polyneuropathy.*

The Role of Hyperfiltration in Diabetic Nephropathy
David Cherney, MD, Assistant Professor of Medicine, University of Toronto; Clinician Scientist, University Health Network, Toronto, ON

1. *To review the physiologic mechanisms leading to hyperfiltration*
2. *To understand systemic vascular associations in hyperfiltering humans*
3. *To review pharmacologic agents that reduce the hyperfiltration state*

When Chicken Soup Isn't Enough for Nurses or Patients
Suzanne Gordon, Journalist, Author, Editor, The Culture and Politics of Health Care Work Series, Cornell University Press

1. *To understand the misuses of altruism and self-sacrifice embedded in many of the "inspirational models" served up to them.*
2. *To articulate the historical rationale for these models*
3. *To craft alternative messages and strategies to advance their professional needs and quality patient care*

Economics of Newborn Screening: Assessing Costs and Benefits
Scott D Grosse, PhD, Research Economist and Associate Director for Health Services Research and Evaluation, Division of Blood Disorders, National Center on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention, Atlanta, GA

1. *To understand how economic evaluation methods can be used to assess the value of screening and the efficiency of alternative screening strategies*
2. *To list the types of data needed to assess health outcomes attributable to newborn screening, early detection and treatment*
3. *To identify the key uncertainties involved in assessing the cost-effectiveness of newborn screening for congenital adrenal hyperplasia*

Immunogenetic Prediction of Type 1 Diabetes in the General Pediatric Population
William Hagopian, MD PhD, Scientific Director, Pacific Northwest Diabetes Research Institute; Clinical Associate Professor of Medicine, University of Washington, Seattle, WA

1. *To discuss that genetic risk of diabetes resides primarily in HLA Class II genotypes*
2. *To discuss that prediction is most cost effective as a 2-step process starting with HLA screening then islet antibody testing*
3. *To discuss the risk assessment and possible clinical applications of immunogenetic testing*

Raising the Bar: A New Paradigm for Growth Standards for Infants and Young Children
Cutberto Garza, MD, PhD, Provost and Dean of Faculties, Boston College, Chestnut Hill, MA

1. *To review the basis for and conceptual framework underpinning the WHO growth standards for infants and children*
2. *To review highlights of the new international growth standards, with a special focus on growth velocity*
3. *To consider next steps for enhancing the clinical utility of growth standards and references for infants and young children*

Field Testing of the World Health Organization Growth Standards From Birth To 2 Years in Term and Preterm Infants: Assessment of Hospital Undernutrition and Overnutrition Rates, Usefulness of BMI and Association with Neurodevelopment

Deborah O'Connor, PhD, RD, Professor, Department of Nutritional Sciences, Faculty of Medicine, University of Toronto; Director of Clinical Dietetics and Senior Associate Scientist, Physiology and Experimental Medicine Program, The Hospital for Sick Children, Toronto, ON

1. *To understand at the end of this talk if and how the WHO growth standard (WHO-GS) differ from the Centers for Disease Control and Prevention reference growth charts (CDC-RGC).*
2. *To appreciated that assessments based on the WHO BMI-for-age percentiles rather than weight-for-length percentiles, results in a significant change in the number of children younger than 2 years identified as at risk of undernutrition or overnutrition.*
3. *To examine the associations between the pattern of growth of very low birthweight VLBW preterm infants during the first 2 years of life, as assessed by the WHO-GS and the CDC-RGC, and neurodevelopment at 18-24 months corrected age (CA)*

WHO Growth Curves – A Clinician's Perspective
Beth Cummings, MD, FRCPC, Division Head, Pediatric Endocrinology, IWK Health Centre; Associate Professor, Department of Pediatrics, Dalhousie University, Halifax, NS

4. *To be aware of the new WHO growth charts and their developments*
5. *To know the differences between and implications for clinical practice and teaching of available growth charts*
6. *To foster discussion of the position that CPEG may/should take regarding the WHO growth charts*

Biographies

Dr. Sylvia L. Asa

Sylvia Asa is the Pathologist-in-Chief and Medical Director of the Laboratory Medicine Program at the University Health Network and Professor in the Department of Laboratory Medicine and Pathobiology at the University of Toronto. A Clinician-Scientist with a focus on Endocrine Pathology, her research aims to identify the basis for development of endocrine tumors, to improve diagnostic tests and to identify targets for therapy of those diseases. She has published more than 300 scientific articles, written four books, co-edited three books and written more than 50 book chapters. Her work ranges from clinical observation to fundamental research studies, and her publications have achieved a citation impact in the top 1% of scientists. She is an invited speaker at many major international meetings.

Dr. Asa has received many honors, including the Arthur Purdy Stout Society of Surgical Pathologists Award (1998), the Novartis Canada Senior Scientist Award (2001), the Professor C.F.A. Culling Memorial Lecture Award of the National Society for Histotechnology (2004), the ICRF Woman of Action Award (2009), the William Boyd Lecture Award of the Canadian Association of Pathologists (2010) and several awards from the University of Toronto. She has served as President of the Endocrine Pathology Society (1997–1998) and the US-Canadian Academy of Pathology (2005-6).

As head of the largest pathology department in Canada, Dr. Asa has made innovative changes to the practice of the discipline, with emphasis on subspecialization, automation, electronic initiatives and telepathology. The department focuses on education and research to understand mechanisms of disease and translate new information into diagnostic and prognostic information for patient care. To ensure public knowledge of the role of Pathology and maintain a direct connection with patients, Dr. Asa is a consultant to several patient groups.

Dr. Pranesh Chakraborty

Dr Chakraborty is a Pediatrician, Medical Biochemist and Biochemical Geneticist serving as the Director of the Ontario Newborn Screening Program and co-Medical Director of BORN Ontario while also maintaining a clinical practice as a Metabolic Physician and Pediatrician at the Children's Hospital of Eastern Ontario (CHEO). He is active academically and is an Assistant Professor of Pediatrics and Pathology & Laboratory Medicine at the University of Ottawa with a cross appointment to the Department of Biochemistry, Microbiology and Immunology.

Sara Chang

Sara Chang graduated from McMaster University in 2003 with a BScN. She began nursing at Toronto's Hospital for Sick Children in general pediatrics and went on to case management in its metabolic genetics clinic. After moving to Ottawa she worked in pediatric palliative care at Roger's House, and joined the endocrine team at the Children's Hospital of Eastern Ontario in 2009.

Dr. David Cherney

After completing medical school at McGill University, Dr. Cherney was trained in internal medicine and clinical nephrology at the University of Toronto. Following his clinical training, Dr. Cherney completed his PhD in human renal physiology at the Institute of Medical Science, University of Toronto under the supervision of Dr. Judith Miller. He is currently an Assistant Professor in the Department of Medicine, University of Toronto and a Clinician Scientist in the Division of Nephrology, University Healthy Network, and is Director of the Human Physiology Laboratory. Dr. Cherney has expertise in renal hemodynamic function and endothelial function testing, arterial stiffness studies, and biomarkers in skin and urine. His current research interests in type 1 diabetes mellitus include the physiology of renal hyperfiltration in diabetic nephropathy, cardiorenal interactions and endothelial function, the effect of pharmaceutical agents on the urinary proteome, and functional gene polymorphisms in humans.

Dr. Elizabeth Cummings

Dr. Beth Cummings is a pediatric endocrinologist at the IWK Health Centre and Associate Professor of Pediatrics, Dalhousie University. She began at Dalhousie in January of 1999 and is currently the division head. She obtained her medical degree at the University of Western Ontario and did her specialty training at Dalhousie University and the University of Toronto. She has a busy clinical practice caring for children and adolescents with diabetes and endocrine problems. She is also a very involved in teaching and in 2010 was awarded the Teacher of the Year Award by the PARI-Maritime Provinces for excellence in teaching. She is involved in research in type 1 diabetes, particularly in prevention of type 1 diabetes in at risk individuals and in bone health in chronic pediatric disease. She is also a medical advisor to the Diabetes Care Program of Nova Scotia. She is married and has 2 school aged children. She is a member of the Board of Directors of World Vision Canada.

Brenda Fraser

Brenda is a pediatric endocrinology nursing case manager at the Children's Hospital Of Eastern Ontario (CHEO). She received her diploma in Nursing in 1989 and completed her degree in 2007 at Charles Sturt University in Australia. She has been at CHEO since 1994, her experience includes a case management position in the Neurooncology service and outpatient Oncology clinic, staff nursing positions with the IV team, and the Emergency dept. She joined the Division of Endocrinology in 2006.

Suzanne Gordon

Suzanne Gordon is an award-winning journalist and author. She has written for the New York Times, the Los Angeles Times, the Washington Post, the Altantic Monthly, the American Prospect, the Globe and Mail, the Toronto Star, and others.

She is the author and editor of 13 books including *Life Support: Three Nurses on the Front Lines; Nursing Against the Odds: How Health Care Cost Cutting, Media Stereotypes, and Medical Hubris Undermine Nurses and Patient Care;* and *Complexities of Care: Nursing Reconsidered*. With Bernice Buresh, she authored *From Silence to Voice: What Nurses Know and Must Communicate to the Public.*

Her latest book, which she edited, is called *When Chicken Soup Isn't Enough: Stories of Nurses Standing Up for Themselves, Their Patients and Their Profession*. It contains stories by over 70 nurses from all over the world and has just been published by Cornell University Press' culture and Politics of Health Care Work series, which Ms. Gordon edits with Sioban Nelson.

She is also co-author, with playwright Lisa Hayes, of the play about doctor/nurse relationships entitled "Bedside Manners". Hayes and Gordon have just completed a new play about nurse-nurse relationships entitled, "No, We Don't Have to Eat Our Young".

Ms. Gordon has lectured on health care and nursing across the world and is currently working on a book about how the aviation industry became safer and what health care can learn from its recent efforts to create genuine teamwork among all levels of workers.

Dr. Cutberto Garza

Cutberto Garza joined Boston College in 2005 where he serves as Provost and Dean of Faculties. Previous to 2005 he held the rank of full professor at Baylor College of Medicine and Cornell University. He received his BS from Baylor University, his MD from Baylor College of Medicine, and a PhD in nutrition and food science from MIT. Dr. Garza is a specialist in pediatric nutrition and has worked on projects sponsored by the United Nations University, World Health Organization, UNICEF, and other international and national organizations with interests in infant and young child health. He serves as chair of the WHO Steering Committee that developed the new WHO growth standards for infants and young children. He is a member of the Institute of Medicine and was named to the inaugural class of the National Associates of the National Academies of Science. He also is a member of the American Society of Clinical Nutrition, the Society for Pediatric Research and the American Pediatric Society, among other organizations.

Dr. Scott Grosse

Scott Grosse is Health Economist and Associate Director for Health Services Research and Evaluation in the Division of Blood Disorders, National Center on Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention (CDC) in Atlanta, Georgia, which is part of the US Department of Health and Human Services. Dr. Grosse has published widely on health care costs and outcomes associated with a range of pediatric conditions, including autism, cerebral palsy, congenital heart defects, congenital hearing loss, congenital hypothyroidism, cystic fibrosis, Down syndrome, fetal alcohol syndrome, muscular dystrophy, orofacial clefts, sickle cell disease, and spina bifida. He has also conducted research on the impacts of pediatric chronic conditions on the employment and health-related quality of life of parental caregivers. He has published cost-effectiveness and policy analyses on folic acid fortification and supplementation, newborn hearing screening, newborn blood spot screening for diseases such as congenital adrenal hyperplasia and cystic fibrosis, and newborn pulse oximetry evaluation for congenital cyanotic heart disease. He led a 2004 evidence review which contributed to the subsequent US adoption of CF newborn screening. He has degrees in economics and public health from the University of Michigan and joined the CDC in 1996.

Dr. Jill Hamilton

Jill Hamilton (MD, MSc, FRCPC) is a Pediatric Endocrinologist at the Hospital for Sick Children, Associate Scientist at the Research Institute, and Associate Professor of Paediatrics at University of Toronto. Her clinical research interests are in the areas of (i) obesity, insulin resistance and pancreatic beta cell function in childhood and (ii) management of endocrine cancers including differentiated thyroid cancer.

Dr. William Hagopian

William Hagopian is the Scientific Director at the Pacific Northwest Diabetes Research Institute. He completed both medical and research doctorates at the University of Chicago. He then completed Internal Medicine residency and Endocrinology Fellowship at the University of Washington, where he remains Clinical Associate Professor of Medicine treating inpatients and outpatients. Prior to coming to PNDRI, Dr. Hagopian was on fulltime faculty at the University of Washington. He serves as Chair of various scientific and safety committees and boards, and on several grant review committees at the National Institutes of Health. He has published 65 manuscripts and reviews.

Dr. Hagopian's research focuses on strategies in immunology and molecular biology for predicting and preventing type 1 diabetes before clinical disease onset. These studies include screening over 140,000 Washington State newborns to a) determine what environmental factors trigger the disease in those with genetic susceptibility and b) develop and implement cost-effective strategies for population-based pediatric screening for preclinical diabetes prediction. Finally, Dr. Hagopian pursues several clinical trials to find low-toxicity immunotherapies to interrupt the inappropriate autoimmune response that attacks and kills the insulin-producing pancreatic islet cells. In addition to their potential to prevent diabetes before the clinical onset, these studies show promise for newly diagnosed patients to preserve the remaining insulin producing cells, enabling better management of blood sugar.

Irena Hozjan

Irena Hozjan is a Pediatric Nurse Practitioner in the ambulatory Endocrinology Clinic at SickKids. She completed her Master of Nursing Acute Care Nurse Practitioner Program in 2004 at the University of Toronto, and has been a member of the Endocrinology department for 6 years. She has held various nursing and nursing leadership roles, at SickKids, for over 21 years.

Irena's clinical practice includes providing ambulatory health care to individuals and families dealing with Turner Syndrome, precocious puberty and growth hormone deficiency as well as a variety of other endocrine issues and conditions. She is one of the editors, chapter authors and chapter co-authors of the Turner Syndrome: Across the Lifespan book published in 2008. A French language translation of this book was published in 2009.

Nicole Kirouac

Nicole is a Registered Nurse with a Bachelor's Degree in Nursing from the University of Manitoba from 1995. Nicole has been practicing as the Pediatric Endocrine Nurse Clinician at the Children's Hospital Health Sciences Centre in Winnipeg, Manitoba, Canada since 1998. She has been very active in Pediatric Endocrinology as past-president of the Canadian Pediatric Endocrine Nurses Group, graduate of the first Pediatric Endocrinology Nursing Society Research Academy and past member and chairperson of the PENS Nominating Committee. Nicole attributes some of her success in Pediatric Endocrinology to the PENS preceptor program and to the relationships she has built over the years with fellow nurses like you. Returning from a Maternity leave in the fall of 2009, Nicole was fortunate to be able to step back to work in a part-time temporary position as the Clinical Research Nurse for a Bone Health Project at her centre. In the following presentation she will share with you the fruits of that year and how it can be incorporated into other centres to help identify children at risk for osteoporosis and educate staff and caregivers to prevent fractures in these children. Nicole will also take some time to discuss the results of the Survey of the Canadian Pediatric Endocrine Nurses from 2009 in relation to responsibilities and staff ratios.

Dr. Farid Mahmud

Farid Mahmud is currently a Staff Physician in the Division of Endocrinology, Department of Paediatrics and Assistant Professor at the University of Toronto. Dr. Mahmud received his medical degree at The University of Alberta and completed his training in Paediatric Endocrinology and Metabolism at The Mayo Clinic in Rochester. Dr. Mahmud is a clinical investigator with a special interest in the study of diabetes complications. This includes celiac disease in type 1 diabetes as well as the evaluation of early atherosclerosis in young patients with endocrine conditions who are at high risk of cardiovascular disease. Dr Mahmud is an -investigator in AdDIT (Adolescent Type 1 Diabetes Cardio-Renal Intervention Trial) and CD-DIET (Celiac and Diabetes- Dietary Intervention and Evaluation Trial) as part of the Canadian Clinical Trials Network (CCTN).

Dr. Lois M. Mulligan

Dr. Mulligan is a Professor of Pathology and Molecular Medicine, Queen's University and is a member of the Division of Cancer Biology and Genetics, Queen's Cancer Research Institute. She received her Ph.D. in Medical Genetics from Queen's University in 1986 and went on to Post Doctoral training first at the Ludwig Institute for Cancer Research (Montreal) with Dr. Web Cavenee, and then in the CRC Cancer Genetics Group at Cambridge University with Professor Sir Bruce Ponder. Dr. Mulligan is the Director of the Terry Fox Foundation Training Program in Transdisciplinary Cancer Research in partnership with CIHR, and Chair of the Collaborative Graduate Program in Cancer Research at Queen's University. Dr. Mulligan's research interests lie in exploring the regulation of key signaling pathways involved in both tumorigenesis and normal development. Her lab focuses on the RET receptor tyrosine kinase and its role in neuroendocrine development, and in initiation, progression and spread of multiple cancer types, including thyroid and pancreatic carcinomas. Her work is currently funded by grants from the Cancer Research Society and the Canadian Institutes of Health Research.

Susan Murphy

Susan Murphy graduated from Vancouver General Hospital School of Nursing in 1983 and completed her Bachelor of Science in Nursing degree in 2000. Susan is a registered nurse working in the Endocrine Clinic at BC Children's Hospital. She has worked in Pediatric Endocrinology for 10 years and is currently covering the Endocrine Nurse Clinician role at the hospital. Susan is also a study coordinator for the Growth Hormone studies at BC Children's. Prior to specializing in Pediatrics, Susan worked as a registered nurse in Maternal-Child Health at Lions Gate Hospital in North Vancouver.

In addition to her nursing role, Susan has been a member of "Partners In Care" the Family Advisory Board at BC Children's Hospital for the past 18 years. She was honoured to receive a Lifetime Achievement award from BCCH in 2010 for her work promoting the principles and practices of family centered-care at the hospital. Susan is passionate about family-centered care and has presented at numerous conferences and workshops on the topics of Family Centered Care and Raising Children with Chronic Illness.

Dr. Deborah O'Connor

Deborah O'Connor received her B.A.Sc. in Applied Nutrition at the University of Guelph and her MS and PhD in Nutritional Sciences at the University of Illinois. She received her clinical training at Kingston General Hospital. Dr. O'Connor was an Associate Professor in the Division of Applied Human Nutrition at the University of Guelph, and in the Department of Nutrition at The Ohio State University. She also served as the Group Leader for the Premature Infant Nutrition Research Group at Abbott Nutrition's global research headquarters in Columbus, Ohio. Dr. O'Connor is currently a Full-Professor in the Department of Nutritional Sciences, Faculty of Medicine, at the University of Toronto, and is a Senior Associate Scientist in the Physiology and Experimental Medicine Program at The Hospital for Sick Children. She is also the Director of Clinical Dietetics and Breastfeeding support at the same institution. Her research interests are in the area of maternal and infant nutrition, folate metabolism, and provision of human milk for very low birth weight infants. She is a member of the International Society of Research on Human Milk and Lactation, Coalition for Research in Women's Health and the American Society of Nutrition. She is currently serving on the Expert Advisory Group for revision of the National Nutrition Pregnancy Guidelines by Health Canada and on the Executive for the Canadian Nutrition Society.

Dr. Karel Pacak

Graduated with summa cum laude in 1984 at the Charles University, Czech Republic. In 1990 Dr. Pacak started his postdoctoral fellowship at NIH, Bethesda, USA. In 1995 Dr. Pacak started his residency in internal medicine at the Washington Hospital Center under Dr. L. Wartofsky and then a fellowship in endocrinology, diabetes, and metabolism at NIH. He was Board certified in 1998 and 1999 in all those disciplines. In 1998 he established a new Program for Neuroendocrine Tumors focusing on pheochromocytoma and paraganglioma at NIH. He is an internationally recognized expert in diagnosis and treatment of neuroendocrine tumors, especially pheochromocytoma and paraganglioma. Dr. Pacak also introduced and established International Symposia on Pheochromocytoma. Dr. Pacak also co-founded a new Asian Alliance for the Study of Neuroendocrine Tumors in 2010. He is a recipient of numerous awards including Peter Heimann Memorial Award, International Association of Endocrine Surgeons; NIH Director's Mentor Award, Award for Cure, Pheo & Para Alliance and NICHD Director Award of Merit, Pincus Taft Memorial Lecture Award, Endocrine Society of Australia, and Jessenius Gold Medal, Slovak Academy of Sciences. Dr. Pacak is the author of more than 265 scientific peer review articles, 88 book chapters and 5 books.

Dr. Bruce Perkins

Bruce Perkins, MD MPH is Assistant Professor and Endocrinologist at the University of Toronto's University Health Network who practices general endocrinology and is a Diabetes Complications Clinician-Investigator. He obtained his MD and Internal Medicine Residency training at the University of Toronto. He then obtained his endocrinology subspecialty training at Harvard University and the Joslin Diabetes Center, and his Masters of Public Health in Epidemiology at the Harvard School of Public Health. He is a Canadian Diabetes Association Scholar and has JDRF, CIHR and Banting and Best Diabetes Center funding. His research methods focus on cohort studies and clinical trials in diabetic nephropathy and neuropathy. His particular interests are in the identification of valid early phenotypes of these complications, and the natural history and determinants of the early phenotypes. He has described novel phenotypes: For early nephropathy, he has introduced "early GFR loss" as the alternative to microalbuminuria as a predictive marker, using the serial analysis of serum cystatin C. For early neuropathy, he has developed the operating characteristics of Corneal Confocal Microscopy as a method to analyze early small fiber morphological abnormalities as an alternative to intra-epidermal nerve fiber analysis by skin biopsy. In his clinical practice - and in some of his research - he focuses on technologies in diabetes care such as insulin pumps and continuous glucose monitoring.

Susan Rybansky

Susan Rybansky is an RN who earned her BScN in 1980 from the University of Western Ontario in London. She has been employed at the London Health Sciences center as a nurse clinician in pediatric endocrinology since 1988 or over 20 years. Susan is currently the treasurer for the Canadian pediatric endocrinology nurses group. Her previous experiences include working as a certified diabetes educator, research study coordinator, and public health nurse and fitness instructor.

Phaedra P. Thomas

Phaedra P. Thomas RN, BSN is the Nurse Educator and Co-Director for the Center for Young Women's Health, Children's Hospital Boston where she oversees the daily operation of the Resource Center, Youth Advisory Program, community programs, "*Teen Talk"* newsletter, *TeenSpeak blog, Internet chats* and award winning website: www.youngwomenshealth.org. Ms. Thomas has written many of the website's health guides for teens and parents including a resource book for teens; *"PCOS Resources for a Healthier You"*- a collection of resources for teens with polycystic ovary syndrome and moderates the Center's monthly *PCOS Internet Chat for Teens.*

Additionally, Ms. Thomas has created educational curriculum for both educators and teens on topics such as safety, nutrition, and fitness and authored a chapter in the 5th edition of Pediatric & Adolescent Gynecology- Laufer, Emans & Goldstein on *Education of the Child and Adolescent.* She has presented on topics such as *Empowering Girls about their Health, Counseling Young Women with Reproductive Disorders,* and teaches a yearly workshop on *How to Write Patient Educational Materials to residents and fellows at Harvard Medical School.* She also coordinates two yearly international medical conferences on endometriosis and MRKH for teens and their families. Ms. Thomas is passionate about empowering girls about their health and has been recognized by her colleagues at Children's Hospital Boston, (the primary pediatric teaching hospital for Harvard Medical School) for her leadership and innovations in child health.

Disclosure of Conflict of Interest

All Speakers and Planning Committee members must disclose whether they do or do not have a relationship with a commercial entity such as a pharmaceutical organization, medical device company or a communications firm. Please see below for all of our speakers, and planning committee to see their relationships, if any.

Speakers

Sylvia L Asa
- Is a member of an Advisory Board or equivalent with Cerner Corporation and Aperio Inc. as an unpaid advisor

Pranesh Chakraborty
- No relationship

Sara Chang
- Has received payment from a commercial organization: Eli Lilly

Beth Cummings
- Am currently participating in or have participated in a clinical trial with Eli Lilly Canada and Novantas

David Cherney
- Has received a grant or an honorarium from Boehringer-Ingelheim
- Am currently participating in or have participated in a clinical trial with Boehringer-Ingelheim

Brenda Fraser
- Is a member of an Advisory Board or equivalent with Lilly, EMD Serono and Sandoz
- Has received a grant or an honorarium from EMD Serono

Cutberto Garza
- No relationship

Suzanne Gordon
- No relationship

Scott Grosse
- No relationship

Jill Hamilton
- Am currently participating in or have participated in a clinical trial with EMD Seron and Lilly (Easypod Registry and Genesis Registry)

William Hagopian
- Is a member of an Advisory Board or equivalent with Bayhill Pharma
- Hold a patent for a product referred to in the CME/CPD program or that is marked by a commercial organization (pending)
- Am currently participating in or have participated in a clinical trial within the past two years

Irena Hozjan
- Has received a grant or an honorarium from Serono, Lilly, Sandoz, and Novo Nordisk
- Is a member of an advisory board or equivalent with Serono, Sandoz, and Lilly

Nicole Kirouac
- No relationship

Farid Mahmud
- Is a member of an advisory board or equivalent with Novo Nordisk

Lois Mulligan
- No relationship

Susan Murphy
- Is a member of an Advisory Board or equivalent with Serono and Sandoz

Deborah O'Connor
- Intends to make therapeutic recommendations for medications that have not received regulatory approval (i.e. "off-label" use of medication)

Karel Pacak
- No relationship

Bruce Perkins
- No relationship

Susan Rybansky
- Is a member of an Advisory Board or equivalent with EMD Serono, Eli Lilly
- Has received a grant or an honorarium from EMD Serono, Eli Lilly and Sandoz

Phaedra Thomas
- No relationship

Committee (if not indicated under "Speakers")

Jean-Pierre Chanoine
- Is a member of an Advisory Board or equivalent with Lilly, Serono and Roche
- Has received a grant or an honorarium from Hoffman La Roche
- Am currently participating in or have participated in a clinical trial with Lilly, Roche, and Serono

Danièle Pacaud
- Am currently participating in or have participated in a clinical trial with Eli Lilly, EMD Serone Novo Nordisk, Sanofi Aventis and Marcogenics
- Has received an unrestricted educational grant from Sanofi-Aventis

Mark Palmert
- Has received a grant or an honorarium from Eli Lilly (pediatric grand rounds at McGill University)

Wendy Schwarz
- Has received an honorarium from EMD Serono, Sandoz, and Lilly Canada

Elizabeth Sellers
- No relationship

Jonathan Wasserman
- No relationship

Diane Wherrett
- Is a member of an Advisory Board or equivalent with Eli Lilly (gave a talk at an advisory board)
- Has received a grant or an honorarium from Medtronic (chaired an educational presentation)

Oral Abstract 1

Vitamin D Status in a Subgroup of Adolescents with Type 1 Diabetes

Y Yeshayahu, EB Sochett, S Sud, FH Mahmud
Division of Endocrinology, The Hospital for Sick Children, Toronto, CANADA

Introduction: Toronto is a very diverse city with 47% visible minorities. Therefore children here are at risk for vitamin D deficiency. Studies have shown an association between T1D and low vitamin D status mostly at time of diagnosis, suggesting that vitamin D plays a role in the pathogenesis of diabetes.

Aims: To characterize the state of vitamin D in adolescents with T1D, and to determine whether our data compares to healthy Canadian adolescents.

Methods: Adolescents with T1D for at least 2 years, aged 12-18 years were enrolled in our study between January-March 2010, and information including ethnicity and use of vitamin D supplements was self reported. Additional data including duration of disease, A1c, BP, BMI, was extracted from the charts. Serum levels of 25-OH D were obtained, and results were compared to the 2010 Statistics Canada report of healthy adolescents which served as our control group.

Results: 271 patients were enrolled, 51% males and 49% females, ethnic distribution was 59% Caucasian, 18% African-Canadian, 5% Asian, 8% south Asian, 6% Latin American, 4% mixed. Prevalence of D deficiency (<37.5nmol/l) was higher in the diabetes group (17% vs 12%). 17% were deficient, 60.5% were insufficient (37.5<D<75) and 22.5% were sufficient (>70). Vitamin D deficiency was present in 44% of the African-Canadian population compared to 4% of Caucasians (P<0.0001). A1c was 9.7, 8.7 and 8.8 in the D deficient, insufficient and sufficient groups respectively (P=0.002).

Conclusions: This descriptive data demonstrates the high prevalence of vitamin D deficiency in our population with T1D, which is significantly higher in ethnic minorities as compared to Caucasians. Routine screening for vitamin D levels and supplementing during winter should be considered, in high risk ethnic groups.

Data from our interventional trial linking vitamin D to vascular function is currently analyzed and will be presented as it becomes available.

Oral Abstract 2

Identifying Hypertension in Children and Adolescents with Type 1 Diabetes: A Cross-Sectional Study

Caroline Zuijdwijk, Janusz Feber, Olivia Murnaghan, and Meranda Nakhla, Division of Endocrinology and Metabolism, Children's Hospital of Eastern Ontario, Ottawa, ON.

Arterial hypertension (HTN) is a contributor to the development of micro-and macro-vascular complications in patients with type 1 diabetes (T1D). Up to 75% of cardiovascular disease (CVD) risk in T1D is attributable to HTN. Although the identification and management of HTN is recognized as being vital in preventing and slowing the progression of end-organ damage, studies in both the general pediatric and diabetes populations have shown that HTN may not be routinely recognized. In children and adolescents, blood pressure (BP) standards are based on gender, age and height percentile. These standards are presented in the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents (Standard BP tables). A potential barrier to the diagnosis of HTN is the hundreds of normal and abnormal BP values contained in the Standard BP Tables. To address these issues, a simplified table of BP values (Simple BP table) has been proposed as an effective screening tool for pediatric HTN.

Objectives: (1) To determine the prevalence of unrecognized HTN in children with T1D using (a) the Standard BP Tables and (b) the Simple BP Table, and (2) To determine the sensitivity and specificity of the Simple BP Table for the diagnosis of HTN in T1D.

Method: Retrospective chart review from January 2008 to December 2009 of patients, ages 3 to 18 years, with T1D for at least 1 year. Data collection included: baseline characteristics (including gender, age and height), blood pressure measurements, documented recognition of abnormal BP readings and a diagnosis of HTN. We will be presenting the prevalence of undiagnosed HTN as identified by both the Standard and Simple BP tables, as well as the sensitivity and specificity of the Simple BP table.

Oral Abstract 3

The Association Between Physical Activity, Fitness and Insulin Sensitivity in a Cohort of School-Aged Children with an Obese Parent

M Henderson[1], K Gray-Donald[2], ME Mathieu[3], J Hanley[1], R Rabasa-Lhoret[4,5,6], M Lambert[7].
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[3] Department of Kinesiology, Université de Montréal, and CHU Sainte-Justine, Montréal, QC, Canada
[4]Department of Nutrition, Université de Montréal, and [5]Diabetes Research Center (MDRC), Centre Hospitalier de l'Université de Montréal (CHUM), and [6]Institut de Recherches Cliniques de Montréal, Montréal, QC, Canada.
[7]Department of Pediatrics, CHU Sainte-Justine and Université de Montréal, Montréal, QC, Canada

Introduction: Our objective was to examine the association between physical activity (PA) and fitness on insulin sensitivity (IS); to determine whether these relationships differed across sexes, and to assess whether there was an interaction between PA and fitness.

Methods: Baseline data were obtained from the QUALITY cohort, an ongoing longitudinal cohort of 632 Caucasian youth, aged 8 to 10 years at recruitment, with at least one obese biological parent. IS was measured by 2 fasting indices, insulin and HOMA-IR, and an OGTT-based index, Matsuda ISI. Fitness was measured by VO_2 peak; PA was measured over 7 days using an accelerometer (counts per minute). Potential confounders included age, sex, fat mass index [FMI=total body fat measured by DXA (kg)/height $(m)^2$] and Tanner stage (pre-pubertal/pubertal).

Results: Increasing fitness levels were associated with improved IS as measured by HOMA-IR ($p=0.062$), fasting insulin ($p=0.069$) and Matsuda ISI ($p=0.005$), while PA was not associated with IS after controlling for age, sex, fitness, Tanner stage and FMI. Increasing age and FMI predicted poorer IS. Pubertal children tended to have poorer IS as well. We found no difference in these associations across sexes, nor did we find a significant interaction between fitness and PA.

Conclusions: In children with an obese parent, fitness is associated with improved IS, independently of PA, age, FMI, Tanner stage and sex, although this reached statistical significance only with the OGTT derived measure of IS. PA did not appear to be an independent predictor of IS Longitudinal studies are required to assess how these associations change over time.

Oral Abstract 4

Cardiac Function During Exercise in Adolescents with Type 2 Diabetes (T2DM)

Teresa E Z Pinto M.D.[1], Silmara Gusso MSc[1], José G B Derraik PhD[1], Tim S Hornung[2], J Chris Baldi[3], Wayne S Cutfield[1], Paul L Hofman M.D. PhD[1]
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2 Department of Paediatric Cardiology, Auckland District Health Board, Auckland, New Zealand
3 Department of Biological Sciences, Northern Arizona University, Flagstaff, Arizona, USA.

Maximal exercise capacity may be limited as a result of T2D, irrespective of deconditioning in both adults and adolescents. This may be secondary to central (cardiac) and/or peripheral (vascular) abnormalities.

Aims: 1) determine exercise capacity (VO2peak) in adolescents with T2D compared to an obese, non-T2D group and a non-obese control group; 2) assess cardiac function in these groups using cardiac magnetic resonance imaging (MRI) at rest and during sub-maximal exercise; 3) assess femoral artery flow (FAF) at rest and immediately post- exercise as a marker of peripheral vascular function.

Methods: 13 subjects with T2D, 27 overweight/obese subjects and 19 non-diabetic, non-obese controls, (12-20 years) performed incremental exercise testing on a cycle ergometer. Body composition was determined using dual-energy x-ray absorptiometry (DEXA). Cardiac and FAF MRI images were taken at rest and during or immediately after sub-maximal exercise using an MRI-safe cycle ergometer.
Results: Maximum heart rate was higher in the controls than in the two other groups ($p<0.01$) despite comparable maximum workloads. There was no difference in cardiac output (CO) indexed for fat free mass (FFM) between groups at rest however, during exercise CO/FFM was lower in T2D compared to the obese and control groups ($p<0.01$, $p<0.001$ respectively) likely secondary to a lesser increase in stroke volume. End-diastolic volume (EDV)/FFM was significantly lower in T2D at rest and during exercise. End-systolic volume (ESV)/FFM was lower in T2D compared to controls. The average FAF/minute/FFM and the net forward volume/ FFM were lower in T2D post exercise compared to the other groups.

Conclusion: Independent of obesity, T2D negatively affects both central and peripheral vascular function during exercise in adolescents. Central causes appear to be secondary to impaired filling and possible diastolic dysfunction, while peripheral blood flow to exercising muscles may also be impaired. Both may hinder one's ability to engage in physical activity.

Oral Abstract 5

Adrenal Suppression and Fat Replacement of the Right Ventricle Myocardium in a Severely Obese Adolescent Girl

Paola Luca MD and Jill Hamilton MD, FRCPC

A 17 year old girl presented with an 8 month history of rapid weight gain (100 lbs), striae, generalized edema, and recurrent lower leg skin infections. She had a history of >100 Emergency department visits in the previous 3 years for dyspnea. Her medical history included obesity (BMI 55.1 kg/m^2), asthma, reflux, anemia, acquired Chiari I malformation, and an extensive psychiatric history. She was admitted to the intensive care unit for septic shock after presenting with tachycardia, hypotension and extensive skin breakdown. Cushing's syndrome was suspected, however, on further questioning, she reported taking 6000 to 8000 mcg of inhaled steroids per day in the past year for shortness of breath. A random cortisol was <28 nmol/L. She was started on stress doses of hydrocortisone which were slowly weaned to maintenance requirements with clinical improvement.

Multiple services were consulted. She experienced significant issues related to low albumin due to poor nutrition and interrupted skin integrity. This was complicated by lymphedema, poor venous return and significant painful extremity swelling impairing mobility. Investigations for underlying causes were negative. Specifically, an echocardiogram failed to identify any abnormalities, although the visibility was limited due to her obesity. She arrested suddenly with ventricular fibrillation, moments after complaining of feeling 'unwell'. Despite inotropes, amiodarone, antibiotics, and hydrocortisone, she was unable to be resuscitated. Autopsy revealed morbid obesity with multiple skin ulcers, bilateral adrenal atrophy, pulmonary edema, and cardiomegaly with fat replacement of the right ventricle myocardium, suggestive of arrhythmogenic right ventricular cardiomyopathy (ARVC).

The prevalence of ARVC has been estimated at 1:2500 to 1:5000 and affects children and young adults. Although the etiology is not entirely clear, it may represent an inflammatory process following myocarditis modulated by genetic influences in desmosome-related proteins in the cardiac muscle. This case highlights the complex medical issues in adolescents with severe obesity and the challenges performing investigations in this population.

Oral Abstract 6

Prevalence and Function of p53 Mutations Among Children with Adrenocortical Carcinoma

Jonathan D. Wasserman, Ana Novokmet, Claudia Eichler-Jonsson, David Malkin. The Hospital for Sick Children, Toronto, ON, Canada

Adrenocortical carcinoma (ACC) is a rare tumour in children, affecting 0.4 per million children worldwide. Incidence in adults is approximately 5-fold higher and may represent a distinct genetic and biologic entity. Mortality rates among patients with ACC are high, and treatment options are limited. Previous studies have suggested that up to 90% of children who develop ACC possess germline mutations in the p53 tumour suppressor gene. In this study we sought to define the prevalence and mutational spectrum of p53 among a large, multi-center cohort of children with ACC. We further compared the mutations found amongst these children with mutations found in individuals with the Li-Fraumeni syndrome (LFS), a familial cancer predisposition syndrome which is strongly associated with germline p53 mutation.

Among 59 independent pedigrees assessed for p53 mutation, 30 (51%) carried a heterozygous mutation in p53. Only one mutation was identified more than once among this cohort. This is in contrast to families with the LFS, where 6 "hotspot" mutations are disproportionately over-represented, combined accounting for 20% of patients with p53 mutations.

In the course of this analysis, we identified several novel p53 mutations among children with ACC. These mutations were functionally characterized *in vitro* based on the ability of mutant p53 to activate transcription of known p53 target genes and to suppress growth. In contrast to classic "hotspot" mutations, several of the novel mutations demonstrated either full p53 function or partial loss-of-function. The association of these seemingly milder mutations with adrenal tumourigenesis suggests that adrenal tissues may be more sensitive to partial decrease in p53 function than other LFS-affected tissues and that other predisposing or disease-modifying genetic alterations may propel these cells towards a transformed fate.

Further efforts to identify disease-modifying loci, as well as pathogenic loci in children with wild-type p53 genes are underway via genome-wide analysis.

Oral Abstract 7

Unusual Presentation of Combined Pheochromocytoma and Paraganglioma in a Teenage Girl

Mohamad Sharkia, MD and Daniel L. Metzger MD, FAAP, FRCPC.
Department of Pediatrics, Division of Pediatric Endocrinology, BC Children's Hospital, Vancouver, BC.

Introduction: Pheochromocytomas and paragangliomas (PHEO/PGL) are rare neuroendocrine neoplasms in children that arise from sympathetic and parasympathetic paraganglia. Given the limited experience with PHEO/PGL in children, it is still very important to identify clinical presentation, diagnosis, and treatment among this age group, particularly with the evolving understanding of their pathophysiology.

Case report: We report a 17-year-old female who presented with 5-year history of excessive episodic sweating mostly associated with palpitations, tiredness, redness and swelling in both hands and feet, and numbness in the periphery when exposed to cold. There was no history of headaches, weight loss, fainting, dizziness, chest pain or pallor.
Past medical history: Unremarkable.
Family history: Father 55 years, HTN since age 35 years and hyperthyroidism.
Exam: BP 191/127 recumbent and 156/121 standing. Pulse 120/min. Skin showed small nevi on chest. Hands and feet are puffy and red. Fine tremor in outstretched hands.
Lab investigations: Chemistry, CBC, coagulation studies all normal; TSH 1.5 mU/L; fT4 7.6 pmol/L. 24-hour urine collection: epinephrine <21 nmol/L (<21), norepinephrine 10,771 nmol/L (92–272), dopamine 1896 nmol (1400–2600), VMA 114 µmol(<31).
Imaging: abdominal U/S: 4.8 × 2.8-cm mass in left adrenal bed. MIBG: intense tracer uptake in left adrenal mass. MRI and PET scan confirmed left adrenal mass, and two more intra abdominal foci.
Management: patient was prepared with alpha blockade, left adrenalectomy, and removal of two other intra abdominal tumors. Postoperative urine catecholamines are normal.
Genetics: mutation analysis revealed a *SDHD gene* mutation. The other family members are being screened.

Discussion: In a recent literature review, up to 20% of PHEO/PGL are diagnosed in children. Most are functional tumors, and clinical presentation includes symptoms related to catecholamine hyper-secretion and/or tumor mass effect. Plasma and/or urine metanephrines are the best diagnostic test for a functional tumor, and imaging studies should be directed to diagnose multifocal disease. The management of pediatric patients is, in large, similar to adults. Genetic counseling should be undertaken in all cases.
PGL1 (*SDHD* gene mutations) is associated primarily with parasympathetic head and neck PGL (also known as glomus tumor or chemodectoma), with a 68% penetrance of this phenotype by age 40. In our case, the expression was both as pheochromocytoma and abdominal paragangliomas, both of which are very rare in association with this mutation.

Conclusion: PHEO/PGL is still a rare diagnosis during childhood and often presents without the classical signs and symptoms. Even so, the pediatrician/pediatric endocrinologist should be able to recognize and screen for such tumors, particularly in the context of a known genetic predisposition.

Oral Abstract 8

A Novel Germline*CDC73 (HRPT2)* Mutation in An Adolescent with Atypical Parathyroid Adenoma: A Case Report

Dr Dalia Alabdulrazzaq. B.Med. Sc. B.M.B.Ch. FRCPC, Dr Etienne Sochett. MD.FRCPC

Introduction: Atypical parathyroid adenomas are a rare cause of primary hyperparathyroidism in children and adolescence. Germline mutations in *CDC73* (previously known as *HRPT2*) have been identified in many cases associated with atypical parathyroid adenomas. These cases are mostly associated with Hyperparathyroidism-jawtumor (HPT-JT) syndrome and *CDC73*-related familial isolated hyperparathyroidism (FIHP). This highlights the importance of *CDC73 (HRPT2)* as a causative mutation in cases of hyperparathyroidism secondary to atypical adenomas.

Material and Methods:A 17-year-old boy, who was previously healthy, was admitted to hospital with acute history of hip pain on the background of chronic generalized bone pain. Initial workup revealed hypercalcemia.Further investigations had diagnosed primary hyperparathyroidism. A right inferior parathyroid adenoma was identified by ultrasonography and was surgically resected. Atypical histological features of the resected adenoma were identified including mitotic activity and nuclear pleomorphisim.

Results: DNA testing was performed and identified a novel mutation for *CDC73 (HRPT2)* with inframe deletion in exon 3. Screening for *CDC73*-associated conditions was undertaken. Dental examination showed no symptomatic jaw fibromas. There was no associated renal lesion identified by ultrasonography and renal function tests were normal. Genetic testing of first degree family members is done and results are pending. The case is being monitored currently with no symptoms or signs of recurrence.

Conclusions: Despite the reported rarity of *CDC73(HRPT2)* mutations in cases with primary hyperparathyroidism, the presence of atypical parathyroid adenomas mandates consideration of *CDC73 (HRPT2)* mutation. Furthermore,identification of such mutation should also mandate for serious consideration of genetic testing of the family and investigating for *CDC73 (HRPT2)*-associations namely, jaw tumors, renal lesions, and recurrence of parathyroid lesions.

Oral Abstract 9

A Retrospective Review of Pituitary MRI Findings in Pediatric Patients on Growth Hormone Therapy

S. L. Tsai, E. Laffan, S. Lawrence

Introduction: Magnetic resonance imaging (MRI) is an important tool for delineating pituitary gland anatomy. Previous reports have shown that patients with multiple pituitary hormone deficiencies (MPHD) have more pituitary anatomy abnormalities compared to those with isolated growth hormone deficiency (IGHD). Correct identification of the classic triad (interrupted or thin pituitary stalk, absent or ectopic posterior pituitary and anterior pituitary hypoplasia) and its variants has important clinical implications.

Methods/Results: The MRI findings in 52 pediatric patients, who were diagnosed with growth hormone deficiency from 1988 to 2010 were reviewed. Fifteen patients had MPHD and thirty-seven had IGHD. Thirteen patients had the classic triad. Of the 15 patients with MPHD, 9 had the classic triad, 5 had variants of the classic triad and 1 had a normal MRI. Of the 37 patients with IGHD, 4 had the classic triad, 8 had varying degrees of pituitary anatomy abnormalities, and 25 had normal MRI findings.
The MRI images were reviewed by an expert neuroradiologist, and the results were compared to the findings from previous reports. There were discrepancies found with the original reports in 9/52 cases. The discrepancies ranged from inappropriate identification of a pituitary microadenoma (n=4) to misidentification of one or more elements of the classic triad (n=5).

Conclusions: The number of patients identified as having abnormal pituitary anatomy in this study is consistent with current literature. Imaging of the pituitary gland is an important clinical tool, as those with the classic triad are at higher risk of developing MPHD, and therefore, should be screened more closely. The level of discrepancy between the initial report by a non-neuroradiologist and that of an expert neuroradiologist is important to note, as this can have important clinical implications for patients.

Oral Abstract 10

Can Recovery from Hypothalamic-Pituitary-Adrenal (HPA) Axis Suppression Following Supraphysiological Doses of Glucocorticoids be Predicted?

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Background: Supraphysiological doses of glucocorticoids (GC) may cause adrenal insufficiency, which can be lethal if left unrecognized or untreated.

Objective: To determine which biomedical variables (8am serum cortisol, peak cortisol and Δ cortisol level after ACTH stimulation) and clinical variables (patient and pretest factors) are important for predicting recovery of the HPA axis.

Design/methods: We included patients who underwent low dose ACTH (1µg) testing between October 2008-June 2010 after receiving at least one month of supraphysiological doses of GCs (prior 8 am cortisol level ≥200nmol/L required).

Results: Data from 104 patients were analyzed (age 8.4±5.4yrs, median 8.0; 57 girls). Baseline cortisol on the day of testing was normal (≥193nmol/L) in 77 pts. Sixty-three pts (57%) had a normal response to ACTH stimulation (peak cortisol ≥500nmol/L). Peak cortisol was not correlated to baseline cortisol, duration of treatment or cumulative dose of GC; a normal baseline cortisol did not preclude subnormal response to ACTH (21%).
While clinical signs of hypoadrenalism were rare (n=2), the following were observed: growth deceleration (n=38; 37%), excessive weight gain (n=34; 33%), underlying disease (asthmatic n=24, dermatologic n=25, gastroenterological n=6, hematological n=25, nephrological n=2, oncological n=5, rhumatological n=14, cardiological n=3); duration of treatment (608±770days; median 370; 5-4226), maximal daily and cumulative dose of GC in hydrocortisone equivalent, respectively (427±693mg/m^2/day; median 200; 12-3700: 27209±36079mg/m^2; median 15011; 82-178210); timing since last dose of GC (105±253days; median 46.5; 1-1584) and duration of physiological replacement with GC (215±255days; median 118; 0-1089).

Conclusion: Following treatment with suppressive doses of GC, a normal 8 am cortisol level is not helpful for predicting response to low dose ACTH stimulation. Given the absence of positive clinical and biomedical predictors, physicians must continue to screen patients with ACTH testing and maintain a state of vigilance for the risk of acute adrenal failure under situations of stress.

Oral Abstract 11

Expression of Lin28a and Lin28b Decreases Across the Pubertal Transition

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50-80 % of the variation in pubertal timing can be accounted for by genetic factors. However, the genetic regulation of pubertal timing is not well understood, and specific genes and pathways are largely unknown. Recently, genome-wide association studies have demonstrated that variations in and around the gene *LIN28B* associate with variation in pubertal timing in humans. We and collaborators have subsequently demonstrated that over-expression of *Lin28*a leads to delayed puberty in mice, establishing the Lin28 pathway as an important regulator of the reproductive endocrine axis.

To determine if the expression of *Lin28a* and *Lin28b* changes across the pubertal transition, hypothalamic, pituitary, and ovarian tissue was collected from female mice at 20, 25, 30, 35 and 45 days (the mean age of vaginal opening is 31.5 ±2.4 days in our colony). RNA was extracted from these tissues and the mRNA levels of *Lin28a* and *Lin28b* were quantified and normalized to ß-actin mRNA levels using quantitative PCR. Statistical significance was assessed by using one-way ANOVA followed by Bonferroni multiple comparison test. Given that over expression of *Lin28a* results in delayed puberty in mice, we hypothesized that the expression of *Lin28a* would decrease across the pubertal transition.

The mRNA levels of *Lin28a* and *Lin28b* decreased in ovarian tissue from day 25 onward ($p<0.01$ for both). The expression did not change significantly in hypothalamic and pituitary tissue.

We have previously demonstrated that over expression of *Lin28a* leads to delayed puberty in mice. Here we report that *Lin28a* and *Lin28b* expression decreases across the pubertal transition, providing further evidence that this newly identified pathway is an important modulator of pubertal timing and may be part of the brake that inhibits pubertal onset. Moreover, our data suggest that a main site of *Lin28a* and *Lin28b* regulation of may be the ovary.

Oral Abstract 12

Do Epigenetic Mechanisms Regulate the Timing of Puberty? Initial Evidence from Studies in Female Mice

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The timing of pubertyis characterized by wide, almost normally distributed variation and regulated by multiple factors including genetic background, environment and general health. Epigenetic regulation is a strong determinant of many disease and developmental phenotypes and may also modulate pubertal timing. Epigenetic mechanisms include differential DNA methylation and histone acetylation that alter gene expression, arisingsporadically or from environmental differences.

To begin to assess whether epigenetic mechanisms modulate pubertal timing, we established the distribution of pubertal timing (assessed by age at vaginal opening, VO) among a population (n=239) of genetically identical C57BL/6Jfemale mice raised in a uniform macro-environment. We then assessed variables within the micro-environment that could affect variation in VO independent of epigenetic effects. Pups per litter,males:females in litter, age of parents, and mother's first or second pregnancy did not affect pubertal timing; nor was there evidence that VO occurred at a critical weight. However, weight at weaning and rate of weight gain between weaning and VO correlated negatively ($p<0.01$) with VO, suggesting an association between faster growth and earlier puberty.

To assess whether epigenetic factors influence growth and VO, we treated mice with anepigenetic modifying agent, histone deacetylase inhibitor suberoylanilidehydroxamic acid (SAHA), known to increasehistone acetylation and decrease DNA methylation. SAHA was administered at25mg/kg in drinking water from weaning to VO. Treated mice exhibited significantly delayed puberty compared to controls (VO at 33.3±1.90 days, n=23 vs. 32.0±2.06, n=26, $p<0.05$). Ovarian weights at day 32 were also smaller (1.87±0.31mg, n=16 vs. 2.30±0.32mg, n=10, $p<0.01$). Although perhaps limited by number of mice, rate of growth was not significantly different between the treated and control mice.

Our initial data suggest that epigenetics may be an important modulator of pubertal timing. Future investigation will focus on identification of mechanisms and responsible genes/pathways.

Poster Abstract 1

A Case of Autoimmune Hyperthyroidism in a Child with End-Stage Renal Disease

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We report a case of autoimmune hyperthyroidism in an 11-year-old girl with chronic renal disease, secondary to familial nephronophthisis. The diagnosis was delayed despite a suppressed TSH level (<0.01mU/L) at the onset of symptoms. Thus, the hyperthyroidism progressed and her renal function deteriorated. Repeated thyroid function showed TSH 0.04 mU/L; FT4 57.6 pmol/L (normal 7.5- 17.2); and a FT3 17.9 pmol/L (normal 3.5-6.7). Soon after dialysis therapy and endocrinology visit, she was treated with methimazole at a dose 0.3 mg/kg.

In theory, in the setting of end-stage renal disease (ESRD), hyperthyroidism may occur with a frequency similar to that of the general population. That this study centers on a young girl adds to a dearth of less than 15 reported cases (a third describes adults over the age of 60, a third under 40, and only one describes a 19-year-old male) and offers rare insight into the disease. Thyroid hormone is essential for adequate growth and development of the kidney. Hyper-thyroidism affects mature kidney function as it is associated with decreased systemic vascular resistance, increased cardiac output, increased renal blood flow, hypertrophic and hyperplastic tubuli, and an increased glomerular filtration rate. The net effect of hyperthyroidism is a worsening of renal function which progresses to glomerular sclerosis, proteinuria, oxidative stress, and/or renal failure. Biochemical manifestations of hyperthyroidism may be masked by changes due to nonthyroid illness, including reduced T4 binding to serum carrier proteins and impaired T3 conversion from T4. There also are central effects resulting in "sick euthyroid syndrome." Thus, normal or reduced T3 values may not exclude hyperthyroidism.

Poster Abstract 2

When is a Pheo Not a Pheo? Depression in an Adolescent Leading to a Pheocromocytoma-Like Biochemical Profile

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A 14 4/12 year old boy presented with a 6 months history of a 10 kg weight loss, profuse sweating, fatigue, myalgia, sleep difficulties and loss of appetite. The sweating and resting tachycardia of 130 bpm prompted his primary team to perform an evaluation for pheochromocytoma with concerning results: his 24-hour urine collection revealed elevated dopamine, norepinephrine, and metanephrine levels (table). Other history was notable for a history of being physically abused, ongoing feelings of sadness, and three suicide attempts.

To confirm the urinary catecholamine results and to localize the presumptive tumor, an extensive workup was carried out over the course of 1.5 years, which included multiple blood and urine tests and several negative imaging studies. Because of worry about false positive catecholamine data, a clonidine suppression test was performed to differentiate between a benign state of catecholamine elevation and a true pheochromocytoma. The results were consistent with a pheochromocytoma, showing no suppression of plasma catecholamines; additional imaging studies were performed, but all were negative (table).

In parallel to the medical workup, he was followed by psychiatry for his depression. Seven months after his initial presentation, treatment with Fluoxetine was initiated, and titrated up to 30 mg daily. He also underwent occupational and physical therapy. This led to marked improvement in his mood and function, and a concomitant, progressive drop in his catecholamine levels, which on last measurement had normalized (table).

Pheochromocytoma is a challenging but important diagnosis, with plasma metanephrines being the gold standard for diagnosis. In cases where this is insufficient, a clonidine suppression test is considered the most sensitive and specific additional diagnostic test. To our knowledge, this case is the first report of depression in an adolescent leading to a convincing biochemical profile of pheochromocytoma, which then normalized with treatment with antidepressants and supportive therapy.

Poster Abstract 3

Suspected Antacid-Induced Rickets and Multiple Fractures in a Child with Rubinstein-Taybi Syndrome

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Objective: To describe a case of hypophosphatemic rickets and multiple fractures in a child with Rubinstein-Taybi syndrome receiving aluminum-containing antacids.

Case summary: A 6-year 3 month-old boy with Rubinstein-Taybi syndrome with developmental delay and gastroesophageal reflux was initially referred to our clinic for precocious adrenarche.

On history during a follow-up visit, parents reported two fractures in the left foot in the last 3 months. Radiographs of his left foot showed evidence of rickets and diffuse osteopenia. Vitamin D 2000 IU daily was started after the history of fractures. Initial investigation revealed: ionized calcium 1.21 mmol/L, phosphate 0.65 mmol/L, alkaline phosphatase 477 U/L, intact parathyroid hormone 1.5 pmol/L (1.6–9.3), 25-hydroxy-vitamin D 71 nmol/L (optimal 75–225), 1,25-dihydroxy-vitamin D >250 pmol/L (40–190). He returned 2 weeks later and had repeat blood work and a 24-hour urine collection for tubular reabsorption of phosphate. The results are as follows: total calcium 2.42 mmol/L, phosphate 0.31 mmol/L, and alkaline phosphatase 453 U/L, 24-hr urinary phosphate <0.26 mmol/d, 24-hr urinary calcium 3.58 mmol/d (<0.15 mmol/kg/d).

On medication history, it was apparent that the patient had been taking aluminum-rich antacid (5 ml= 153 mg aluminum hydroxide) 5-10 ml five days a week for the past 23 months. The antacid was discontinued. The patient continued on Vitamin D 2000 IU daily and was started on Rocaltrol 0.25 µg bid and phosphate 250 mg QID. Two weeks after initiating treatment, patient presented to the emergency with pain with movement and bilateral femoral neck fractures was revealed. Patient underwent open reduction with internal fixation of the fractures and was placed in a hip spica cast. The serum aluminum level that was drawn three weeks after stopping the antacid was 306 nmol/L (normal <400). Discontinuation of the antacid combined with phosphate, calcium, vitamin D and rocaltrol treatment resulted in resolution of biochemical abnormalities and proper healing of fractures.

Conclusion: Aluminum-containing antacids should be avoided or used with extreme caution in the pediatric population. These phosphate-binding agents can bind large amounts of phosphorus, causing hypophosphatemia rickets and multiple fractures.

Poster Abstract 4

Clitoromegaly in the Extremely Premature Infant - A Case Series of Four Infants

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Background: Clitoromegaly in the neonate is often the result of in utero androgen exposure or disorders of steroid biosynthesis. To date, 2 case reports of transient acquired clitoromegaly in the extremely premature infant have been published. Both cases were associated with significantly elevated androgen levels and spontaneous resolution. We report a series of 4 extremely premature infants found to develop clitoromegaly during the latter portion of their course in a neonatal intensive care.

Method: A retrospective chart review was performed for this descriptive case series.
CASES: Four extremely premature infants were referred to Pediatric Endocrinology over an 18 month period. These patients were born to non-consanguineous parents of different ethnic backgrounds. All cases were born < 27weeks GA with normal genital anatomy. They developed marked clitoromegaly, but no other signs of sex steroid exposure by 37 weeks CGA.
Clinical courses in NICU were varied. Several unifying features included birth weight <750g, treated sepsis, use of prophylactic fluconazole, PDA requiring medical or surgical (3 patients) closure**,** and treatment in the same nursery. Patients had varying degrees of intraventricular hemorrhage, jaundice, retinopathy of prematurity, length of ventilation, chronic lung disease, anemia, feeding issues, use of corticosteroids, and necrotizing enterocolitis.
Laboratory investigations in all patients revealed 46, XX karyotype, elevated androstenedione, and normal 17-hydroxy progesterone. Three patients had significantly elevated estradiol and gonadotropin levels. Most striking of all, testosterone levels were far above norms for females of any age.

Conclusion: We report 4 cases of clitoromegaly of unknown etiology in extremely premature infants. To date, despite exhaustive workup, no unifying diagnosis has been identified. All patients had improvement of the clitoromegaly and resolution of biochemical abnormalities. One was treated with Lupron, while the other three were managed expectantly. To date, little has been published on this rare clinical finding. Further exploration is required to better understand the pathophysiology and projected clinical course.

Poster Abstract 5

Relationship Between Body Mass Index and Metabolic Parameters in Obese Adolescents and Birth Size

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Epidemiological studies have demonstrated that both small and large birth size for gestational age represent risk factors for insulin resistance, metabolic syndrome and type 2 diabetes in adolescents. We have examined a cohort of obese adolescents for relative body mass index (BMI), blood lipids, glycemia, insulin resistance and blood pressure in relation to their birth weight.

The REACH study is a 2-year multidisciplinary, family based, adolescent obesity intervention (ClinicalTrials.gov number NCT00934570) to assess the effects of a structured lifestyle intervention and metformin (or placebo) on BMI and other risk factors for type 2 diabetes and cardiovascular disease in obese adolescents (age 10-16 years, BMI > 95th centile). The study design includes measures aimed at optimizing adherence to the recommended lifestyle changes. Study entry data were analyzed prior to any lifestyle or pharmacologic interventions for 92 subjects (mean age 13.6 years, 49 F, 43 M; mean BMI 33.1) with a mean birth weight of 3525gm (1899 – 4990 gm; gestational age 36 – 42 weeks). BMI z-score at study entry was positively correlated with birth weight (r^2 = 0.048, p = 0.03), but not with waist circumference. Insulin resistance, as measured by HOMA (Homeostasis Model Assessment), was negatively correlated with birth weight (r^2 = 0.05, p = 0.04), as was fasting plasma insulin (r^2 = 0.05, p = 0.03), but not fasting glucose values. A positive correlation existed between birthweight and AIC (r2 = 0.07, p = 0.03). Mean systolic BP z-score was 0.36 and diastolic z-score was 0.32. Despite the relative hypertension, no significant associations existed between birth weight and BP, plasma lipid levels, or endothelial function as measured by peripheral arterial tomography.

The results show that BMI in a cohort of obese adolescents is positively related to size at birth. However insulin resistance was worse in adolescents who were relatively small at birth, despite the lower BMI. The difference in risk factors for obese adolescents associated with a birth weight of 2.5 kg vs. 4.5 kg is approximately 0.2 BMI z-score, 0.2% AIC and a HOMA of 2.0. This demonstrates that consideration of birth size may be valuable in developing intervention strategies in obese youth at risk for metabolic syndrome and type 2 diabetes.

Poster Abstract 6

Lowering Thyrotropin Cutoff for Newborn Screening: Additional Cases of Congenital Hypothyroidism are Identified, But What Do They Really Have?

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Context: An increased global incidence (all diagnostic subcategories included) of congenital hypothyroidism (CH) has been reported from the United States over the past two decades. This may in part reflect changes in screening methods. In Québec, by contrast, the same initial whole blood thyrotropin cutoff (i.e. 15 mU/L) has been used for the last 20 years; the only change occurred in 2001, when the thyrotropin threshold was decreased from 15 to 5 mU/L on the second screening test, which is requested in cases with an intermediate thyrotropin (15-30 mU/L) on the first sample.

Objectives: To determine whether the increased global CH incidence is artefactual (due to changes in screening practices) or real (due to environmental factors such as increasing perchlorate exposure or decreasing iodine intake), we assessed the impact of the change in our screening procedure on the incidence of CH, both globally and by subcategory.

Design, Setting, Patients, and Main Outcome Measure: This is a population-based retrospective study. The Québec provincial newborn screening database was analyzed from January 1990 to December 2009. Incidence of CH and thyrotropin levels were grouped by etiology (i.e., thyroid ectopy, athyreosis, goiter, normal-size gland *in situ* and unknown diagnosis). To allow for non-linear effect, time was categorized in two periods (i.e. 1990-2000 and 2001-2009).

Results: Of 1,660,857 screened newborns over the 20-year period, 621 had confirmed CH (overall incidence: 1:2,674). Of these, 390 had thyroid dysgenesis (overall incidence 1:4,259) from either ectopy (n=291) or athyreosis (n=99); 53 had goiter (1:31,337); 115 had a normal-size gland *in situ* (1:14,184) and 63 had no reported diagnosis (1:26,212). Between 1990-2000 and 2001-2009, the incidence remained stable for dysgenesis (p=0.33; 1:4102 to 1:4,485) and goiter (p=0.41; 1:28,719 to 1:35,656). However, the incidence of normal-size gland *in situ* and CH without diagnosis more than doubled (p=0.0015; 1:22,565 to 1:9,769 and p=0.0037; 1:43,079 to 1:17,393, respectively). Consequently, the global incidence of CH increased (p=0.02; 1:2,889 to 1:2,433). The new screening algorithm identified 48 additional cases (25 normal-size gland *in situ*, 11 no reported diagnosis, 10 ectopies, 2 goiters).

Conclusion: The slightly increased incidence of CH observed in the past two decades resulted from a change in the repeat screening threshold which led to detecting more cases of non-goitrous non-dysgenetic CH, whereas the incidence of dysgenetic and goitrous CH remained stable. Whether the additional cases detected since 2001 were at risk of intellectual impairment and have transient or persistent CH remains to be determined.

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