

BRINGING THE LATEST IN PAEDIATRIC ENDOCRINOLOGY TO YOU

Make your way to Vienna!

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Welcome

The next ESPE Annual Meeting is now little more than a month away. The closing date for registration at the standard rate is Monday 5 August. Make sure you head to www.espe2019.org straight away, to view the programme and reserve your place.

This issue of *ESPE News* gives you a flavour of what you can look forward to. We are lucky enough to have previews from three of the seven plenary lecturers. On **page 5**, Josef Penninger looks at the effects of RANK/RANKL across a range of organ systems. His lecture will focus on intriguing links between the physiology/pathophysiology of bone, lactation, cancer, immunity and female hormones.

On **page 6**, Ulrich Dirnagl examines how we can we organise and reward our endeavours to ensure that our research work is of maximum benefit to humankind. When economic pressures and reward structures might deter scientists from undertaking innovative research, publishing negative studies or depositing complete datasets in public repositories, how should we respond?

The connection between reproductive function and metabolism is evident from the influence of an individual's nutritional and metabolic state on the timing of puberty. As he previews his plenary lecture, Manuel Tena-Sempere considers the relevance of hypothalamic kisspeptin-producing neurones to this link (see **page 7**).

ESPE 2019 also highlights the work of researchers earlier in their careers. On **page 8**, we hear from two people who have benefited from ESPE's financial support. Valentina Chiavaroli, recipient of the ESPE Research Fellowship, has conducted studies into preterm birth and the gut microbiome in metabolism, while Erica van den Akker's ESPE Research Unit Grant enabled her to investigate tools to monitor treatment of congenital adrenal hyperplasia. They will both speak during the Young Investigators session at ESPE 2019.

Read on, and enjoy!

Sarah Ehtisham
Editor, *ESPE News*

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Cover image: Monument to Johann Strauss Jr, Vienna, Austria ©stellalevi/Stockphoto



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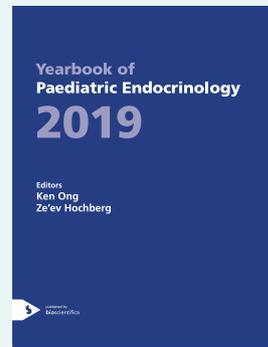
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Yearbook of Paediatric Endocrinology 2019



The 2019 *Yearbook of Paediatric Endocrinology*, edited by Ken Ong and Ze'ev Hochberg, is a collection of abstracts summarising the highlights of paediatric endocrine publications from the past year. The *Yearbook* will once again be freely available online and as a downloadable PDF.



You will find the *Yearbook* at www.espeyearbook.org.

Advancing your career through ESPE

This year's ESPE Meeting in Vienna, Austria, features a special session entitled 'Advancing your career in paediatric endocrinology through ESPE'. Here, you will find out how to advance your career, and the field of paediatric endocrinology, directly from those who have benefited from ESPE's educational activities and grants. Learn about the schools, fellowships, symposia and grants offered by ESPE, and how you can tailor your application to become a successful awardee.

Speakers include:

- **Annemieke Boot** on ESPE e-Learning and using the Resource Limited Countries portal
- **Faisal Ahmed** on the new ESPE Visiting Professorship and Undergraduate Achievement Award, and the successful bid for the ESPE Science Symposium 2020
- **Agnès Linglart** on 'How the Research Fellowship benefited my career'
- **Rasa Verkauskeine/Malcolm Donaldson** on how ESPE schools and educational activities positively shape student careers
- **Violeta Iotova** on the impact of the Clinical Fellowship on paediatric endocrinology.



The session will take place at 12.00–13.30 on **Thursday 19 September 2019**.



Read about ESPE 2019 on **page 9**, and find out more at www.espe2019.org.

With regret

We were incredibly sad to hear that Carlo Acerini (Cambridge, UK) had died in May. Dr Acerini was a great ESPE supporter and contributor. Our thoughts are with his family, friends and colleagues.



New guideline: GnRH analogues in children

The Intersociety Clinical Guidelines Committee, led by Evangelia Charmandari, Chair of ESPE's Clinical Practice Committee, has recently developed a new guideline, entitled 'Use of gonadotrophin-releasing hormone analogues (GnRHa) in children: update by an international consortium'. It will be published shortly in *Hormone Research in Paediatrics*.

Written by authors from multiple paediatric endocrinology societies around the globe, this update concisely addresses changes in GnRHa usage in children and adolescents over the last decade. Topics related to the use of GnRHa in precocious puberty include diagnostic criteria, globally available formulations, considerations of benefit from treatment, monitoring of therapy, adverse events and long term outcome data. Additional sections review use in transgender individuals and other conditions related to paediatric endocrinology. Although there have been many significant changes in GnRHa usage, there is a definite paucity of evidence-based publications to support them. These changes are reviewed not only to point out deficiencies in the literature, but also to stimulate future studies and publications in this area.

Science Committee Expert Panel

The Expert Panel reviews and assesses applications for the grants and activities within the remit of the ESPE Science Committee. If you are keen to review ESPE grant applications, please express your interest to the ESPE Team at espe@eurospe.org.

ESPE Science Symposium 2020 Congenital adrenal hyperplasia

Following a successful bid by Hedi Claahsen-van der Grinten and her team, the ESPE Science Symposium 2020 will be hosted at Radboud University, Nijmegen, The Netherlands.

Its focus will be 'Molecular medical research to clinical application in congenital adrenal hyperplasia' and it will be supported by the Endo-ERN and the ESPE DSD Working Group.

The date, in late Autumn 2020, will be announced shortly.



For details and to register see www.eurospe.org/education/espe-science-symposium

ESPE Winter School 2020 28 February–5 March 2020 near Chisinau, Moldova

We are pleased to welcome applications for the 2020 ESPE Winter School. The Winter School provides a 5-day interactive learning environment for trainee paediatric endocrinologists from Eastern Europe. It includes lectures, small group sessions and case presentations. Accommodation and meals will be provided free of charge, and travel grants will cover train or bus transport or an economy class air ticket between the participant's home and Chisinau airport.

Priority will be given to applicants from Moldova, Romania, Bulgaria, The Balkans, Turkey, Ukraine and Russia, though individuals from across Eastern Europe, Africa and the Middle East are welcome to apply.



The closing date for applications is **29 October 2019**.



Find full details and apply online at www.eurospe.org/education/winter-school.



ESPE e-Learning news

www.espe-elearning.org

Registration is **free!**

New items under 'General Content'

Puberty: Breast disorders in adolescence
Thyroid Disorders: A case entitled 'Thyroid tumours'

New items under 'Resource Limited Countries'

Diabetes ISPAD Guidelines: 'Diabetes in children and adolescents'

Growth and Growth Regulation: 'Short stature' and 'Tall stature'

Gynaecology: 'Menstrual management'

Sodium and Water: 'Disturbances of sodium and water'

Thyroid Disorders: 'Thyroid disorders'

plus seven case presentations

Bringing you recent highlights from the world of research

Low plasma adropin and hyperglycaemia

Adropin is a peptide hormone encoded by the energy homeostasis associated (*ENHO*) gene, linked to biological clocks and to glucose and lipid metabolism.

Butler *et al.* have recently reported that low levels of adropin predicted increased weight gain and metabolic dysregulation in a non-human primate model consuming a high-sugar diet. Expression of *ENHO* was higher in daytime and lower at night in most primate tissues. At night, the body relies on energy reserves stored as lipids, while during daytime it relies more on carbohydrates from the diet. Regulation of adropin expression by our internal clocks may thus contribute to the increased use of glucose during daytime.

Interestingly, development of type 2 diabetes was only observed in animals with low plasma adropin concentrations. Monkeys with low adropin may thus be unable to oxidise glucose during the daytime, explaining their higher fat content, as the glucose is converted to lipids instead of being used as a metabolic fuel.



Read the full article in Butler *et al.* 2019 *Journal of Biological Chemistry* 294 9706–9719

Metabolic bone disease of prematurity

This review by Chinoy *et al.* discusses the aetiology and risk factors of metabolic bone disease of prematurity (MBDP). The authors provide recommendations for prevention, investigation and treatment of the condition.

To prevent MBDP, the optimal enteral and parenteral ratios of calcium to phosphate must be maintained. The article shows how the recent practice of administering phosphate supplements alone has caused an increase in the number of cases of MBDP due to secondary hyperparathyroidism, resulting in pathological fractures. MBDP has traditionally been diagnosed in the presence of raised alkaline phosphatase and low phosphate. Paradoxically, phosphate supplementation alone can reduce the ionised calcium and lead to secondary hyperparathyroidism, which may worsen MBDP.

Measuring parathyroid hormone is essential in establishing whether the MBDP is primarily due to calcium or phosphate deficit, and this will guide management with the appropriate supplements and optimal calcium to phosphate ratios.



Read the full article in Chinoy *et al.* 2019 *Archives of Disease in Childhood Fetal & Neonatal Edition* doi: 10.1136/archdischild-2018-316330

Liraglutide in childhood type 2 diabetes

Liraglutide is the first non-insulin drug approved to treat type 2 diabetes in paediatric patients since metformin in 2000. It is a glucagon-like peptide (GLP-1) receptor agonist and improves blood sugar levels by creating the same effects as the incretin GLP-1: increasing pancreatic insulin release and decreasing excessive glucagon release. In type 2 diabetes, GLP-1 levels are often low. Liraglutide also slows digestion, reduces hepatic glucose output and augments pancreatic prandial insulin release. It is not a substitute for insulin and is contraindicated in ketoacidosis.

In the recently published Ellipse trial, 134 children over 10 years of age received liraglutide up to 1.8mg daily, for more than 26 weeks. 63.7% of those in the liraglutide group, compared with 36.5% in the placebo group, attained a glycated haemoglobin level of below 7.0%. Those taking liraglutide showed greater weight loss and improvement in lipid profile. Gastrointestinal side effects were common and hypoglycaemia was more frequent in the treated group. We now hope to see more real-world data on the outcomes of liraglutide treatment in this tricky age group.



Read the full article in Tamborlane *et al.* 2018 *New England Journal of Medicine* doi: 10.1056/NEJMoa1903822

Pancreatic adipocytes mediate hypersecretion of insulin in diabetes-susceptible mice

Ectopic fat accumulation in the pancreas or non-alcoholic fatty pancreas disease (NAFPD) is a consequence of obesity, and has been associated with rapid progression of type 2 diabetes mellitus.

Quiclet *et al.* tested whether intermittent fasting would prevent pancreatic lipid accumulation, as well as improve insulin sensitivity and metabolic parameters, in an obesity and diabetes-prone mouse model. Compared with mice fed *ad libitum*, intermittently fasted mice showed reduced body weight gain and fat mass, decreased circulating lipids and better glucose tolerance. Increased triglyceride levels, along with an accumulation of adipocytes, were detected in pancreatic tissue of *ad libitum*-fed mice, with only a few adipocytes found in intermittently fasted mice. Overall, insulin secretion in response to glucose was significantly reduced in islets from intermittently fasted mice. Co-culture experiments point to a role for pancreatic adipocytes in inducing insulin hypersecretion from islets, probably due to increased secretion of non-esterified fatty acids from pancreatic adipocytes.



Read the full article in Quiclet *et al.* 2019 *Metabolism* 97 9–17

RANKL and RANK: beyond bone

Josef Penninger's plenary lecture at ESPE 2019 will look at intriguing links between breast and lung cancer, and female hormones.



Josef Penninger



According to current forecasts, more women will die from lung cancer than from breast cancer for the first time in 2019

One in eight women is diagnosed with breast cancer during her life. This makes breast cancer one of the most common cancers among women. However, according to current forecasts, more women will die from lung cancer than from breast cancer for the first time in 2019.

Women are not only more susceptible to lung cancer, the disease spreads faster than in men. It is known that ingredients in tobacco smoke can cause our body's cells to mutate. But cancer is a very complex disease that breaks out because of a combination of environmental factors and genes.

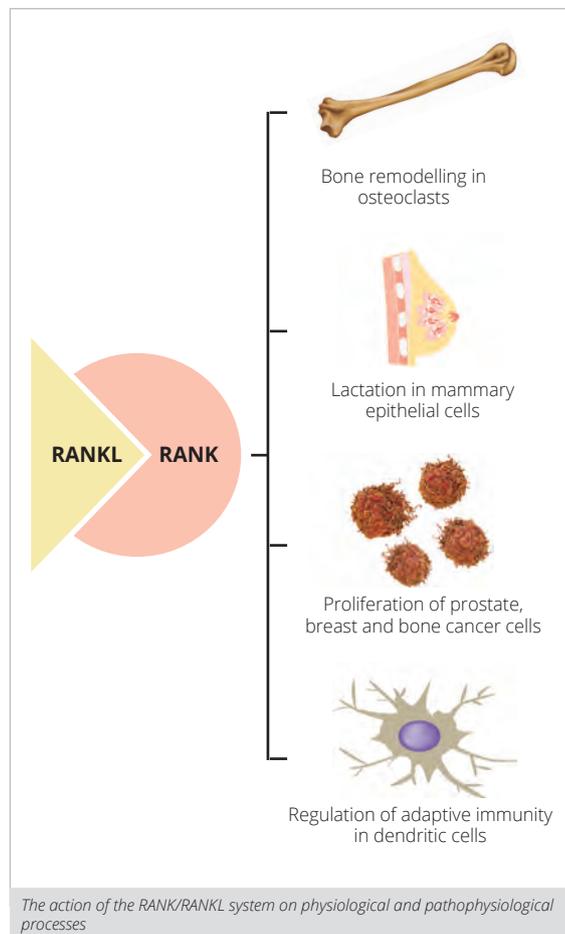
The role of RANK/RANKL

RANK/RANKL are important proteins that regulate bone metabolism in our body. RANK (*TNFRSF11A*) and RANKL (*TNFSF11*) are a receptor-ligand pair of the tumour necrosis factor receptor superfamily. They have emerged as the key molecular pathway in bone physiology, being essential for osteoclast development.

RANKL, present in either a soluble or a membrane-bound form, can be induced by various stimuli, including the sex hormone progesterone. Expression of the RANKL-decoy receptor osteoprotegerin is strongly dependent on oestrogen, thereby linking oestrogen to bone turnover.

In addition, RANKL plays a central role in the formation of mammary glands during pregnancy. In evolutionary biology, this is explained by the fact that RANKL breaks down calcium in the mother's bones during pregnancy to supply minerals for the skeleton of the growing child. In pregnant women, RANKL critically stimulates breast tissue to divide. Progesterone, a female sex hormone, sends the hormonal signal, which is then translated by RANKL and its receptor RANK into a proliferation signal for the cells.

Importantly, RANKL also mediates deregulated sex hormone signals and transformation signals, such as in *BRCA1* mutations, to the breast stem cells, thereby promoting an environment for enhanced proliferation and breast cancer. Based on such data, a multinational phase III breast cancer prevention trial is being initiated in *BRCA1* carriers, using denosumab, the RANKL-blocking antibody which has already received approval.



A link with lung cancer

Lung cancer has also long been suspected of having a hormonal trigger. Previous data from a pivotal study, which led to the clinical approval of denosumab for skeletal-related events in cancer, showed that lung cancer patients lived significantly longer when treated with this antibody.

Based on our work on breast cancer and these clinical findings, we therefore assessed the role of the RANK/RANKL system in the development of lung cancer. We found that the RANK/RANKL signalling pathway is also active in adenocarcinoma, where it promotes rapid tumour growth. RANK/RANKL acts in this carcinoma – especially in lung cancer stem cells – like an amplifier.

Intriguingly, it appears that (in a similar way to that found in the mammary gland) RANKL/RANK drives lung cancer in response to female sex hormones, identifying RANKL/RANK as a key molecular pathway that links female sex hormones not only to breast cancer, but also to lung tumorigenesis.

Josef Penninger

Canada 150 Research Chair in Functional Genetics, Director of the Life Sciences Institute, University of British Columbia, Vancouver, Canada

Reducing waste in translational biomedical research

Ulrich Dirnagl will use his plenary lecture at ESPE 2019 to ask how we can maximise the value of the research we undertake.

Recent advances in biomedical research provide innumerable opportunities to develop novel preventive and therapeutic strategies. However, only a small fraction of biomedical discoveries are successfully translated into clinical applications.

Potential 'breakthrough' therapies, which are spectacularly successful in animal models of disease, often fail in clinical trials. This translational bottleneck imposes burdens on research and healthcare systems, as well as on patients who participate in trials of novel strategies.

The high attrition rate of preclinical to clinical development may directly relate to concerns about the reliability and reproducibility of biomedical research. Apparently, there are substantial weaknesses in planning, conducting, analysing and reporting this research. Low internal and external validity, as well as low statistical power – in particular of preclinical research – appears to produce a very high rate of false positives, and inflates effect sizes unrealistically.

Not surprisingly, then, the majority of scientists believe that we are in the midst of a 'reproducibility crisis'. The immense proliferation in research outputs, combined with increasing methodological complexity and the size of datasets, greatly complicates the sharing, evaluation and synthesis of high quality evidence. At the same time, non-publication of results leads to duplicative research and deprives medical decision makers of the totality of evidence. But how can we overcome this crisis?

Incentives and disincentives

How research is organised and rewarded shapes our ability to realise its promise. For instance, economic pressures and reward structures in academic institutions can deter researchers from undertaking innovative research. They can also discourage researchers from publishing negative studies or depositing complete datasets in public repositories.

Even more importantly, the way incentives and rewards in biomedicine are currently balanced antagonises methodological robustness and rigour, as well as stimulating over-interpretation of results. Jobs and promotions depend on having published in a small

set of top-tier journals ('publish or perish'). Innovation and excellence are measured by simple, but insufficient, metrics – such as the 'journal impact factor'.

At the same time, quality and reliability, or the societal relevance of research, carry little weight for careers in biomedical research. Since biomedicine operates under a trust model that is no longer considered appropriate in corporate life or in government, researchers will neither have the incentive nor be able to solve the current problems on their own.

Seeking solutions

Instead, funders and institutions, such as the universities, research organisations, professional societies and journals, need to establish structures and alternative incentives that prevent research practices from being co-opted by the wrong interests. This requires an integrated approach to rethinking rewards and governance.

Compliance of the research with established guidelines need to be enforced, while full access to an institution's research results need to be provided through open access and open data. Researchers should be provided with tools like electronic laboratory notebooks, which enable electronic record keeping, and data and project sharing. Institutions need to establish, enforce, and reward standards for the conduct of experimental research, thereby safeguarding measures to prevent bias, such as blinding, inclusion of controls, replicates and repeats etc.

Registration of study protocols ('preregistration'), as well as provision of access to original data in publications, needs to be incentivised. Institutions need to establish programmes to train academic clinicians and basic researchers at all professional levels in experimental design, data analysis and interpretation, as well as reporting standards.

There is a plethora of measures through which we can reduce waste and increase the quality of research: some examples have been given above. However, they will only gain traction if we begin to reward robust and reproducible research when appraising academic careers or grant applications.

Institutions and funders, together with the scientists, have the power to transform biomedical research. This will take determination, time and resources. However, we will be rewarded with more robust and predictive research, which lives up to the tremendous potential of modern biomedical research to improve human health.



Ulrich Dirnagl
(BIH/Thomas Rafalzyk)

“
Funders and institutions need to establish structures and alternative incentives that prevent research practices from being co-opted by the wrong interests”

You can hear **Ulrich Dirnagl's** plenary lecture on **Saturday 21 September 2019** at **11.00–11.30**

Ulrich Dirnagl

Department of Experimental Neurology, Charité Universitätsmedizin Berlin, Germany, and QUEST Center for Transforming Biomedical Research, Berlin Institute of Health, Berlin, Germany

Nutrition and implications for the control of puberty

In his plenary lecture at ESPE 2019, Manuel Tena-Sempere will examine our understanding of the way obesity and other metabolic and feeding disorders may affect puberty.

Reproductive function, and its awakening at puberty, are essential for perpetuation of species. As such, they are under the precise control of sophisticated regulatory networks, which integrate a large number of endogenous and environmental signals.

Among them, metabolic and nutritional cues are fundamental modulators of fertility, and acquisition and maintenance of reproductive capacity are tightly bound to the state of body energy reserves, especially in females. Accordingly, persistent deregulation of energy and metabolic homeostasis is often associated with alterations in the functioning of the hypothalamic-pituitary-gonadal axis.

This close connection between reproductive function and metabolism is epitomised by the strong influence that the nutritional and metabolic state of the organism has on the timing of puberty. Indeed, while the tempo of puberty is genetically determined, it is also highly sensitive to nutritional and metabolic signals. However, while our understanding of the neuroendocrine circuits involved in the nutritional control of puberty has substantially expanded in recent years, knowledge of the specific signals and central molecular mechanisms whereby puberty onset is modulated by metabolic factors remains contentious and ill-defined.

Kiss1 neurones

Compelling evidence, gathered over the last 15 years, has demonstrated the essential role of hypothalamic neurones producing kisspeptins, encoded by *Kiss1*, in the neuroendocrine pathways that control puberty.

Kiss1 neurones are sensitive to a wide variety of pubertal modulators, from sex steroids to environmental cues, and operate as major determinants of the so-called hypothalamic gonadotrophin-releasing hormone (GnRH) pulse generation, which dictates the pulsatile release of GnRH, mandatory for full activation of the reproductive axis at puberty.

Among the populations of *Kiss1* neurones, those located in the arcuate nucleus (ARC), or its equivalent infundibular region in humans, appear to play an especially relevant role in the control of pulsatile secretion of GnRH and, thereby, pubertal onset.



Manuel Tena-Sempere

“*Perturbations of the signals and molecular pathways described ... might become targets for drugs, for the better management of pubertal disorders*”

You can hear **Manuel Tena-Sempere's** plenary lecture on Friday 20 September 2019 at 16.00–16.30

A role in metabolic cues

Notably, *Kiss1* neurones seemingly participate in transmitting the regulatory actions of metabolic cues in pubertal maturation, as exemplified by the impact of key metabolic hormones (such as leptin) and conditions of nutritional and metabolic stress (ranging from malnutrition to obesity) on the hypothalamic expression of *Kiss1*. Nonetheless, the modulatory influence of metabolic signals (e.g. leptin) on *Kiss1* neurones might be predominantly indirect, and probably also involves interaction with other transmitters (e.g. melanocortins, GABA (γ-aminobutyric acid), nitric oxide and PACAP (pituitary adenylate cyclase-activating polypeptide)) and neuronal populations.

In recent years, our research group has unveiled some of the molecular mechanisms whereby *Kiss1* neurones are directly or indirectly modulated by metabolic signals. Our work has illustrated the important functions of key cellular metabolic sensors, such as mammalian target of rapamycin (mTOR) and AMP-activated protein kinase (AMPK), activated in conditions of energy abundance or deficit respectively, in the metabolic modulation of puberty via regulation of the hypothalamic *Kiss1* system.

Thus, while preserved mTOR signalling is mandatory for proper *Kiss1* expression and the permissive effects of leptin on puberty onset, AMPK operates as a repressor of *Kiss1* expression in conditions of energy deficit.

In addition, the fuel-sensing deacetylase SIRT1 which, like AMPK, is activated in conditions of negative energy balance, also co-operates in the metabolic control of *Kiss1* expression, via epigenetic regulation of the chromatin landscape at the *Kiss1* promoter. Importantly, SIRT1 signalling in ARC *Kiss1* neurones is capable of transducing the effects of both subnutrition and obesity on puberty onset, by respectively repressing or activating *Kiss1* transcription.

Of translational interest

We believe these findings are of considerable translational interest. Compelling epidemiological evidence suggests that alterations in the age of puberty are becoming more frequent, with the escalating prevalence of obesity and other metabolic and feeding disorders possibly being a major contributing factor. Of note, alterations in pubertal timing have been associated with adverse health outcomes, including higher risk of cardiovascular and metabolic diseases, as well as earlier all-cause mortality.

Perturbations of the signals and molecular pathways described above may contribute to the alterations of pubertal timing linked to conditions of metabolic stress in humans, ranging from malnutrition to obesity, and might become targets for drugs, for the better management of pubertal disorders.

Manuel Tena-Sempere

IMIBIC; University of Cordoba; Hospital Universitario Reina Sofia; and Instituto de Salud Carlos III, Cordoba, Spain, and University of Turku, Turku, Finland

Young Investigators reap benefits of ESPE

How financial support from ESPE can boost careers in paediatric endocrinology



Valentina Chiavaroli

Gut microbiome and metabolism

Valentina Chiavaroli received her ESPE Research Fellowship in 2015. Her project 'Preterm birth and the role of the gut microbiome in metabolism' was carried out at the Liggins Institute, Auckland, New Zealand. Here, she tells us what the Fellowship has meant for her career.

My ESPE Fellowship not only made my project possible, but also supported other studies I was involved with at the Liggins Institute. The Institute is one of the world's leading research establishments focusing on fetal and child health, particularly on the long term consequences of early-life events. I worked under the supervision of Wayne Cutfield, Paul Hofman and José Derraik. Their support and my full-time involvement in a variety of studies allowed me to develop my medical research skills in paediatric endocrinology.

The many benefits of the ESPE Fellowship for my professional development included:

- establishing productive long term research collaborations

- learning advanced clinical skills, laboratory techniques and research methods
- further developing my statistical analysis skills
- attending two professional schools in paediatric endocrinology and diabetes (organised by APEG, the Australasian Paediatric Endocrine Group).

Importantly, I also had five manuscripts published in reputable peer-reviewed international journals (three as first author), and made five oral presentations at large international meetings (including the ESPE Meeting), which led to six published conference abstracts. I look forward to talking about my research at ESPE 2019.

Since my ESPE Fellowship ended and I returned to Italy, I have worked as a neonatologist in the Neonatal Intensive Care Unit at Pescara Public Hospital. The impact of early-life events (e.g. being born preterm or small- or large-for-gestational-age) on long term health remains one of my major interests.

I thank ESPE for the extremely valuable support, and the privilege of living and working in New Zealand. I hope that other young paediatric endocrinologists and scientists will apply for an ESPE Research Fellowship, to gain research and clinical experience at leading institutions worldwide.

You can hear **Valentina Chiavaroli's** talk in the Young Investigators session on Saturday 21 September 2019 at 14.00-15.00



Erica van den Akker

Novel monitoring tools in CAH

Erica van den Akker (Erasmus MC, Rotterdam, The Netherlands) received her ESPE Research Unit Grant in 2015. It enabled her to investigate novel tools to monitor treatment in patients with congenital adrenal hyperplasia (CAH), in an international project with colleagues in Bern, Switzerland, and Athens, Greece.

In my clinical work as a paediatric endocrinologist, a substantial part of my work is the treatment and monitoring of children and adolescents with CAH. I found that our current monitoring tools (such as measurement of salivary and serum androgens) were not always able to detect chronic over- or undertreatment.

The ESPE Research Unit Grant has enabled me to investigate novel tools, working with Christa Flück's team in Bern and Evangelia Charmandari's team in Athens. What has been most rewarding is the combined use of each centre's expertise. We measure hair

steroid metabolomics in Rotterdam, serum and urine metabolomics in Bern and leukocyte telomere length in Athens. This provides us with the unique opportunity to integrate all individual metabolomics results to find the optimal monitoring tools for CAH.

We are currently performing the hair metabolomics studies. My talk during the Young Investigators session at ESPE 2019 will give an overview of our preliminary results.

Receiving the Grant enabled us to co-operate at a European level with highly esteemed colleagues, with the opportunity to access innovative tools, so increasing the study quality and scientific validity of our results.

Moreover, it has provided the basis for further collaborative opportunities within and outside Europe. Currently, my research group is collaborating in several European projects in the field of paediatric obesity and steroid profiles.

I'm very grateful for the opportunities in research and collaboration that the Grant has created, and am looking forward to sharing the results of our upcoming projects with you at future ESPE Meetings.

You can hear **Erica van den Akker's** talk in the Young Investigators session on Saturday 21 September 2019 at 14.00-15.00

Find out about **ESPE Fellowships and Grants** at www.europe.org/grants-awards/grants

ESPE 2019: Variety and Variation in Paediatric Endocrinology

19–21 September 2019, Vienna, Austria

Find out more and register at www.espe2019.org

The theme of this year's ESPE Meeting is 'Variety and Variation in Paediatric Endocrinology'. It relates to the many clinical scenarios we face in our discipline, and the need to exercise care when using the term 'normality'.

At ESPE 2019, you will enjoy seven plenary lectures (see panel, right), and symposia and 'meet the expert' sessions will also give you the opportunity to learn from international experts. Free communications and poster sessions will enable delegates from around the world to present and discuss their latest findings in an interactive environment.

Our Young Investigators session allows you to hear about novel research from ESPE grant and award winners, while the 'How Do I...?' sessions provide answers to key clinical questions. Other highlights include special sessions devoted to Novel Advances, Controversies, the *Yearbook of Paediatric Endocrinology* and the Endo-ERN.

The venue for ESPE 2019 is just 7 minutes from Vienna's historic city centre by underground, with a direct link to Vienna International Airport via the airport bus.

The ESPE Meeting's many networking opportunities include your chance to meet with colleagues at our informal welcome event on the first day. Later, after the meeting closes, our ESPE Evening will provide you with a final, very enjoyable, networking event to discuss the event's highlights with your fellow endocrinologists before you return home. Tickets can be purchased online when you register.

We look forward to welcoming you all to Vienna for ESPE 2019.



Connect at ESPE 2019

As always, the ESPE Connect stand will provide a place where you can meet with colleagues and peers, pick up information, explore ESPE's e-Learning offering, or come and chat with us about matters relating to your Society. This year, Tweet us your photo during the meeting and tell us what ESPE means to you. You will be in with a chance of winning a prize! Find us on stand 7 for more details.



ESPE Working Groups

The eight ESPE Working Groups each organise a symposium at the Meeting, bringing you a selection of talks on areas of special interest:

- Bone and Growth Plate
- Diabetes Technology
- Disorders/Differences of Sex Development
- Gender Dysphoria
- Obesity
- Paediatric and Adolescent Gynaecology
- Paediatric Endocrine Nurses
- Turner Syndrome

You can view the programme for each symposium at www.eurospe.org/about/espe-working-groups. All take place at 08.00–10.00 on Thursday 19 September, except for the nurses' session which is at 14.30–16.30 on Friday 20 September.



See **page 2** for full details of the special session at ESPE 2019: 'Advancing your career in paediatric endocrinology through ESPE'

Be aware

Please exercise caution whilst registering for the ESPE Annual Meeting. We are aware that fraudulent websites are in operation, selling fake registration to ESPE 2019. The only official website where you can register to attend is www.eurospe.org.

Plenary lectures at ESPE 2019

RANKL and RANK: bone and beyond
Josef Penninger, Canada

Light, body clocks and sleep: biology to new therapeutics
Russell Foster, UK

Glucocorticoid rhythms, stress response and the brain from neonates to adult
Stafford Lightman, UK

Development of novel therapies for obesity
Matthias H Tschöp, Germany

Nutrition and the hypothalamo-pituitary-gonadal axis
Manuel Tena-Sempere, Spain

How to reduce waste and increase value in translational biomedical research
Ulrich Dirnagl, Germany

Pituitary gigantism – an update
Albert Beckers, Belgium

Future meetings

See www.eurospe.org/meetings for details of all future meetings



58th Annual ESPE Meeting

19–21 September 2019
Vienna, Austria



59th Annual ESPE Meeting

10–12 September 2020
Liverpool, UK



60th Annual ESPE Meeting

6–8 May 2021
Copenhagen, Denmark



11th International Meeting of Pediatric Endocrinology

25–28 September 2021
Buenos Aires, Argentina



OTHER EVENTS

SEPTEMBER

ESPE Summer School
Burg Feistritz, Austria
16–18 September 2019

ESPE Diabetes, Obesity & Metabolism School
Burg Feistritz, Austria
22–24 September 2019

OCTOBER

ESPE Caucasus & Central Asia School
Astana, Kazakhstan
11–15 October 2019

6th ASPED-ESPE Endocrine Academy
Casablanca, Morocco
15–17 October 2019

NOVEMBER

ESPE Maghreb School
Sousse, Tunisia
19–22 November 2019

FEBRUARY

ESPE Winter School
Chisinau, Moldova
28 February–5 March 2020

DEADLINES

AUGUST

ESPE 2019 standard registration 5 August 2019
DOM School Co-ordinator applications 5 August 2019

SEPTEMBER

Syllabus Short-Term Task Force applications
1 September 2019

OCTOBER

Winter School Co-ordinator applications
18 October 2019
Winter School applications 29 October 2019
Early Career Scientific Development Grant applications 31 October 2019

DECEMBER

ESPE Prize nominations:
- Andrea Prader Prize
- Research Award
- International Award
- Outstanding Clinician Award
- International Outstanding Clinician Award
- Young Investigator Award
10 December 2019

HELP RUN YOUR SOCIETY

Find details of vacancies at
www.eurospe.org/about/vacancies.

ESPE

European Society for
Paediatric Endocrinology

Improving care of children with
endocrine diseases by promoting
knowledge and research

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ESPE Newsletter

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