Messing with my mind
The psychological impact of endocrine disorders
As the summer warms our days, our latest issue of EYES News is out, opening the way to another great year for young endocrine scientists.

It was a pleasure to meet many of you again in Istanbul during ECE 2023, where we could share views and ideas on our community’s activities. For those who missed it, there is a recap of some of the main EYES activities in this issue, including the EYES Symposium and our sensational social event!

Meanwhile, the 2023 EYES Annual Meeting is just around the corner. Read on to discover all the information you need to submit your contribution and join our community in Würzburg, Germany, in September.

We have dedicated this issue to the relationship between hormones and one of the most underestimated determinants of our health status: psyche. You’ll find great insights from leading experts on the impact of hormonal changes on our psychological balance and psychiatric disease. Conversely, you will learn how sometimes psychological stress can impact our endocrine system.

Be delighted and inspired by the story of Dr Manuel Gahete, to whom we have dedicated our amazing career interview. You can also learn about the process of preparing ESE Clinical Practice Guidelines, through the first-hand experience of Dr van Hulsteijn and Professor Dekkers. Finally, we introduce to you the ‘newborn’ YES Group, young representatives of the European Society for Paediatric Endocrinology. And don’t forget to read our short ‘refresher’ on upcoming key ESE dates and the latest research.

Get ready and enjoy the read!

Walter Vena
Editor, EYES News

EYES News is also available as a fully digital issue at www.ese-hormones.org/eyesnews
From your EYES Co-Chairs

The EYES community gathered once again; this time in Istanbul during ECE 2023. It was great to meet you during the EYES Symposium and the amazing EYES social event in the vibrant heart of the city. You can find more about it on page 13.

A sad but necessary activity was to say goodbye to two very valuable members of your EYES Committee. Indeed, our beloved Co-Chair, Lina Paschou, had come to the end of her term of office. The Committee wholeheartedly thank her for her continuous work throughout her time in the role. She has passed the baton to Walter Vena, who will bring new energy and effort to lead the group. Our team also sadly salutes and thanks Alessandro Prete for his valuable and wise contribution to our activities and meetings. The Committee will miss you guys!

It’s no surprise that the EYES Observership Programmes keep expanding, with even more host centres, mentors and stories of participation. We couldn’t be more proud of how the project keeps developing and growing. On page 12, you can read first-hand about the experiences of Narjes Nasiri Ansari and Fabio Bioletto, who both recently finished their month abroad. Stay tuned for more news!

Exciting news is also coming from one of the biggest ESE projects. The European Research Roadmap (now known as the EndoCompass Project) will see a huge contribution from the EYES community. There will be a young representative in every working group, under the guidance of Jonathan Mertens, who is the EYES lead for this collaborative initiative.

Finally, the 10th EYES Annual Meeting in Würzburg, Germany, is just around the corner (see page 16). We are looking forward to celebrating this important milestone in the good company of our German friends from YARE (Young Active Research in Endocrinology), who will co-host the event with us, alongside their annual national meeting. Abstract submissions and registration are open now, and close on 30 July and 15 August respectively. We can’t wait to meet you there!

Antoan Stefan Šojat
Walter Vena
EYES Co-Chairs

Key dates for your diary

See www.ese-hormones.org/events-deadlines and watch your inbox for emails with details, Early Bird rates, free places and grant information!

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Würzburg, Germany, location of the 10th EYES Annual Meeting in 2023
Amazing careers: Meet Manuel Gahete

Dr Manuel Gahete is the Head of Molecular Hepatology at the Maimonides Biomedical Research Institute of Córdoba (IMIBIC), Spain. He is a member of the ESE Science Committee and received the prestigious Jens Sandahl Christiansen Basic Science Award from ESE in 2023. His research focuses on dysregulation of mRNA and protein homeostasis, as well as the involvement of metabolic changes in different liver pathologies. Here, he talks to Juan Manuel Jiménez-Vacas, Deputy Editor of EYES News.

What led you to choose endocrinology?
I have had contact with endocrinology from the beginning of my career, as I joined the Molecular and Cellular Endocrinology Group at the University of Córdoba during my graduate studies. That was my first experience in the field, but most of my subsequent research activities have been related to endocrinology.

How did your journey begin?
Joining the Molecular and Cellular Endocrinology Group early in my biological studies allowed me to closely experience laboratory research. During that time, through an Endocrine Society Summer Research Fellowship, I had a short research stay at the University of Illinois in Chicago, USA (2004). This was so gratifying that I decided to pursue a scientific career and, in 2006, I started my PhD.

What key moments have there been in your career?
Although the negative and frustrating moments, such as inexplicable results, grant rejections etc., are usually when you learn the most, I maintain that the positive moments are the most important. In my early career, I remember with special warmth the acceptance of my first research article as first author and my first oral communications at ECE and the Endocrine Society meeting. More recently, the first national project as principal investigator and the first article published as last author have been very special milestones.

What has been your greatest achievement so far?
From a professional point of view, it has been the initiation and consolidation of an independent line of research as Head of Molecular Hepatology at IMIBIC. From a scientific point of view, it is the teamwork we have developed in recent years in the University of Córdoba (along with the groups of Professors Justo P Castaño and Raúl M Luque), demonstrating the implications of the alteration of the splicing process in endocrine and metabolic diseases.

What have been your greatest challenges?
One major challenge in our careers is to develop clinically relevant research that could generate impactful results with the potential to be translated to clinical practice. I think it is crucial to build a proactive relationship between translational and clinical researchers to align research efforts and build research teams that are capable of working together and providing a more profound knowledge of endocrine diseases.

Have the obstacles facing early career endocrinologists changed?
The obstacles have mostly been maintained in recent years, including limited opportunities to access a scientific career, unstable (or even precarious) work conditions, lack of a wisely designed scientific career path, insufficient research funds and the (unfortunately common) ‘publish or perish’ dilemma. However, all these obstacles can be overcome by means of interest, perseverance and commitment. In this regard, it is important to find the appropriate mentor(s), and to have a proactive and positive attitude, including active participation in scientific activities organised by scientific societies.

Who or what has inspired you?
Although it is a difficult question, I think my mentors have had a big impact on my scientific career. From my initial determination to pursue a research career to defining my current research direction, my mentors from my predoctoral period (Professors Castaño and Luque) and my postdoctoral training (Professor Rhonda D Kineman) have been a source of inspiration. At the moment, defining novel research lines and interacting and discussing results with the young investigators in my group are also extremely inspiring.

What advice would you give to people starting their career today?
Endocrinology and metabolism are very exciting, and areas remain to be explored in clinical and translational research. There are many questions to be resolved in the clinical management of patients and in understanding the biological bases of endocrine diseases. Therefore, my recommendation would be to stay motivated and curious to address such uncertainties, and also open-minded in order to provide fresh, motivating hypotheses and answers.

Where can EYES make the biggest and most useful impact?
I think the impact EYES is having, fostering the active engagement of early investigators with endocrinology societies and supporting them in their careers, is enormous. Young investigators face a series of challenges, and EYES can help by promoting activities aimed at increasing research opportunities or stimulating the development of collaborative connections with other European groups.

What are your top tips for success?
I think there are no general tips apart from enjoying research. How to develop a scientific career and achieve success are highly dependent on a multitude of intrinsic and extrinsic factors, such as personal expectations, laboratory (or clinic) environment, research opportunities, or even your city or country. However, being engaged and motivated by the research and being able to enjoy the small (or, sometimes, big) achievements is crucial for scientific success.

What hobbies do you have that are unrelated to science?
I think having non-scientific interests is crucial, both to celebrate the good moments in science and to disconnect from stress and the not-so-good moments. When I’m out of the office and the lab, I like to enjoy life with my family and friends, to travel and visit new cities and countries, and to practise sports.
The psychology of endocrine disease

Psychoneuroendocrinology – and our understanding of the impact of endocrine disorders on mental health – is a growing area of our field. Read on for important expert insights.

Cortisol excess and psychological distress

Overt endogenous hypercortisolism induces psychiatric and neurocognitive impairment in the vast majority of cases. Here, we consider the likely mechanisms.

Glucocorticoids (GCs), particularly cortisol, play a crucial role in the allostatic process of adjustment to stressors, and can determine important changes in central nervous system (CNS) structures. The CNS expresses GC receptors ubiquitously, with the highest levels in the hippocampal area. Prolonged GC excess has been linked to both structural and functional modifications in the CNS, ultimately leading to brain atrophy.1

GC excess seems to act on brain cells through several mechanisms: a) minimisation of glucose utilisation rates in different brain areas, as observed in patients with overt Cushing’s syndrome b) insufficient production of trophic factors which are protective for brain cells, such as nerve growth factor-β and brain-derived neurotrophic factor c) defective neurogenesis in the dentate gyrus, resulting in hippocampal atrophy.

These pathophysiological mechanisms could explain the brain damage, mostly in the hippocampal area, associated with GC excess, with particular relevance to the development of depressive symptoms.1

Psychopathological alterations

From a clinical perspective, it is well established that overt endogenous hypercortisolism induces psychiatric and neurocognitive impairment in the vast majority of cases, affecting patients’ daily lives and well-being. The psychopathological alterations in Cushing’s syndrome mainly include depression and anxiety, although other symptoms and disturbances have been reported, including emotional lability, reduced libido, irritability, insomnia and maladaptive personality traits.

In Cushing’s syndrome, the most frequently reported alterations to cognitive functions are memory impairment and reduced concentration in at least two-thirds of patients.

Similarly, there has been evidence of early low degree of cortisol excess may lead to psychological and neurocognitive impairment.2,3

Importantly, whether remission of Cushing’s syndrome can resolve psychiatric and neurocognitive disorders is debatable, as results suggest that complete remission is rare.

Role of the HPA axis

On the other hand, many neuropsychiatric disorders have been associated with increases in hypothalamic–pituitary–adrenal (HPA) axis activity, including most forms of major depression – especially psychotic depression. These states of physiologic or non-neoplastic cortisol excess have been described as ‘pseudo–Cushing’s syndrome’, in which decreases in the sensitivity of the GC receptors may lead to resistance to cortisol negative feedback. Moreover, successful therapy often normalises HPA axis function, while ineffective psychopharmacotherapy is usually associated with persistent cortisol excess.

Clinically, these patients tend to have an impaired low-dose dexamethasone suppression test (LDDST), as well as increased late-night and free urinary cortisol. Psychiatrists have utilised both the LDDST and the dexamethasone–corticotrophin-releasing hormone test to address these conditions and their response to psychopharmacotherapy.

Two sides of the same coin

One could hypothesise that these conditions represent two sides of the same coin, sharing a common origin in prenatal distressors or in daily hassles, as suggested by murine models.4 In contrast to major traumatic life events, daily hassles include all those mild, common types of stressful experience that we face on a daily basis, which have a distinct contribution to our general health and psychological distress.

Indeed, while the stress response is adaptive by nature, the effect of chronic stress, especially during developmental periods, could favour the acquisition of a dysfunctional stress system response (upregulation of HPA axis activity), with lifelong negative effects on endocrine, metabolic and general health.

Epigenetics

Interestingly in this regard, results in recent years have shown a possible relationship between the physiological stress response and epigenetic changes in the genetic loci related to the stress system.

It is known that epigenetic changes can modulate gene activity under environmental influences and that such changes occur in early life but can last throughout the lifespan. To date, one of the most studied epigenetic changes is DNA methylation. Research found that this can modify the GC receptor gene (NR3C1), which is responsible for activation of GC-responsive elements in the nucleus and is therefore a key mediator of GC actions on cells. In a recent clinical study on a cohort of 101 young adolescents, it was found that higher levels of DNA methylation in the NR3C1 gene (together with higher daily stress rates) were associated with both lower reactivity of the HPA axis to stressors and longer ‘cortisol stress recovery’, reflecting an overall lower capacity to adapt to external stressors in these subjects.5

In conclusion

Both overt and low degrees of cortisol excess are linked to higher chances of neurological and psychological disorders. In turn, ‘subtle’ daily stressors seem to be sufficient to disrupt our adaptive response and change, through epigenetic interference with GC receptor functions, our cortisol homeostasis, and ultimately lower our ability to deal with stress.

Endocrinologists, more than other specialists, need to raise awareness of such a relationship and recognise the biological burden of psychological distress on patients, pointing research efforts towards better understanding of the complex underlying mechanisms involved and developing efficient management strategies on the clinical side.

Valentina Morelli and Walter Vena, Italy

REFERENCES

Mood disorders and metabolic diseases

The prevalence of anxiety and depression in the general population has been further amplified by obesity and diabetes mellitus, particularly in females. Individuals with obesity have an approximately 55% higher chance of developing depression. Similarly, patients with type 2 diabetes (T2DM) are estimated to have double the incidence of depression, and those with type 1 diabetes (T1DM) have a threefold higher incidence. The prevalence of anxiety in patients with T2DM ranges from 13 to 38%, whereas obesity is associated with an approximate 25% increase in the odds of anxiety disorders.

In fact, the famous Latin expression ‘mens sana in corpore sano’ (a healthy mind in a healthy body) has never been more appropriate in this context.

An interaction between mind and body

There is a close interaction between the effects of chronic psychological alterations on metabolic processes and the consequences of poor lifestyle choices, usually expressed as overeating and physical inactivity, for mental health. Classical clinical expression of anxiety and depression, in particular in the atypical subtype more than the melancholic subtype, is often represented by alterations in appetite, food choices, immune response and sleep, all factors that dramatically affect body weight. Several mechanisms have been hypothesised as acting directly or indirectly in inducing the bidirectional link between increased body weight and altered mood.

Body weight control is tightly regulated by genes that are mostly expressed at the level of the brain. Genome-wide association studies have confirmed that depression-associated loci partially overlap with some obesity traits, although it seems that there are no common genetic factors accounting for the association between T2DM and depression.

Dietary fat and sugar overload are hallmarks of obesity and, at least in rodents, a clear association has been demonstrated between obesogenic diets and anxiety- and depressive-like behaviour. Moreover, an excessive intake of carbohydrates and saturated fats tends to generate the typical chronic, low-grade inflammation that characterises obesity, particularly the abdominal phenotype. The dysfunctional adipose tissue located at the visceral level clearly plays an important role in exacerbating inflammation. It is also worth highlighting that visceral adiposity is a better predictor of the risk of anxiety and depression than is body mass index. Chronic inflammation and visceral adiposity may also play a role in hypothalamic–pituitary–adrenal (HPA) axis dysfunction, which is one of the most consistent findings in biological psychiatry. On the other hand, chronic stress that induces heightened HPA activation may stimulate the chronic consumption of calorie-rich foods, thus favouring visceral adiposity and the subsequent vulnerability to psychiatric disorders. An association between depression (but not anxiety) and abdominal obesity and cardiovascular disease has also been detected in T2DM.

Changes in the brain

Intriguingly, the peripheral hyperimmune chronic response that characterises obesity and T2DM may extend its action to the central nervous system, inducing profound structural and functional changes in the brain, particularly in terms of neuronal excitability, connectivity and plasticity. Microglia activation represents the typical neuroinflammatory process that may contribute to generating depressive- and anxiety-like behaviours. Long term exposure to glial reactivity may thus profoundly impair the homeostatic and hedonic control of food intake, but also may extensively influence and modify neural circuits controlling mood and emotion. Many other neurotransmitter and hormone perturbations characterise patients with obesity and mood alterations. A hypodopaminergic tone is a distinct characteristic of some patients with obesity. On the other hand, anhedonia, which is a significant symptom of depression, is also characterised by a reduced dopaminergic signal at the level of the mesolimbic system. Leptin and insulin resistance associated with obesity and diabetes not only reduce the signalling of these two hormones at the peripheral level but, importantly, dysregulate their message at the level of the brain. At least in animals, central leptin administration is known to induce antidepressant-like effects, whereas the mechanism by which insulin exerts antidepressant-like and procognitive action is still under debate.

Treatment strategies

Due to the close association between metabolic diseases, such as obesity and diabetes, and mood disorders, such as depression and anxiety, treatment strategies should target the shared mechanisms described above. However, the pharmacotherapy used to treat depression is not always neutral, and great attention is needed to try and avoid psychotropic compounds which may increase body weight. Conversely, there is much evidence that body weight reduction obtained by any means is associated with a net amelioration in mood disturbances. In fact, cognitive behavioral therapy, collaborative care and health education have shown positive results for the treatment of depression and anxiety in patients with obesity and diabetes. Great attention has recently been given to the successful use of naltrexone combined with bupropion, not only as an effective anti-obesity treatment, but also to improve anxiety expressed as a binge-eating disorder. This combination also seems to be effective in promoting weight loss in patients with depression and obesity, regardless of antidepressant use. In view of the ability of glucagon-like peptide–1 (GLP–1) analogues to improve brain glucose metabolism and glucose transport across the blood–brain barrier in Alzheimer’s disease (thus alleviating depressive symptoms), future research could test the various combinations of GLP–1 analogues and other incretins aimed at ameliorating mood symptoms together, with the already well known important effect of reducing body weight.

Uberto Pagotto
Italy

‘Several mechanisms have been hypothesised as acting directly or indirectly in inducing the bidirectional link between increased body weight and altered mood.’

FURTHER READING
- Milaneschi et al. 2019 Molecular Psychiatry 24 18–33 https://doi.org/10.1038/s41386-018-0057-5.
Psychoneuroendocrinology and the thyroid

During development, thyroid hormones have been shown to affect neuronal proliferation, migration, differentiation and synapse formation.

In the adult brain, they interact with glial cells that modulate immune responses, regulate neurotransmitter release and control neurone metabolism. Additionally, they are important for modulation of dopaminergic, serotonergic, glutamatergic and GABAergic networks.

Several studies have suggested that thyroid dysfunction (both hyper- and hypothyroidism, including variations in the normal range) increases the risk of depression, its severity and the likelihood of resistance to treatment. It has been reported that the hypothalamic–pituitary–thyroid axis is altered in patients with major depression disorder (MDD) and suicidal behaviours, as illustrated by a blunted thyrotrophin (TSH) response to TSH-releasing hormone stimulation, a decreased amplitude of the nocturnal TSH surge, and slight elevation of serum thyroxine (T4).

In addition, a relevant pathogenetic mechanism relating thyroid dysfunction to MDD suggests that the latter could be attributed to a state of local cerebral hypothyroidism with normal peripheral thyroid hormone concentrations. This hypothesis, referred to as ‘brain hypothyroidism’, is mainly based on the observation of type 2 deiodinase inhibition in the brain, and of impaired T4 transport across the blood–brain barrier, in depressed patients. However, it is not clear whether the observed associations are causal, as observational studies are often prone to selection bias, residual confounding and reverse causality, and large and well-planned studies have failed to find a relationship between MDD and thyroid dysfunction.

Hypothyroidism, even in its milder forms, has also been associated with treatment-resistant depression (TRD), leading to studies that evaluated the effect of tri-iodothyronine (T3) and/or T4 administration as an add-on to MDD therapeutic regimens, with some showing that adding T3 to treatment in MDD seems to have some effect in TRD and, additionally, that T3 seems to be superior to T4. Nevertheless, the effectiveness of thyroid hormone adjunctive therapy in MDD is, to date, not extensively documented, and there is insufficient evidence to support the use of thyroid hormones in TRD.

Further research is needed to strengthen the link between thyroid dysfunction and depression, and to establish proper therapeutic guidelines.

Sandra Belo
Portugal

REFERENCES


Living with androgen deprivation

People say to me, ‘At least prostate cancer is the good one to get.’ When I tell them that the treatment for late stage prostate cancer includes chemical castration, they usually go pale and can’t decide what to say!

When you then go on to tell them about the horrendous side effects associated with androgen deprivation therapy (ADT), the narrative starts to change.

Let’s be clear, no cancer is a ‘good cancer’ to get. If you are diagnosed with stage 4, incurable prostate cancer – as I was in 2017 – you face massive changes to every aspect of everyday life, as well as the prospect of a premature death, assuming that something else doesn’t get you first.

Let’s just spend a little time considering the side effects. Weight gain, reduced muscle mass and fatigue impact your ability to perform normal functions but, in my case, I went from being a sub-elite athlete to a back-of-the-pack plodder. There were days when I’d go for a run and realise that the fatigue was so great that I had to stop and return to base, usually in floods of tears, because cancer had robbed me of something I loved.

Then you have to deal with lack of libido and loss of sexual function. Now I wasn’t exactly a stud, but you don’t expect your sex life to be ended at 60! Inevitably you go to a dark place, and I spent the first 18 months thinking way too much about dying and not enough about living. The anxiety led to depression and, eventually, a breakdown, for which I required counselling. That helped enormously and, since then, I’ve made a point of living every day to the full.

Every 12 weeks there’s the spectre of a prostate specific antigen test to check that the treatment is still working, which massively ramps up the levels of anxiety.

‘Every 12 weeks there’s the spectre of a prostate specific antigen test to check that the treatment is still working, which massively ramps up the levels of anxiety.’

Tony Collier
UK

My wife and I were lucky to be able to access psychosexual therapy, which was a godsend. I learned that ADT may remove drive but it doesn’t remove desire! It is shameful that men can’t access this as the norm.

Ultimately, I have learned that you shouldn’t lose the joy of living through the fear of dying!
Mental impact of the menopause

The perimenopausal transition represents a window of vulnerability for developing depressive symptoms,\(^1\) presenting in combination with hot flashes and night sweats.\(^2\)

Risk factors for perimenopausal mood disorders include premenstrual symptoms, nulliparity, separation from a prior spouse, and smoking.\(^3\)

Perimenopausal women tend to experience irritability, poor concentration, impaired memory, difficulties in multitasking, loss of interest and enjoyment, and mood swings.\(^4\)

**Mood disorders**

Mood disorders in ageing individuals are closely related to neuroendocrine alterations in the central nervous system (CNS) (Figure).\(^1,3\)

Women who experienced a rapid increase in levels of follicle-stimulating hormone (FSH) levels before the final menstrual period have a lower risk of perimenopausal depression in comparison with women with a slower increase in FSH levels. Moreover, the availability of noradrenaline and serotonin at the synaptic level is reduced, secondary to oestrogen deficiency, further predisposing to mood disorders.

In addition, menopause-related depressive symptoms have been associated with fluctuations in allopregnanolone. This progesterone-derived neurosteroid regulates the GABA receptor. The inability to maintain the control of GABAergic homeostasis, secondary to the alternating hormonal environment, leads to a dysregulated response of the hypothalamus–pituitary–adrenal axis to stress. These changes increase the vulnerability to depression during midlife, particularly in women with a history of postpartum depression or premenstrual dysphoric disorder.\(^1,3\)

**Hot flashes and night sweats**

Climacteric symptoms, namely hot flashes and night sweats, overlap with depressive symptomatology (Figure). At the same time, they are also known to modify the risk of future depressive symptoms in women with prior history of depression.

The neuroendocrine pathophysiology of hot flashes has received significant attention lately. The hypothalamic neurones of the infundibular nucleus (KNDy neurones) produce neuropeptides such as kisspeptin, neurokinin B (NKB) and dynorphin, and also express oestrogen receptors. The KNDy neurones regulate the function of the gonadotrophin-releasing hormone (GnRH) pulse generator and project to the preoptic thermoregulatory area. Ageing and oestrogen fluctuation lead to hypertrophy of the neurones in the infundibular nucleus and, later on, to reduced expression of the dynorphin gene and increased expression of the genes for kisspeptin and NKB. These genomic changes lead to increased signalling to GnRH neurones and to heat dissipation effectors in the CNS. The KNDy neurones are also thought to regulate body temperature, playing a role in the emergence of vasomotor symptoms.\(^1\)

**Treatment options**

The choice of treatment is related mainly to the severity of the symptoms. Counselling and psychoeducation are required, while the treatment plan should be based on shared decision making.\(^2\)

In cases with mild symptoms, primary care physicians are encouraged to adopt watchful waiting, with a low intervention threshold, reassessing the patient at 2 weekly intervals. In cases with persistent or worsening symptoms, further treatment is indicated, which usually consists of psychotherapy or pharmacotherapy, perhaps including menopause hormone replacement therapy (MHT). An imminent referral for psychotherapy or pharmacotherapy is encouraged in cases with moderate or severe symptoms. However, MHT initiation is not beneficial in cases with severe depression. The next step consists of starting the treatment with weekly monitoring and clinical re-evaluation after 3–4 weeks. The treatment can be continued if the symptoms improve by more than 50%. If the symptoms do not improve significantly, the treatment should be adjusted, and regular monitoring is required (every 1–2 weeks).\(^2\)

**Pharmacotherapy**

Therapeutic alternatives vary from regular antidepressants to psychoeducation and MHT. Pharmacotherapy includes options as follows:\(^2\)

a) Selective serotonin reuptake inhibitors (citalopram: starting dose 20mg/day, usual dose 20–40mg/day; escitalopram: starting dose 10mg, usual dose 10–20mg/day; fluoxetine: starting dose 20mg/day, usual dose 20–60mg/day).

b) Serotonin–norepinephrine reuptake inhibitors (desvenlafaxine: starting dose 25–50mg/day, usual dose 50–100mg/day; venlafaxine: starting dose 37.5–75mg/day, usual dose 75–375mg/day; duloxetine: starting dose 30–60mg/day, usual dose 60–120mg/day).

c) Other options include selective norepinephrine–dopamine reuptake inhibitors (e.g. bupropion), α1-receptor antagonists (e.g. mirzapapine), serotonin modulators, tricyclics, and tetracyclics (e.g. amitriptyline).

**Menopause hormone replacement therapy**

The use of MHT in peri- and postmenopausal women with depressive symptomatology remains inconclusive.\(^2\) MHT is not approved for the pure treatment of perimenopausal depression. However, MHT is an effective option for controlling the severity of vasomotor symptomatology and other menopause-related complaints. Hence a positive effect on negative mood is anticipated.\(^2\) On the other hand, recent data\(^5\) described that regular intake of antidepressants could impact serum levels of sex hormones by dysregulating the function of the hypothalamus–pituitary–adrenal axis and, consequently, the female response to triggers of anxiety and stress. The precise direction of this interaction and the related clinical implications are still to be investigated by future studies.\(^5\)

**In summary**

The use of antidepressants with or without psychoeducation and MHT has proven beneficial in controlling mood disorders. However, the administration of MHT mono-therapy to regulate mood disorders is not indicated.

**Eleni Armeni**

UK

**REFERENCES**

Psychological distress and infertility

Infertility is a challenging condition that affects millions of couples around the world. Besides the physical and financial burdens associated with infertility, it can also negatively impact the mental health and quality of life of those experiencing it.¹

Couples living with infertility still experience stress, even though it is not a life-threatening condition. There is a complex relationship between infertility and psychological stress. Among the emotional factors that can affect infertile couples are the stress of infertility, the burden of fertility treatments, and the uncertainty of the outcome. Many studies have found that subfertile couples have lower physical, mental and emotional health compared with the general population. Additionally, uncertainty about fertility treatment outcomes can exacerbate feelings of stress and anxiety, leading to depression and anxiety disorders.²

For some individuals, the experience of infertility can trigger feelings of loss and grief, particularly if they have experienced pregnancy miscarriage or failed fertility treatments. For others, the financial burden of fertility treatments can be a significant source of stress. In some cases, the social stigma associated with infertility can also contribute to psychological distress. Moreover, in addition to the psychological side effects that may occur from infertility itself, a range of other side effects can be caused by hormones and drugs used to treat infertility.³

On the other hand, strong psychological distress could increase infertility. Women’s emotional stress can be indicated as tubal spasms, anovulation and vaginismus. In addition, during ovulation, women can unintentionally avoid sexual intercourse. In men, higher levels of stress are related to sexual dysfunction, and erectile and/or ejaculatory disorders.³

In conclusion, subfertility can significantly impact the quality of life of affected couples. It is a challenging condition and psychological counselling could be proposed upfront, before any medical intervention. However, the outcomes of psychological and educational interventions on mental health and live birth or pregnancy rates are still uncertain, due to the very low quality of the evidence. There is a need for further studies to explain the exact role of mental disorders in fertility and the contribution they have to infertility.

Settimio D’Andrea
Italy

REFERENCES
Impact of behavioural modification in women with PCOS and obesity

A randomised controlled trial aimed to evaluate the effect of behavioural modification on psychological well-being and weight loss in overweight women with polycystic ovary syndrome (PCOS), a group in which psychological well-being is known to be severely affected. Oberg and colleagues showed that behavioural modification can positively impact dimensions of well-being, and that personality factors could contribute to the understanding of successful weight loss.

See European Journal of Endocrinology 2020 183 1−11 https://doi.org/10.1530/EJE-20-0066

Cardiometabolic and psychological effects of dual-release hydrocortisone

In this prospective trial, Dineen et al. treated 51 patients with adrenal insufficiency with dual-release hydrocortisone (DR-HC). After 12 weeks, they were reassessed. Administration of DR-HC was found to decrease blood pressure, weight and body mass index compared with conventional hydrocortisone treatment. Additionally, the group receiving DR-HC showed significant improvements in quality of life compared with those treated with immediate-release hydrocortisone.

See European Journal of Endocrinology 2021 184 253−265 https://doi.org/10.1530/EJE-20-0642

Fatigue and quality of life among thyroid cancer survivors

Maki and colleagues studied the prevalence of fatigue in papillary thyroid cancer survivors without persistent or recurrent disease. About 40% of the 292 thyroid cancer survivors who were enrolled reported fatigue, measured using the cancer fatigue scale. A higher prevalence of fatigue correlated with poor quality of life. Interestingly, free tri-iodothyronine levels correlated with fatigue score and quality of life score.

See Endocrine Connections 2022 11 e210506 https://doi.org/10.1530/EC-21-0506

Mindfulness affects stress, ghrelin and BMI in childhood obesity

In this trial by López-Alarcón and coworkers to assess the efficacy of mindfulness in childhood obesity, participants were assigned to receive a conventional nutritional intervention with or without mindfulness-based intervention. After 8 weeks, children in mindfulness therapy showed a significant reduction in body mass index (BMI), fat mass, anxiety score, ghrelin and cortisol serum levels. Those studied for 16 weeks showed significantly decreased BMI after intervention, which remained lower 8 weeks later. The authors concluded that mindfulness is an adjunctive tool in therapy for childhood obesity.

See Endocrine Connections 2020 9 163−172 https://doi.org/10.1530/EC-19-0461

Changes in serum cortisol and ghrelin were statistically different between groups. CNI, conventional nutritional intervention; MND, mindfulness. Reproduced under CC BY-NC 4.0 licence (https://creativecommons.org/licenses/by-nc/4.0) from López-Alarcon et al. (details above) ©2020 The Authors.

Health status of young people with CAH

Bacila and coworkers conducted this study involving 14 tertiary endocrine centres in the UK. It included 101 patients aged 8−18 years with classic 21-hydroxylase deficiency and 83 controls. The authors demonstrated growth deficiency and weight gain in children with congenital adrenal hyperplasia (CAH) that affected the psychological well-being of these young patients.

See European Journal of Endocrinology 2022 187 543−553 https://doi.org/10.1530/EJE-21-1109

‘A higher prevalence of fatigue correlated with poor quality of life [in papillary thyroid cancer survivors]. Interestingly, free tri-iodothyronine levels correlated with fatigue score and quality of life score.’
How to write a guideline

In this article, we shed light on the process behind the creation of ESE Clinical Practice Guidelines, from formulating clinical questions to developing clinical recommendations.

ESE has a Clinical Practice Guideline programme, aiming to publish at least one (revised) guideline yearly. The first ESE guideline was published in 2015; the first revision of an ESE guideline (adrenal incidentalomas) has recently been published. The guidelines are widely read: the paper concerning adrenal incidentalomas was downloaded 21,330 times in a year.

Workflow

ESE’s Clinical Practice Guidelines aim to provide recommendations for patient care for specified endocrine conditions (see www.ese-hormones.org/publications/guidelines/about-our-guidelines). Guideline topics are directed by the ESE Clinical Committee, with input from ESE members. The topics ideally address areas not previously covered by other influential endocrine societies/specialty organisations, or areas where available recommendations are not applicable to the European standard of care.

A dedicated working group, consisting of experts in the field and supported by a methodologist, is appointed for each guideline by the Clinical Committee. First drafts of guidelines are reviewed by ESE members and relevant parties (such as patient support groups and other specialist societies) before publication, and are presented during the Society’s annual Congress (ECE). Where considered beneficial, patient leaflets are prepared alongside the guidelines, to provide patients with assistance in understanding and managing their condition.

Methodological process

The guidelines use GRADE (Grading of Recommendations Assessment, Development and Evaluation) as a methodological base. This is a systematic approach for synthesising evidence and grading of recommendations.

The first methodological step for the working group is defining clinical questions to be addressed in the guideline. These clinical questions form the basis of a systematic literature search. An example of such a clinical question is: which biochemical test predicts clinically relevant adrenal incidentalomas?

After the search is performed and relevant articles are included, evidence is synthesised, for example by estimating the average effect for a specific outcome. Also, the quality of the evidence is graded in a standardised manner using GRADE, which results in a summary rating across studies for each outcome. Four categories are used: high, moderate, low and very low quality.

The quality of evidence forms a core element of the final recommendations. The second element is the balance between desirable and undesirable outcomes, the third element is values and preferences (patient preferences, goals for health, costs etc.). For example, a specific test or imaging modality might have the best diagnostic performance. However, if the test is very expensive or not widely available, the guideline may not recommend it as first choice.

Consensus on final recommendations is reached upon discussion within the working group. Minority positions are taken into account in the rationale behind recommendations.

The final recommendations are worded as recommend (strong recommendation) or suggest (weak recommendation). For a strong recommendation, it can be stated that reasonably informed persons (clinicians, politicians and patients) would act in accordance with the recommendation, whereas for a weak recommendation most persons would still act in accordance with the guideline, but a substantial number would not. The quality of evidence behind the recommendations is classified as very low (○○○○), low (○○○), moderate (○○), and strong (○○○○○).

When there is no high quality evidence

In an ideal setting, guidelines are based on evidence from randomised trials and accompanied by strong recommendations. Unfortunately, for many areas in endocrinology, high quality evidence is scarce or even non-existent. Another scenario is that high quality evidence is available, but does not translate into a strong recommendation, for instance because the outcome under study is judged as not important. Hence, standardised quality judgement still does not eliminate the need for clinical judgement.

These limitations in evidence challenge a guideline working group. However, recommendations still have to be made, based on the simple fact that treatment decisions for patients have to be made. The lack of solid evidence and the fact that the recommendation concerned is based on good clinical practice are emphasised in the guideline. Of note, good practice recommendations are not graded, since the lack of apparent evidence might wrongly suggest the lack of a good rationale for clinical practice.

One example is that it is good clinical practice to treat patients with Addisonian crises with i.v. glucocorticoids, even though there are no randomised controlled trials showing its superiority over placebo. In addition, all ESE guidelines incorporate a paragraph with uncovered gaps in clinical knowledge and specified suggestions for future research.

In summary

ESE’s Clinical Practice Guidelines are produced through a standardised process; the methodological part is based on GRADE. Lack of (high quality) evidence does not automatically mean lack of clinical recommendations, but merely uncovers literature gaps, leading to (ungraded) good practice recommendations and directing future research.

Leonie T van Hulsteijn and Olaf M Dekkers
The Netherlands

Leonie van Hulsteijn is a post-doc who works for ESE in the role of Clinical Guideline Methodologist.

You can access ESE’s Clinical Practice Guidelines at www.ese-hormones.org/publications/guidelines

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Observership success stories

EYES Research Observership grants were awarded for the first time in 2022. Narjes Nasiri Ansari and Fabio Bioletto are two of the seven people to first benefit from this new programme. Here, they tell us about their experience.

NARJES NASIRI ANSARI
Greece

I chose to visit the Genetics, Reproduction and Development Institute (iGReD) in Clermont-Ferrand, France. Dr Pierre Val was very welcoming; his team performs high quality research on an interesting, high impact subject. He introduced me to all his lab members and collaborators and provided me with material regarding the lab as well as ongoing projects. He was always around if I had any questions, even when I asked him for some professional advice.

During my stay, I felt extremely supported, both scientifically and socially, by Dr Diana Garcia Garcia (Dr Val’s current Post-Doctoral fellow). She spent a tremendous amount of effort and time to train me on how to use ‘R studio’ for single cell RNA-Seq data analysis.

During my stay in the CRNS, I had the opportunity to see research from a more professional point of view and challenge myself in new ways. This short Observership helped me to acquire new knowledge of research methodologies and disciplines, which will add value to my career.

From a cultural point of view, during my time in Clermont-Ferrand, I had the chance to visit some small cities, and gain new knowledge of French history, architecture and cuisine. I made some friends that I would love to keep for a lifetime.

I believe the EYES Research Observership Programme provides young scientists with a unique opportunity to expand their network and gain new knowledge in their field of interest. I thank Dr Val and the EYES Committee for this enjoyable experience.

FABIO BIOLETTO
Italy

I spent my EYES Research Observership at Leiden University Medical Center in The Netherlands. During my time there, I had the privilege of collaborating with a group of brilliant researchers on a project about radiomic analyses in non-functioning pituitary adenomas. This turned out to be a truly enriching experience.

At the beginning of my stay, I was warmly welcomed by the faculty and colleagues. The research team I joined was diverse and included individuals with several areas of expertise. This made for a very dynamic and stimulating work environment. Throughout the project, we worked closely together, with frequent meetings and discussions. Additionally, we were able to collaborate with other departments, covering areas such as epidemiology and statistics, which gave us a more comprehensive understanding of the whole project.

I was most impressed by the level of organisation and the state-of-the-art facilities available at the university, which allowed our work to be more efficient.

Another highlight of my experience was the cultural exchange. During my stay, I was able to immerse myself in the local culture and make new friends. It was fascinating to learn about the customs and traditions of the country and share my own culture with the people I met.

Overall, my experience as a visiting researcher was unforgettable. I was able to contribute to ground-breaking research, collaborate with amazing people and immerse myself in a new culture. It was a truly rewarding experience that I will always treasure.

2023 recipients

We are delighted to announce the following recipients of the EYES Clinical and Research Observership Programmes for 2023.

ROP awardees
Onur Elbasan (Germany)
Dorota Filipowicz (UK)
Faheem Seedat (The Netherlands)

plus nine self-funded awardees: Ayça Hacioglu (Italy), Baris Karagun (France), Müge Keskin (Switzerland), Carmen Lambert (Italy), Ana Milošević (Italy), Roberto Novizio (Spain), Katarzyna Paczkowska (UK), Patricia Vaduva (Germany) and Rodanthi Vamvoukaki (Italy).

COP awardees
Anastasia Armeni (Italy)
Alba Hernández Lázaro (Italy)
Zvezdana Jemuovic (Serbia)

plus ten self-funded awardees: Ioanna Balla (Germany), Ilaria Bonaventura (Germany), Leonardo Dalla Valentina (Greece), Simona Gabriela Duta-Ion (Italy), Ketevan Lomidze (Italy), Mia Manojlovic (Serbia), Maria Mathiopoulou (Greece), Ana Maria Moyano Sanchez (Italy), Iulia Alexandra Pirga (Serbia) and Zeliha Yarar (Croatia).

Find out more and apply

Applications for the 2024 round of the EYES Clinical and Research Observership Programmes will open later in the year. You can find out more at www.ese-hormones.org/eyes-cop-and-rop
Making memories at ECE 2023!

As the 25th European Congress of Endocrinology came to a close in Istanbul, Turkey, it left us with a plethora of cherished memories and valuable experiences.

Set in the breath-taking Haliç Congress Center, with panoramic views of the Golden Horn – a natural estuary connecting with the Bosphorus Strait and the Sea of Marmara – the event was a feast for the senses.

Scientific excellence was at the forefront of the Congress, with the highly anticipated EYES Symposium shining brightly! We extend our sincere gratitude to ESE and the ECE 2023 Programme Organising Committee for providing a platform upon which young researchers from around the world could showcase their cutting-edge work.

This year’s EYES Symposium, entitled ‘Feeding the endocrine-related cancers: weight matters’, featured the following insightful talks:

- **James Wilmouth Jr** (France), Ablation of Znrf3 and Trp53 induces metastatic adrenocortical carcinoma in mice
- **André Sarmento-Cabral** (Spain), Obesity fuels prostate cancer: a source of diagnostic biomarkers and therapeutic targets
- **Ludovica Verde** (Italy), Time for food: also for endocrine-related tumours?

The oral and poster communication sessions provided further clear examples of the high scientific quality brought to our Congress by early career investigators, reaffirming a promising future for endocrinology.

Beyond the scientific discussions, ECE 2023 was an opportunity for attendees to reconnect with colleagues as well as experts in the field, and to forge new friendships. The EYES social event held at the Populist Bomontiada (a ‘brewery pub’) was a resounding success, with young endocrinologists and scientists (and others who are ‘young at heart’) gathering to enjoy each other’s company, lively music and delightful refreshments. It was heartening to witness the exchange of ideas and experiences in a relaxed and friendly atmosphere, including engaging conversations with esteemed figures such as the new ESE President, Jérôme Bertherat.

We congratulate the recipients of the ESE Young Investigator Awards: Elena Armeni (Greece), María Claro (Spain), Menic Coskun (Turkey), Antonio García-Estrada (Spain), Anne Jouinot (France), Yasmine Kemkem (UK), Punith Kempegowda (UK), Ewa Młyczynska (Poland), Sophie Monnerat (Switzerland), Saroj Sehoio (India), Soham Tarafdar (India) and David Veríssimo (Portugal). Your exceptional work has earned well-deserved recognition!

As we look back on ECE 2023, anticipation of ECE 2024 in Stockholm, Sweden, is already building. Join us on 11–14 May next year, as we begin to prepare the EYES Symposium for another exciting journey in the field of endocrinology. See you there!

Juan Manuel Jiménez-Vacas
UK
At ECE 2023, Jérôme Bertherat became President of ESE. Jérôme is Professor of Endocrinology at Paris Descartes University, Chief of the Endocrinology Department of Cochin Hospital, Head of the National Center for Rare Adrenal Diseases and leads a research team on genomics and signalling of endocrine tumours in the Cochin Institute. We look forward to working with him in his new role. We thank Martin Reincke for his support and leadership over the last 2 years.

**Other changes at ECE 2023**

We welcomed Wiebke Arlt (UK) as ESE’s new President-Elect at ECE 2023, as well as Eleanor Davies (UK) as Science Committee Chair and Sebastian Neggers (The Netherlands) as Rare Disease Committee Chair. Thanks are due to Martin Fassnacht and Simona Glasberg, who are standing down having completed their terms of office.

**A date for hormones**

European Hormone Day aims to build awareness of hormones in health and disease. The 2023 event took place during ECE in Istanbul. It was organised by ESE and the European Hormone and Metabolism Foundation (ESE Foundation).

The day included the launch of the guide *10 Recommendations for Good Hormone Health*, highlighting easy ways for people to improve hormone health. Could you distribute or display a poster to promote the guidance? See [www.ese-hormones.org/media/5057/10-recommendations-infographic-a2-landscape-v01.pdf](http://www.ese-hormones.org/media/5057/10-recommendations-infographic-a2-landscape-v01.pdf).

Please also circulate the leaflet *Because Hormones Matter*, which was published for European Hormone Day and lists awareness-raising initiatives, including a printable calendar of upcoming endocrine-related events: [www.ese-hormones.org/media/5085/awareness-raising-brochure-web-final.pdf](http://www.ese-hormones.org/media/5085/awareness-raising-brochure-web-final.pdf).

**Board exam goes virtual**

This year’s European Board Examination in Endocrinology, Diabetes and Metabolism takes place on 8 November. It will be held virtually, using remote invigilation. You will be able to register in August. Find out more, including grant application information, at [www.ese-hormones.org/education/european-board-examination](http://www.ese-hormones.org/education/european-board-examination).

**Get ready for EndoBridge**

The year’s EndoBridge meeting takes place on 19–22 October in Antalya, Turkey. It includes 3 days of lectures by experts and interactive sessions discussing interesting clinical cases across the breadth of endocrinology. You can submit clinical cases until 25 August. Full details of the programme and online registration are at [www.endobridge.org](http://www.endobridge.org).
Just say YES!

The YES (Young ESPE) Group is a group for early career members of the European Society for Paediatric Endocrinology (ESPE). The Group was launched at the 60th ESPE Annual Meeting in Rome, Italy, in September 2022.

Why say ‘YES’?

Our primary purpose is increasing the visibility and involvement of early career paediatric endocrinologists within ESPE. We aim to create a community to help develop and promote dedicated activities, to maximise the direct participation of YES members in the Society, including collaboration with various ESPE Committees. Being a YES member can provide you with an opportunity to participate in different ESPE research projects, reports and publications.

We conducted a survey to better understand the needs of YES members and the ways in which they wish to engage with and contribute to ESPE. The results enforced our primary belief that people in their early careers need support, orientation and guidance on how to get involved in activities within the Society and build experience in clinical and research fields.

Educational programmes

ESPE has a long history of running different educational programmes, schools and fellowships for early career paediatric endocrinologists from around the world, giving equal opportunities for education to all. One of our goals is to be involved in ESPE Schools as mentors, assisting the faculty and serving as a bridge between teachers and students.

Our first successful project was a webinar entitled ‘How to publish a scientific paper’ with Professor Stefano Cianfarani (Editor-in-Chief of Hormone Research in Paediatrics), which was moderated by Dr Hussain Alsaffar, one of the YES leaders. Currently, we are conducting a collaborative study with the JENIOUS Group (the early career group of the International Society for Pediatric and Adolescent Diabetes, ISPAD). We are sure this will be the first of many future collaborations with similar early career groups from other endocrine societies.

Overcoming early career challenges

As a very broad field, with many opportunities for clinical and research work, paediatric endocrinology can throw you ‘in a loop’ when it comes to setting a path for your career. We believe colleagues who are just starting their journey would mostly benefit from experienced guidance. Coming from all parts of the world, we understand and can relate to the needs of all our members.

Young physicians starting their journeys are often hesitant to immerse themselves in societies or to make a contribution. This is usually because of feeling too ‘green’ and inadequate. The YES Group understands that this silence on the part of young colleagues leaves both them and the Society weakened rather than stronger − and many potentially great ideas consequently don’t see the light of day. It is by empowering up and coming generations that we will provide growth for both them and for ESPE as a whole.

‘Our primary purpose is increasing the visibility and involvement of early career paediatric endocrinologists within ESPE.’

Who is behind the YES Group?

The group is managed by six YES leaders from around the world:

Sommayya Aftab, Pakistan
Hussain Alsaffar, Oman
Domenico Corica, Italy
Katja Dumić Kubat, Croatia
Meera Shaunak, UK
Rade Vukovic, Serbia

We are supported by Professor Rasha Hamza, the Chair of the ESPE Education and Training Committee.

Each YES leader is assigned to a different ESPE Committee (e.g. Education and Training, Communication, Clinical Practice, Science Committee etc.) as a YES representative. This provides us with the opportunity to give early career members a voice within ESPE, while also nurturing future key opinion leaders by allowing them to be involved with ESPE from the core of its structure.

The YES group currently has 110 members from 28 different countries, spanning six continents. To find out more, see www.eurospe.org/about/organisation/yes-group.

Katja Dumić Kubat
Croatia
Welcome to Würzburg!

The 10th EYES Meeting (EYES 2023) will take place together with the Young Active Research in Endocrinology (YARE) Annual Meeting in Würzburg, Germany, on 8–10 September 2023.

YARE is the early career branch of the German Society of Endocrinology (Deutsche Gesellschaft für Endokrinologie, DGE). Both groups – EYES and YARE – support early career clinicians and researchers in endocrinology. The EYES–YARE meeting will be a fantastic education and training event in endocrinology, covering the latest basic, translational and clinical research. It will offer you and many other early career endocrinologists the opportunity to present your work in an international context and the chance for you to network and begin collaborations with colleagues from across Europe and beyond.

Discover Bavaria

Würzburg lies in the German state of Bavaria. It is located halfway between Frankfurt and Nürnberg on the banks of the River Main. The city is encircled by vineyards and is one of the most important wine-producing districts, renowned for its white wines. Würzburg is also famous for the Marienberg Fortress, the Old Main Bridge (Alte Mainbrücke) and the Residenz: a UNESCO World Heritage Site. Its diverse museums include the Röntgen Memorial Site, the laboratory where Wilhelm Conrad Röntgen discovered X-rays. The city is home to several festivals, including the largest stage-free street music festival in Europe (STRAMU, the International Festival for Street Art).

Join us in Würzburg, take part in the meeting and enjoy our city!

Barbara Altieri (EYES) and Laura-Sophie Landwehr (YARE) Co-Chairs, on behalf of the Local Organising Committee Eyes2023@ukw.de

Submit your abstracts by 30 July 2023

Register by 15 August 2023
Registration (with hotel and social programme): €150
Find out more at www.eyes-2023.com

All abstracts will be published in Endocrine Abstracts, an open access, citable resource

PLENARY LECTURES
A new name for diabetes insipidus
Mirjam Christ-Crain
Microenvironment in pituitary tumours
Pedro Marques*
Gonadal dysfunction in women with diabetes mellitus
Stavroula A. Paschou
Translational obesity research: from rodents to humans
Ulrich Dischinger
*Representative of EYRC, the European Neuroendocrine Association Young Researchers Committee

ORAL COMMUNICATIONS/ GUIDED POSTER TOURS on:
• Adrenal and NEFs
• Pituitary and Neuroendocrinology (with EYRC)
• Thyroid
• Calcium and Bone
• Diabetes, Obesity and Metabolism
• Interdisciplinary Endocrinology
• Reproductive Endocrinology
• Environment, Society and Governance

SOCIAL EVENTS
River trip, including dinner (8 September)
Biergarten Dornheim (9 September)