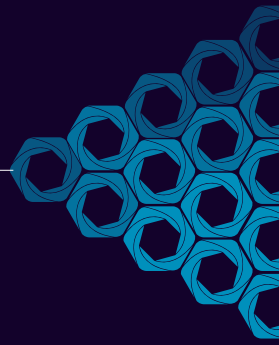


ESE Recommended Curriculum of Specialisation in **Clinical Endocrinology, Diabetes and Metabolism**

At the centre of Europe's endocrine community



This document was produced in collaboration with the ESE Council of Affiliated Societies (ECAS).

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The ESE Recommended Curriculum of Specialisation in Clinical Endocrinology, Diabetes and Metabolism has received official endorsement from:



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1. Introduction

The European Society of Endocrinology (ESE) was founded in 2006 to promote research, education and clinical practice in endocrinology for the benefit of the public. Today, ESE has over 3000 members and the society continues to grow, providing support to endocrinologists throughout Europe and beyond.

In 2013, the ESE Council of Affiliated Societies (ECAS) was established providing a forum for the 47 National Affiliated Societies of ESE to discuss the key issues facing the field of endocrinology and to better understand how ESE can support these national endocrine societies in addressing these.

At the first ECAS meeting, in September 2013, the council members identified the current lack of harmony of education during the specialisation of clinical endocrinologists across Europe as a serious concern. That there are differences in the requirements for trainees specialising as clinical endocrinologists in different countries within Europe is seen as a factor likely to inhibit the development of a more collaborative and cooperative future for clinical endocrinology.

To address this, ESE's Education Committee established the ESE National Curricula Working Group, comprising Jens Bollerslev (Norway), Michal Krsek (Czech Republic), Karim Meeran (UK) and Misa Pfeifer (Slovenia), with the responsibility of creating an ESE Recommended Curriculum of Specialisation in Clinical Endocrinology, Diabetes and Metabolism. This working group reviewed and compared curricula from 15 countries in Europe (Austria, Estonia, Finland, France, Germany, Greece, Israel, Latvia, the Netherlands, Norway, Romania, Spain, Sweden, Switzerland and the UK) to better understand the current situation. Following this review, the Swiss and UK curricula, having been identified as the most comprehensive documents, were used as the backbone for the ESE recommended curricula alongside other recognised sources such as the 10th revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10).

The working group established the key criteria required to practice as a clinical endocrinologist, listing those areas in which an endocrinologist should be expected to be proficient. These areas were then arranged into sections to form a draft of the final curriculum. Consultation with the members of ECAS was carried out before the ESE Recommended Curriculum of Specialisation in Clinical Endocrinology, Diabetes and Metabolism was finalised.

With the creation of this document, ESE provides a resource for trainee endocrinologists to refer to in order to assess their knowledge, skills and behaviour, ensuring they are proficient in all of the required areas to practice with competence throughout Europe. Additionally, this document will enable education providers to monitor their endocrine training programmes, ensuring that they cover adequately their intended subject areas. ESE will use this curriculum in the development of our future Postgraduate Courses in Clinical Endocrinology, Diabetes and Metabolism, ensuring that the entire curriculum is covered over a series of consecutive courses, therefore providing a more established structure to our education programme. With the development of an examination based Certificate in Clinical Endocrinology, Diabetes and Metabolism coming in 2018, ESE hopes to promote harmony in endocrine clinical education, while facilitating the learning process for trainee endocrinologists.

The ESE Recommended Curriculum of Specialisation in Clinical Endocrinology, Diabetes and Metabolism focuses on the knowledge requirements for the clinical treatment of adult endocrine disorders. Paediatric endocrinology, as a separate specialty, is not considered within the scope of this document. The document aims to provide an overview of the expected areas of knowledge and is not intended to provide specific details of disorders or their treatment. This document will be subject to regular review and evaluation and will be modified as required.

Defining Clinical Endocrinology

Clinical Endocrinology is an area often poorly defined, even at times by those within the field. The endocrine system controls many processes within the body, including growth, maturation, metabolism and reproduction. Endocrine disorders are extremely diverse, making a wide understanding crucial to the practicing clinical endocrinologist, who must have a thorough understanding of the diagnosis, prognosis, treatment and care of patients with disorders of the hypothalamus, pituitary, thyroid, parathyroid, pancreatic islets, adrenal glands, testes and ovaries as well as hypertension, nutrition, obesity, osteoporosis and hyperlipidaemia.

ESE, in an effort to provide a more consistent Pan-European understanding of clinical endocrinology, has defined the key areas in which a clinical endocrinologist should be expected to have sufficient knowledge and understanding. This document represents the minimum criteria and local development, using this as a template, is expected. Which particular drugs or treatments are to be used may vary depending on local availability or regulations and should therefore be defined locally. For example, not all areas require the endocrinologist to perform fine needle aspiration of the thyroid, but this technique has been included in the curriculum. Some areas might develop this further and require this technique as an approved procedure. Iodocholesterol scanning (NP59) is not available in many countries, but it is listed here for those countries which do perform this practice.

The aspiring clinical endocrinologist should aim to be able to demonstrate knowledge and understanding of the physiology, epidemiology and pathology, appropriate patient consultation, diagnostic techniques, treatment options and follow-up procedures for each of the endocrine disorders listed in the curriculum.

Similarly, the clinical endocrinologist should in each case know how and be able to correctly diagnose endocrine disorders in patients showing symptoms indicative of the relevant endocrine dysfunction. They should be able to select, and interpret correctly, appropriate diagnostic tests for endocrine dysfunction and explain and provide treatments to, and follow-up procedures for, patients with diagnosed endocrine disorders. They should recognise that some cut off values for dynamic tests have a poor evidence base, and aim to interpret the results in the light of the clinical context. They should also have an understanding of clinical biochemistry, determination of reference ranges, and recognise that investigations have false positive and false negative rates. The clinical endocrinologist should also be able to consider and account for additional complications arising as a result of endocrine dysfunction in the patient.

There is a further requirement that the clinical endocrinologist understands the importance of dynamic testing for endocrine dysfunction. Understanding the interplay between natural cycles and environmentally induced variability of hormone levels is crucial, as is a solid understanding of the

need to dose appropriately hormone replacements to ensure an adequate treatment of the disorder while avoiding the induction of other complications through over-replacement.

An additional section highlights the key diagnostic techniques required routinely for endocrine clinical diagnosis. For each of these techniques the clinician should understand the theory, practical application and interpretation of the data obtained. They should be able to explain these techniques to the patient taking into account the patient's knowledge level and emotional condition.

Endocrinologists should not work in isolation. They should lead and build multidisciplinary teams in order to obtain the best outcome for patients. Such a team may include specialist surgeons or neurosurgeons, oncologists, radiotherapists, specialist nurses and other allied health professionals.

A clinical endocrinologist able to demonstrate the above competency for each of the sections outlined in this curriculum should be confident of their ability to practice clinical endocrinology throughout Europe.

2. Adrenal

- 2.1. Adrenal insufficiency
 - 2.1.1. Addison's disease
 - 2.1.2. Mineralocorticoid deficiency
 - 2.1.3. Unspecified adrenal deficiency
- 2.2. Adrenocortical hyperfunction
 - 2.2.1. Primary aldosteronism
 - 2.2.2. Cushing's syndrome (see also 7.4)
- 2.3. Congenital adrenal hyperplasia and adrenal hyperandrogenism
- 2.4. Adrenal tumours
 - 2.4.1. Adrenal incidentaloma
 - 2.4.2. Adrenal hyperplasia and adenoma
 - 2.4.3. Adrenal cortical and medullary cancer
- 2.5. Adrenal medullar hyperfunction
 - 2.5.1. Adrenomedullary hyperfunction, pheochromocytoma, paraganglioma

3. Diabetes mellitus

- 3.1. Type 1 diabetes
- 3.2. Latent autoimmune diabetes in adults (LADA)
- 3.3. Type 2 diabetes
- 3.4. Gestational diabetes
- 3.5. Maturity onset diabetes of the young (MODY)
- 3.6. Maternally inherited Diabetes and Deafness (MIDD)
- 3.7. Pre-diabetes
- 3.8. Malnutrition-related diabetes mellitus
- 3.9. Age-related conditions and diabetes
 - 3.9.1. Young people
 - 3.9.2. Elderly people
- 3.10. Diagnosis and management of diabetes mellitus
 - 3.10.1. Management of diabetes care
 - 3.10.2. Diabetic emergencies
 - 3.10.3. Management of patients with diabetes during acute illness or surgery
 - 3.10.4. Conception and pregnancy in diabetes
- 3.11. Complications of diabetes
 - 3.11.1. Screening for the complications of diabetes

- 3.11.2. Cardiovascular macrovascular complications
- 3.11.3. Eye disease
- 3.11.4. Renal disease and hypertension
- 3.11.5. Neuropathy and erectile dysfunction
- 3.11.6. Autonomic neurological complications
- 3.11.7. Foot disease
- 3.11.8. Lipid disease
- 3.11.9. Diabetic coma, ketotic and non-ketotic
- 3.11.10. Multiple complications
- 3.12. Hypoglycaemic disorders
 - 3.12.1. Drug-induced hypoglycaemia
 - 3.12.2. Hypoglycaemia unawareness
- 3.13. Disorders of pancreatic internal secretion
 - 3.13.1. Insulinoma
 - 3.13.2. Increased secretion of glucagon
 - 3.13.3. Abnormal secretion of gastrin
- 3.14. Nutrition disorders and management
 - 3.14.1. Functional foods and dietary supplements
 - 3.14.2. Nutrition and metabolic support
 - 3.14.3. Nutritional guidelines for the general population
 - 3.14.4. Nutritional therapy for patients with diabetes, dyslipidaemia, insulin resistance syndrome, and/or cardiovascular disease
- 3.15. Diabetes technology
 - 3.15.1. Appropriate use of insulin pumps
 - 3.15.2. Appropriate use of continuous glucose monitoring

4. Obesity and bariatric endocrinology

- 4.1. Etiology and pathogenesis of obesity
- 4.2. Adipose tissue, metabolism, endocrine functions
- 4.3. Clinical presentation and complications of obesity
- 4.4. Approach to obese patients, examination, measurement of body composition
- 4.5. Treatment of obesity
 - 4.5.1. Diet, psychobehavioral approach and medical approach
 - 4.5.2. Bariatric surgery
 - 4.5.2.1. *Complications to bariatric surgery*

5. Serum lipid disorders

- 5.1. Advanced lipoprotein testing
- 5.2. Genetic lipid disorders
- 5.3. Disorders of high-density lipoprotein cholesterol
- 5.4. Atherogenic dyslipidemia (of insulin resistance and diabetes mellitus)
- 5.5. Hyperlipidemia in solid-organ transplantation
- 5.6. Serum lipid disorders in patients with HIV

6. Parathyroid, calcium and bone

- 6.1. Hypoparathyroidism (HypoPT)
 - 6.1.1. Idiopathic hypoparathyroidism
 - 6.1.2. Post-surgical hypoparathyroidism
 - 6.1.3. Pseudohypoparathyroidism (inactivating PTH/PTHrP signalling disorder' (iPPSD)).
 - 6.1.4. Genetic disorders giving hypoparathyroidism (including Di George syndrome (22.q11 deletion syndrome))
- 6.2. Hyperparathyroidism and other disorders of parathyroid gland
 - 6.2.1. Primary hyperparathyroidism (PHPT)
 - 6.2.2. Familial hypocalciuric hypercalcemia (FHH)
 - 6.2.3. Secondary hyperparathyroidism
 - 6.2.4. Tertiary hyperparathyroidism
 - 6.2.5. Parathyroid carcinoma
 - 6.2.6. Genetic disorders giving PHPT (not covered elsewhere)
- 6.3. Bone and mineral disorders
 - 6.3.1. Hypercalcemia other than PHPT
 - 6.3.2. Hypocalcemia other than HypoPT
 - 6.3.3. Hypophosphatemia
 - 6.3.4. Hypophosphatasia
 - 6.3.5. Hypomagnesemia
 - 6.3.6. Rickets and osteomalacia (including genetic disorders)
 - 6.3.7. Osteitis deformans (Paget's disease of bone)
- 6.4. Osteoporosis
 - 6.4.1. Postmenopausal osteoporosis
 - 6.4.2. Osteoporosis in men
 - 6.4.3. Osteoporosis secondary to endocrine disorders or medical treatment
 - 6.4.4. Osteoporosis secondary to organ transplantation

6.5. Vitamin D

- 6.5.1. Deficiency/insufficiency
- 6.5.2. Classical effects of vitamin D

6.6. Biochemical analyses in bone metabolism

- 6.6.1. Biochemical markers of bone turnover
- 6.6.2. Measurements of vitamin D metabolism

6.7. Measurement of bone mass and fracture assessment

- 6.7.1. DXA
- 6.7.2. Tools for fracture risk assessment

7. Pituitary

7.1. Hyperfunction of pituitary gland

- 7.1.1. Hyperprolactinemia
- 7.1.2. Acromegaly and gigantism
- 7.1.3. Cushing's disease
 - 7.1.3.1. *Nelson's syndrome*
- 7.1.4. TSH-oma
- 7.1.5. Syndrome of inappropriate secretion of antidiuretic hormone (SIADH)
 - 7.1.5.1. *Hyponatremia*

7.2. Hypofunction and other disorders of pituitary gland

- 7.2.1. Anterior pituitary deficiency
 - 7.2.1.1. *GH-deficiency in adulthood*
 - 7.2.1.2. *Kallman syndrome*
- 7.2.2. Diabetes insipidus
- 7.2.3. Hypothalamic dysfunction

7.3. Pituitary tumours

- 7.3.1. Pituitary adenomas – hormone secreting – non-hormonal aspects
- 7.3.2. Non-functioning pituitary tumours
- 7.3.3. Pituitary incidentalomas
- 7.3.4. Familial inherited pituitary adenomas
- 7.3.5. Craniopharyngioma
- 7.3.6. Rathke's cleft cysts

7.4. Cushing's syndrome (other than Cushing's disease)

- 7.4.1. ACTH-independent CS (see 2.2.2)
- 7.4.2. Ectopic ACTH syndrome

7.4.3. Drug-induced Cushing's syndrome

7.4.4. Pseudo-Cushing's syndrome including alcohol induced

8. Reproductive endocrinology and sexual function

8.1. Basic evaluation for infertility

8.2. Clinical evaluation of the menstrual cycle

8.3. Disorders of sexual development or intersex disorders

8.4. Sex hormone disorders

8.4.1. Hypogonadotropic hypogonadism (gonadotropin deficiency)

8.4.1.1. *Inherited (genetic and chromosomal) disorders*

8.4.1.2. *Acquired disorders*

8.4.2. Disorders of puberty

8.4.2.1. *Delayed puberty*

8.4.2.2. *Precocious puberty*

8.4.3. Menstrual dysfunction and fertility disorders

8.4.3.1. *Amenorrhea, oligomenorrhea, anovulation*

8.4.3.2. *Hyperandrogenism, hirsutism, polycystic ovary syndrome*

8.4.3.3. *Ovulation induction and assisted reproduction*

8.4.4. Contraception

8.4.5. Menopause

8.4.5.1. *Premature ovarian failure*

8.5. Ovarian dysfunction

8.5.1. Oestrogen excess

8.5.2. Primary ovarian failure

8.5.3. Ovarian tumours

8.6. Testicular dysfunction

8.6.1. Anorchia

8.6.2. Leydig cell hypoplasia

8.6.3. Germ cell aplasia (Sertoli cell only syndrome)

8.6.4. Spermatogenic arrest

8.6.5. Hypospermatogenesis

8.6.6. Numerical chromosome aberrations

8.6.6.1. *Klinefelter syndrome*

8.6.6.2. *XX-male syndrome*

8.6.6.3. *XYY-syndrome*

8.6.7. Structural chromosome aberrations

8.6.7.1. *Structural aberrations of the autosomes*

8.6.7.2. *Structural aberrations of sex chromosomes*

8.6.7.3. *Y chromosome microdeletions*

8.6.8. Testicular tumours

8.7. Management of male infertility

8.8. Enhancement of physical performance with abuse of hormones

8.8.1. Physiological effects of hormones during exercise

8.8.2. Testosterone and anabolic steroids abuse and modification of side effects

8.8.3. Abuse of peptide hormones

8.9. Gender dysphoria

8.9.1. Male-to-Female

8.9.2. Female-To-Male

9. Thyroid

9.1. Nontoxic goitre

9.1.1. Nontoxic diffuse goitre

9.1.2. Nontoxic single thyroid nodule

9.1.3. Nontoxic multinodular goitre

9.2. Hyperthyroidism

9.2.1. Hyperthyroidism with diffuse goitre (Graves-Basedow)

9.2.1.1. *Endocrine ophthalmopathy*

9.2.2. Hyperthyroidism with toxic single thyroid nodule

9.2.3. Hyperthyroidism with toxic multinodular goitre

9.2.4. Hyperthyroidism from ectopic thyroid tissue

9.2.5. Hyperthyroidism factitia

9.2.6. Thyroid crisis or storm

9.2.7. Amiodarone induced hyperthyroidism

9.2.8. Hyperthyroidism in pregnancy

9.3. Hypothyroidism

9.3.1. Autoimmune hypothyroidism

9.3.2. Congenital hypothyroidism in adulthood

9.3.3. Hypothyroidism due to medicaments and other exogenous substances

9.3.4. Post-infectious hypothyroidism

9.3.5. Atrophy of thyroid

- 9.3.6. Myxoedema coma
- 9.3.7. Hypothyroidism and thyroid hormone replacement therapy
- 9.4. Thyroiditis
 - 9.4.1. Acute thyroiditis
 - 9.4.2. Subacute thyroiditis
 - 9.4.3. Chronic thyroiditis with transient hyperthyroidism
 - 9.4.4. Autoimmune thyroiditis
 - 9.4.5. Drug-induced thyroiditis
 - 9.4.6. Riedel's thyroiditis
 - 9.4.7. Radiation-induced thyroiditis
 - 9.4.8. Postpartum thyroiditis
- 9.5. Thyroid cancer
- 9.6. Reduced sensitivity to thyroid hormone and thyroid hormone resistance
- 9.7. Drug-thyroid interactions
- 9.8. Evaluation and treatment of thyrotoxicosis
- 9.9. Non-thyroidal illness syndrome
- 9.10. Thyroid disease in pregnancy
- 9.11. Thyroid nodules
- 9.12. Disorders of iodine-deficiency
 - 9.12.1. Congenital iodine-deficiency syndrome
 - 9.12.2. Iodine-deficiency-related thyroid disorders and allied conditions
 - 9.12.3. Subclinical iodine-deficiency hypothyroidism

10. Neuroendocrine tumours

- 10.1. Medullary thyroid carcinoma
- 10.2. Thymus and mediastinal carcinoid tumours
- 10.3. Pulmonary neuroendocrine tumours
 - 10.3.1. Bronchus
 - 10.3.2. Pulmonary carcinoid tumours
 - 10.3.3. Small-cell lung cancer (SCLC)
 - 10.3.4. Large cell neuroendocrine carcinoma of the lung (LCNEC)
- 10.4. Extra-pulmonary small cell carcinomas (ESCC or EPSCC)
- 10.5. Gastroenteropancreatic neuroendocrine tumours (GEP-NET)
 - 10.5.1. Foregut GEP-NET
 - 10.5.2. Pancreatic endocrine tumours

- 10.5.3. Midgut GEP-NET
- 10.5.4. NET in the Appendix
- 10.5.5. Hindgut GEP-NET
- 10.6. Liver and gallbladder
- 10.7. Breast
- 10.8. Genitourinary tract
 - 10.8.1. Urinary tract carcinoid tumour and neuroendocrine carcinoma
 - 10.8.2. Ovary
 - 10.8.3. Neuroendocrine tumour of the cervix
 - 10.8.4. Testes
- 10.9. Merkel cell carcinoma of skin (trabecular cancer)

11. Genetics of Inherited and related disorders

- 11.1.1. Multiple endocrine neoplasia type 1 (MEN1)
- 11.1.2. Multiple endocrine neoplasia type 2 (MEN2)
 - 11.1.2.1. *MEN type 2a*
 - 11.1.2.2. *MEN type 2b*
- 11.1.3. Multiple endocrine neoplasia type 4 (MEN4)
- 11.1.4. von Hippel-Lindau (VHL) disease
- 11.1.5. Familial paraganglioma
- 11.1.6. Neurofibromatosis type 1
- 11.1.7. Tuberous sclerosis
- 11.1.8. Carney complex

12. Autoimmune and other polyendocrine syndromes

- 12.1. APS 1
- 12.2. APS 2/3
- 12.3. Endocrine and metabolic disorders in long term survivors after neoplastic diseases

13. Diagnostic techniques in endocrinology

- 13.1. Ultrasound
 - 13.1.1. Thyroid including fine needle aspiration
 - 13.1.2. Pancreatic endoscopic ultrasound
 - 13.1.3. Ovarian
- 13.2. CT

13.3. MRI

13.4. Endocrine scintigraphy with SPECT/CT

13.4.1. Technetium, Iodine scintigraphy

13.4.2. Sestamibi scintigraphy

13.4.3. NP-59 iodocholesterol scanning

13.4.4. MIBG scintigraphy

13.4.5. Somatostatin receptor imaging

13.4.6. PET and PET/CT

13.5. Angiographic techniques and localisation with venous sampling

13.5.1. Inferior petrosal sinus sampling for ACTH

13.5.2. Bilateral adrenal venous sampling for aldosterone and cortisol

13.5.3. Intra-arterial calcium stimulation with hepatic venous sampling for functioning insulinomas and gastrinomas

13.5.4. Parathyroid venous sampling for recurrent primary hyperparathyroidism

13.6. Assessment of hormones: RIA, ELISA, LC-MS/MS, POCT

