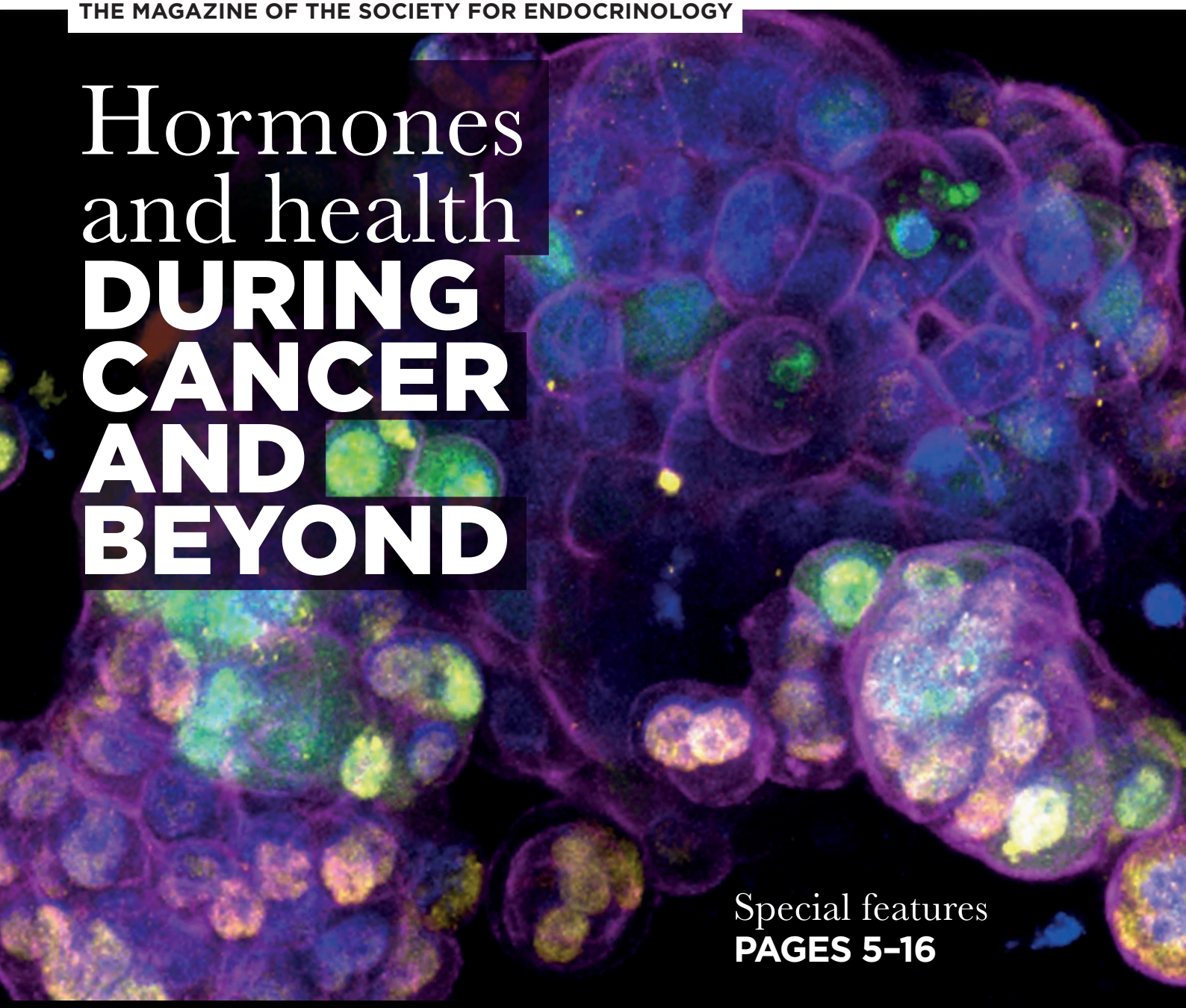
Society for Endocrinology

ISSUE 156 SUMMER 2025
ISSN 2045-6808

THE ENDOCRINOLOGIST

THE MAGAZINE OF THE SOCIETY FOR ENDOCRINOLOGY



Hormones and health DURING CANCER AND BEYOND

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A word from THE EDITOR...

As I'm new to the role of Editor, I want to introduce myself. I'm Kate, I've worked as a scientist in the field of endocrinology for over 10 years now, and for most of that time I have been involved with the Society for Endocrinology.

I started off on the Early Career Steering Group when I was fresh from my PhD. I'm now running my own research team at Oxford Brookes University and am a member of the Science Committee. I therefore hope I have the credentials to do this role justice, and I'm excited to be working with the editorial team to share some great themes and articles with you over the next few years.

As my research career has been dedicated to cancer, it seems fitting that I have joined for this issue. Cancer is still one of the leading causes of death worldwide and, as survival continues to improve (yay!), further post-cancer challenges are arising. This issue shows how we, as a community, are addressing this.

For example, **Stephanie Agbana, Michael O'Reilly and Marie McIlroy** (who provide our striking cover image) describe the development of a model system that accurately recreates the growth patterns of breast cancer using 3D bioprinting. This theme continues in our interview with **Rachael Guenter**, who explains how the NET Models consortium is trying to improve preclinical models for studying neuroendocrine cancer.

With yin comes yang, and that's the case with improving survival, particularly of childhood cancers. In this issue, **Claire Higham and Victoria Chatzimavridou Grigoriadou** describe how cancer survivors have a heightened risk of fractures due to many different factors, including cancer therapies. Fertility of cancer survivors is also an increasing concern, as discussed by **Cecilia Follin**, as she highlights the role that nurses can play.

Finally, the Society is hosting its first **Hormone Dependent Cancer meeting** this year, so check it out if you are interested in endocrine-related cancers.

I hope you sit back and enjoy reading this issue of *The Endocrinologist* as much as I did.

With best wishes

KATE LINES

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Become a contributor... Contact the Editorial office at endocrinologist@endocrinology.org

The Society welcomes news items, contributions, article suggestions and letters to the Editor. We would also like to hear your feedback on this issue of the magazine.

Deadline for news items for the AUTUMN 2025 issue: **10 July 2025**.

Front cover image: 3D bioprinted breast cancer cell line model, JIMT-1 brains (GFP tagged, Her2+, brain metastatic), stained with estrogen receptor (yellow), F-actin (magenta), and DAPI (blue). Marie McIlroy, Stephanie Agbana and Michael O'Reilly. See article on **page 13**.

HOT TOPICS

Hot Topics is written by Victoria Chatzimavridou Grigoriadou, John Hough, Zin Htut, Edouard Mills, Gareth Nye, Bhavna Sharma, Vincent Simpson and Angela Taylor

SOCIETY FOR ENDOCRINOLOGY OFFICIAL JOURNALS

Society members have free access to the current content of *Journal of Endocrinology*, *Journal of Molecular Endocrinology*, *Endocrine-Related Cancer* and *Clinical Endocrinology* via the **Members' Area of the Society website**. *Endocrine Connections*, *Endocrinology, Diabetes & Metabolism Case Reports* and *Endocrine Oncology* are open access and free to all. Publishing in *Endocrine Oncology* is currently free.

JOURNAL OF ENDOCRINOLOGY

Vimentin-expressing α-cell phenotype in cystic fibrosis

Cystic fibrosis (CF) affects multiple organs, including the pancreas, which sometimes leads to CF-related diabetes. Metabolic studies have demonstrated impaired β- and α-cell function in people with CF and pancreatic exocrine insufficiency, but the specific cellular changes remain unclear. Epithelial-to-mesenchymal transition (EMT) is a process commonly linked to pro-fibrotic pathologies. This study aimed to investigate EMT in pancreatic endocrine cells from subjects with and without CF.

From analysis of post-mortem pancreas tissue, Kattner *et al.* identified a previously unrecognised sub-population of α-cells which co-expressed vimentin (a classic mesenchymal marker) and glucagon, in subjects with and without CF. In donors without CF under 31 years, the frequency of these cells correlated positively with intra-islet collagen deposition, suggesting a possible role in tissue stress response or ageing. Single-cell RNA sequence analysis confirmed the presence of vimentin-positive (and -negative) α-cells in non-diabetic donors and in donors with chronic pancreatitis. Co-expressing cells retained normal α-cell gene signatures, but showed upregulation of genes related to extracellular matrix organisation, cell adhesion, migration, proliferation and stress resilience.

This suggests that vimentin-expressing α-cells represent an intermediate epithelial/mesenchymal state, potentially facilitating α-cell adaptation to fibrosis, stress and ageing.

Read the full article in *Journal of Endocrinology* **264** e240190
https://doi.org/10.1530/JOE-24-0190.

JOURNAL OF MOLECULAR ENDOCRINOLOGY

Metformin and the endometrial proteome mice with diet-induced obesity

It is well known that obesity affects fertility by altering ovarian and endometrial function and lowering the chance of successful pregnancy. For those with reproductive disorders such as polycystic ovary syndrome, metformin has proved to be a successful treatment option to increase the likelihood of pregnancy. The theory is that metformin somehow alters the changes in endometrial proteins induced by obesity.

This new study by Malliou-Becher *et al.* aimed to uncover metformin's role in the endometrial layer through use of a C57 mouse model line, fed either a standard diet or a high-fat variation. Following this diet, the mice were either given metformin or remained without additional treatment.

Proteomic analysis uncovered significant alterations in the protein expression of the endometrial tissue specifically, due to the high-fat diet. This was restored to normal levels via metformin in most cases. Some of the specific proteins associated with reproductive health include calcium-independent phospholipase A2γ and apolipoprotein C-III.

These findings suggest there may be a potential therapeutic role for metformin in correcting altered endometrial protein expression, which could improve fertility outcomes in women with obesity or insulin resistance.

Read the full article in *Journal of Molecular Endocrinology* **74** e240098
https://doi.org/10.1530/JME-24-0098

ENDOCRINE-RELATED CANCER

CCL2 in pituitary tumours: beyond chemotaxis to predict clinical outcomes

Pituitary neuroendocrine tumours (PitNETs) display significant clinical variability, making the identification of predictive biomarkers a priority. In this context, Silva *et al.* investigated the role of the chemokine CCL2. It has previously been recognised primarily for mediating immune cell recruitment, but its function in PitNET biology has remained unclear.

Using a well-characterised cohort of 86 patients with PitNETs, associations were reported between elevated CCL2 expression – quantified by mRNA and immunoreactivity – and clinically relevant parameters; these included larger tumour size, hypopituitarism at diagnosis, persistent disease and treatment complexity. Interestingly, elevated CCL2 mRNA was negatively correlated with CDH1 (E-cadherin) expression, suggesting a potential role in promoting epithelial-to-mesenchymal transition. Additionally, higher CCL2 protein expression correlated positively with increased markers of angiogenesis, such as vessel density and area, independent of macrophage infiltration.

While these findings provide novel biological insights into PitNET aggressiveness, the authors acknowledge that their results represent associative evidence rather than direct causation. They emphasise the need for validation through larger, prospective studies before CCL2 can be confidently used as a predictive biomarker or therapeutic target. Nevertheless, the study is a comprehensive, novel exploration of the potential biological roles of CCL2 beyond immune chemotaxis, providing important new insights into PitNET biology.

Read the full article in *Endocrine-Related Cancer* **32** e240293
https://doi.org/10.1530/ERC-24-0293

ENDOCRINE CONNECTIONS

Can PCOS be treated with GLP-1 receptor analogues?

Glucagon-like peptide-1 (GLP-1) analogues are generating interest in the up-to-date management of diabetes. Some research focuses on their potential endocrine impact.

This review by Monney *et al.* thoroughly evaluates the role of GLP-1 in polycystic ovary syndrome (PCOS), as PCOS is associated with various hormonal, reproductive and metabolic alterations, including androgen excess, ovulatory disorders and a hyperinsulinaemic state. GLP-1 receptor analogues are discussed in the context of a variety of features of PCOS and their overall impact in each, including weight gain, various scorings and insulin resistance. This opens up an attractive option for this often under-treated disease.

Read the full article in *Endocrine Connections* **14** e240529
https://doi.org/10.1530/EC-24-0529

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CLINICAL ENDOCRINOLOGY

Heightened prevalence of MASLD in primary aldosteronism

Primary aldosteronism is the most common secondary cause of hypertension, affecting 10% of hypertensive patients. Aldosterone excess in the setting of primary aldosteronism increases the risk of cardiovascular complications and glucometabolic dysregulation, including metabolic dysfunction-associated steatotic liver disease (MASLD).

Tizianel *et al.* assessed the prevalence of MASLD in patients with primary aldosteronism (without cortisol co-secretion) compared with hypertensive patients with non-functioning adrenal incidentalomas (NF-AI), mild autonomous cortisol secretion (MACS) and adrenal Cushing's syndrome. Hepatic steatosis was assessed by liver/spleen ratio from unenhanced baseline abdominal computed tomography imaging.

In their series, hepatic steatosis was significantly higher in patients with primary aldosteronism at diagnosis, compared with those with NF-AI or MACS, but similar to those with adrenal Cushing's syndrome. Medical and surgical treatment in patients with primary aldosteronism significantly reduced hepatic steatosis. The prevalence of MASLD was significantly higher in primary aldosteronism (49%), compared with MACS (25%) or NF-AI (14%), but comparable with adrenal Cushing's syndrome (45%).

Based on their findings, the authors reveal a heightened prevalence of MASLD in patients with primary aldosteronism compared with NF-AI and MACS, as well as improvements in hepatic steatosis after treatment.

Read the full article in *Clinical Endocrinology*
https://doi.org/10.1111/cen.15231

ENDOCRINOLOGY, DIABETES & METABOLISM CASE REPORTS

Immune checkpoint inhibitors triggering sarcoid-like hypercalcaemia

Severe hypercalcaemia is a rare complication of immunotherapy and is associated with immune checkpoint inhibitor treatment. It is termed a drug-induced sarcoid-like reaction (DISR), because it mimics sarcoidosis. DISR commonly presents with bilateral hilar lymphadenopathy, cutaneous lesions, uveitis and hypercalcaemia. There are currently no specific guidelines for treating calcitriol-mediated hypercalcaemia, a form of DISR.

Dharmaputra reports the case of a 72-year-old male with metastatic melanoma, previously treated with dabrafenib–trametinib combination therapy, who presented with polydipsia, polyuria and fatigue. Biochemistry confirmed hypercalcaemia (corrected calcium 3.84mmol/l) with suppressed parathyroid hormone (1.1pmol/l) and raised phosphate (1.62mmol/l) following the second cycle of ipilimumab–nivolumab combination therapy. He had skeletal and pulmonary metastases on a background of prostate cancer and chronic kidney disease (estimated glomerular filtration rate 30ml/min/1.73m^2).

Investigations revealed a significantly elevated serum calcitriol level (429pmol/l), and chest imaging demonstrated hilar lymphadenopathy consistent with DISR. He was successfully treated with intravenous fluids, subcutaneous calcitonin, denosumab and oral prednisolone.

DISR is a rare cause of hypercalcaemia following immunotherapy, which may not be associated with the same increased mortality risk. Prompt recognition and treatment with glucocorticoids and anti-resorptive agents, such as denosumab, can effectively manage calcitriol-mediated hypercalcaemia.

Read the full article in *Endocrinology, Diabetes & Metabolism Case Reports*
EDM-24-0116 https://doi.org/10.1530/EDM-24-0116

ENDOCRINE ONCOLOGY

HHLA2 as a biomarker and treatment target in endocrine cancer

Human endogenous retrovirus-H long terminal repeat-associating 2 (HHLA2), a member of the B7 family, is an emerging immune checkpoint molecule. It has potential therapeutic relevance, particularly in cancers resistant to PD-1 (programmed cell death-1) and CTLA-4 (cytotoxic T-lymphocyte antigen-4) inhibitors. This review by Gruetzmacher *et al.* consolidates data on HHLA2 expression in endocrine-related cancers and its prognostic implications.

Of 117 studies that were screened, 12 met the inclusion criteria. Findings reveal variable HHLA2 expression across endocrine tumours, influenced by both cancer type and tumour microenvironment. In pancreatic and some ovarian cancers, high HHLA2 expression correlated with better survival outcomes. However, in thyroid and neuroendocrine tumours, elevated levels were linked to aggressive features, metastasis and poor prognosis. The dual role of HHLA2 – acting either as an immune activator or suppressor – appears to be context-dependent, shaped by interactions with receptors TMIGD2 and KIR3DL3 on T and NK cells.

Given its limited expression in healthy tissues and its prevalence in PD-L1 (programmed cell death ligand-1)-negative tumours, HHLA2 presents a promising immunotherapy target. The review also highlights the need for further studies, particularly using humanised models and chimeric antigen receptor-modified T (CAR-T) strategies, to fully understand HHLA2's functions and therapeutic potential. Overall, this research lays the groundwork for HHLA2's integration into future cancer immunotherapy approaches.

Read the full article in *Endocrine Oncology* **5** e240034
https://doi.org/10.1530/EO-24-0034

ENDOCRINE HIGHLIGHTS

A summary of papers from around the endocrine community that have got you talking.

Gut microbiota and colonic enteroendocrine cell interaction regulates host metabolism

Body weight regulation relies on complex neurohormonal mechanisms, with the enteroendocrine cells (EECs) of the intestine playing a central role. These specialised cells produce over 20 peptide hormones and can sense small molecules in the gut lumen as well as circulating metabolites and hormones. Research has largely focused on EECs in the proximal gastrointestinal tract, while the role of colonic EECs has been overlooked, partly due to the belief that the colon has a limited metabolic role, while mainly supporting water and electrolyte absorption. However, new insights into the colon's microbiota reveal its important role in fermenting non-digestible nutrients.

Tan and colleagues explored the role of colonic EECs in metabolism by developing a mouse model with EEC deficiency. They observed that the EEC-deficient mice developed hyperphagia and obesity. Experiments indicated that EEC deficiency alters microbiota composition and metabolism. Stool and blood metabolome analyses revealed that differential glutamate production by gut microbiota was linked to increased appetite. Additionally, direct colonic administration of glutamate was shown to boost food intake.

These findings reveal a previously unknown connection between the host and microbiota in the colon, which plays a role in the gut–brain axis that controls metabolism and body weight.

Read the full article in *Nature Metabolism* **6** 1076–1091
https://doi.org/10.1038/s42255-024-01044-5

WHEN BLOOD COUNTS: WHAT INFLAMMATION SCORES REVEAL ABOUT ADRENAL TUMOURS

WRITTEN BY CRISTINA L RONCHI AND ALESSANDRO PRETE

Adrenal tumours have become a familiar finding in endocrine practice, affecting 1–7% of adults. Some of these tumours can be cancerous, and up to half can produce hormones in excess, which is associated with increased cardiovascular, metabolic and mortality risks. Understanding how these risks arise, and how to stratify patients more precisely, remains a key challenge.

A new line of research is now exploring a surprising source of insight: the humble full blood count. By repurposing routine haematology results into inflammation-based scores, our group has uncovered systemic immune signatures linked to malignancy and hormonal activity in adrenal tumours.

HOW EXCESS ADRENAL HORMONES AFFECT THE IMMUNE SYSTEM

Cortisol, a key immunomodulator, exerts complex effects on immune function. Chronic excess, as seen in Cushing's syndrome, is associated with characteristic haematological changes – neutrophilia, lymphopaenia and eosinopaenia – that increase susceptibility to infections. Even mild autonomous cortisol secretion (MACS), which lacks the classic signs of Cushing's syndrome, appears to influence immune function in subtler, but still clinically relevant, ways. Similarly, catecholamines released by phaeochromocytomas can alter immune activation and inflammation, although the mechanisms remain less well defined.

INFLAMMATION-BASED SCORES: A WINDOW INTO SYSTEMIC INFLAMMATION

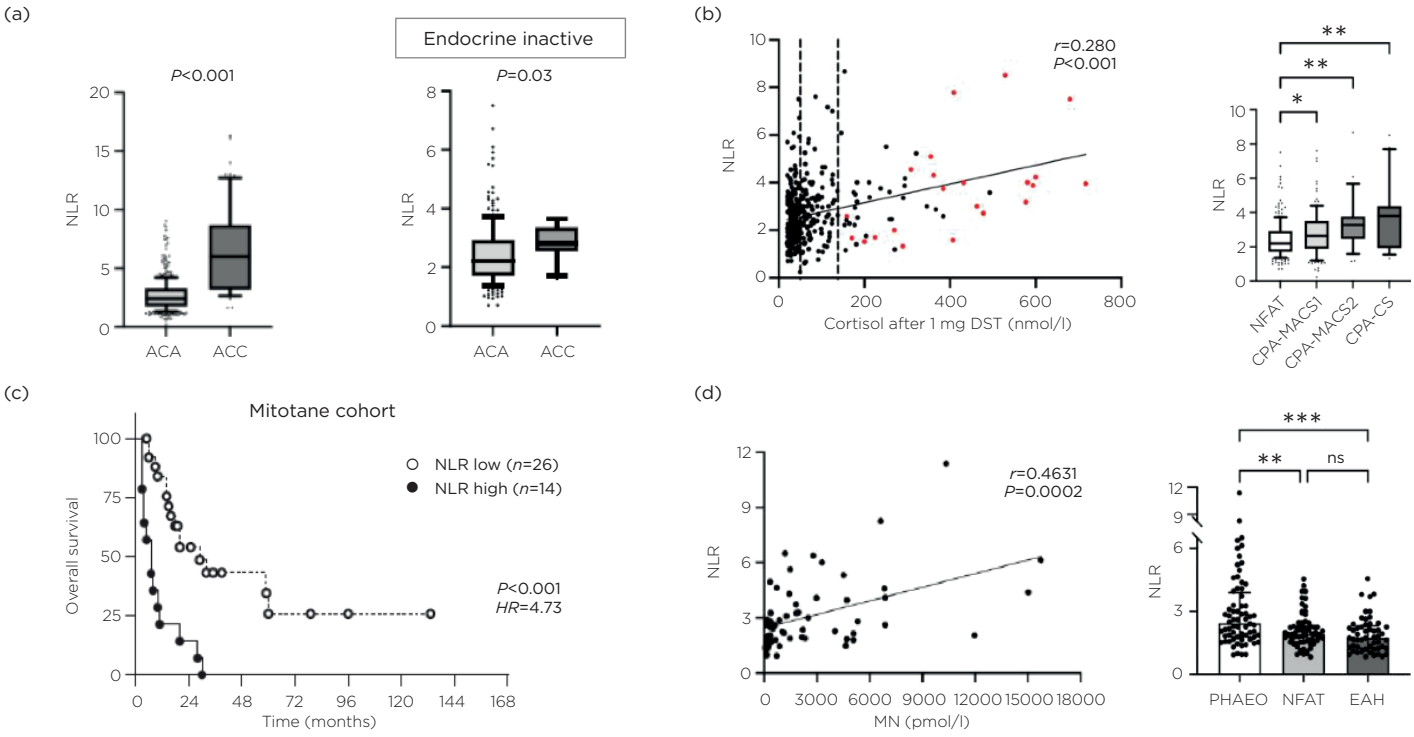
Inflammation-based scores have been proposed as surrogate markers of systemic inflammation in a range of conditions, such as ischaemic heart disease, stroke and cancer. These scores can easily be calculated using standard laboratory tests, such as full blood count and albumin, and correlate with acute and chronic inflammation.

The increasing interest in these markers is due to their recognised prognostic value as well as their cost-effectiveness, wide availability and practicality. For instance, an increase in the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio and systemic immuno-inflammation index (SII, the product of platelet count and NLR), and a decrease in the lymphocyte-to-monocyte ratio (LMR) and prognostic nutrition index (that considers serum albumin and the absolute lymphocyte count), reflect ineffective immune surveillance or an increased inflammatory state.

CORTISOL EXCESS AND INFLAMMATION-BASED SCORES

We recently explored how inflammation-based scores behave in benign adrenal tumours associated with cortisol excess. In a retrospective study of 375 patients, we found that NLR and SII rose in line with cortisol levels – across non-functioning adrenal tumours, MACS, and adrenal Cushing's syndrome – while LMR decreased.[1]

(a) NLR in patients with adrenocortical adenomas (ACAs) versus ACCs. (b) NLR and cortisol secretion in patients with ACAs (NFAT, non-functioning adrenal tumour; CPA, cortisol-producing adenoma; CS, Cushing's syndrome; DST, dexamethasone-suppression test; MACS-1, possible MACS; MACS-2, definitive MACS; *P<0.05, **P<0.01 and ***P<0.001). (c) NLR and overall survival in patients with ACCs. (d) NLR and plasma metanephrines (MN) in patients with phaeochromocytomas (PHAEO) (EAH, essential hypertension; **P<0.002, ***P<0.0001).



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These findings suggest that even modest cortisol excess disrupts immune cell populations and contributes to systemic inflammation. Importantly, this immune signature was detectable even in patients without overt features of Cushing's syndrome. These changes may help explain the increased cardiometabolic burden associated with MACS and point toward potential uses for inflammation-based scores in risk stratification and monitoring.

INSIGHTS FROM ADRENAL CANCER

Inflammation-based scores may also help distinguish benign from malignant adrenal tumours. In a cohort of patients with adrenocortical carcinomas (ACCs) or adrenocortical adenomas, we found significantly different inflammation-based scores in ACCs, even in those without hormone excess. This suggests that the malignancy itself contributes to systemic inflammation.[2] In a separate study of 90 patients with advanced ACCs, an elevated NLR predicted earlier progression during mitotane treatment or systemic chemotherapy.[3] These findings are now being validated in a multicentre study led by the European Network for the Study of Adrenal Tumors (**www.ensat.org**).

PHAEOCHROMOCYTOMAS: INFLAMMATION AND CATECHOLAMINES

Phaeochromocytomas also show evidence of systemic inflammation. We showed that affected patients had elevated preoperative NLR and SII values that correlated with plasma metanephrine levels.[4] These scores improved after adrenalectomy or α-blockade, indicating a dynamic relationship between catecholamine excess and systemic inflammation.[4]

This is clinically important, as inflammation may contribute to the cardiovascular and metabolic complications observed in these patients. By better understanding this link, we may be able to optimise treatment strategies, not just for tumour control, but also to improve long-term outcomes.

CLINICAL TAKEAWAYS AND FUTURE DIRECTIONS

Inflammation-based scores offer a new, promising tool for assessing adrenal tumours. Their simplicity, broad availability and cost-effectiveness make them appealing for integration into routine care, especially when biochemical or imaging findings are inconclusive. They may help stratify cardiometabolic risk in MACS, monitor the systemic effects of adrenal hormone excess, understand how malignancy and hormone excess affect immune function, and predict prognosis in ACC.

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BONE HEALTH IN ONCOLOGY PATIENTS: RECOGNISING AND MANAGING FRACTURE RISK

WRITTEN BY CLAIRE E HIGHAM AND VICTORIA CHATZIMAVRIDOU GRIGORIADOU

With advancements in early cancer detection and improved therapies, the population of cancer survivors continues to grow significantly. In the UK alone, approximately 385,000 new cancer cases are diagnosed annually. Remarkably, about half of individuals diagnosed with cancer now survive for at least 10 years post-diagnosis.[1] However, extended survival brings a series of chronic health issues, prominently including an increased risk of bone fractures.

Fractures in cancer survivors significantly impact quality of life and healthcare resources and are associated with increased mortality.[2] Bone health in oncology patients is therefore one of the three research sub-themes within the **Manchester Biomedical Research Centre (BRC) Living With and Beyond Cancer Theme,** led by Claire Higham.

A recent UK population-based study using the Clinical Practice Research Datalink (CPRD) found that survivors of 15 out of the 20 most common adult cancers had a heightened risk of fractures. This risk persisted up to five years after diagnosis, with major osteoporotic fractures – of the hip, spine, pelvis, wrist and proximal humerus – being especially common across multiple tumour types.[2]

The underlying causes of cancer-related bone fragility are multifactorial. Cancer therapies, particularly hormone treatments and glucocorticoids, play a central role. In breast and prostate cancer, aromatase inhibitors, ovarian suppression and androgen deprivation therapy (ADT) significantly reduce sex steroid levels, leading to increased bone turnover and loss of bone mineral density (BMD).[3] High-dose glucocorticoids, used in various oncological settings, further exacerbate bone resorption.[4] These changes, compounded by age-related frailty and sarcopenia, increase the risk of falls and fractures.[5]

DETERMINING THE RISKS

Despite recognition of these risks, fracture prediction tools like FRAX® may not be as accurate in oncology populations. Recent data from trials

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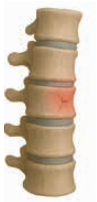
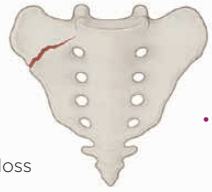


INCREASED RATES OF FRACTURE
Observed across numerous different malignancies

MULTIFACTORIAL CAUSES
- Hormone therapies
- Glucocorticoids
- Radiation
- Increased bone turnover and BMD loss
- Frailty/falls



RESEARCH DIRECTIONS
- Validity of fracture risk algorithms
- Radiotherapy-related insufficiency fractures:
  - RadBone feasibility study
  - Small animal model of pelvic irradiation
  - Radiomics
- BoND study
  Prospective, with clinical and omics data

Bone health in oncology patients.

such as STAMPEDE suggest the need for broader bone protective strategies beyond those guided by current risk thresholds.[6] Building on this, our team at the University of Manchester is working to address key gaps in risk stratification and management.

As part of this work, we are exploring the validity of fracture risk algorithms in 500,000 adult oncology patients using UK CPRD data, in collaboration with Darren Ashcroft at the NIHR Patient Safety Research Centre. Our goal is to assess how well current tools identify patients who go on to fracture, particularly those with limited life expectancy or who are undergoing specific cancer therapies.

A second focus is radiotherapy-related insufficiency fractures (RRIFs), which represent a distinct clinical entity most commonly seen after pelvic radiotherapy. These fractures often occur even in patients with normal BMD, indicating that traditional tools may miss this risk. While best practice guidance from the Pelvic Radiation Disease Association (PRDA) exists, it is not evidence-based.[7] To address this, we recently completed a feasibility study (RadBone), funded by an MRC Clinical Academic Research Partnership (Claire Higham), at the Christie Hospital in Manchester.[8] This study piloted a musculoskeletal health programme combining physical assessment, exercise prehabilitation, fracture risk evaluation and targeted bone treatments. Results are expected in 2025 and will inform a future UK multicentre clinical intervention trial.

UNDERLYING MECHANISMS

Complementing this clinical research, we are also investigating the mechanistic underpinnings of RRIFs through a University of Manchester MRC-funded PhD project (Victoria Chatzimavridou Grigoriadou). In collaboration with the University of Sheffield SkeletAL Lab, we are developing a small animal model of pelvic radiotherapy to explore bone structure, turnover and microdamage using imaging and histomorphometry techniques. This work aims to characterise how radiation affects different bone compartments, including trabecular and cortical regions. Novel radiomic techniques and detailed analysis of RRIFs are also being taken forward as a BRC-funded physics-based PhD at the University of Manchester (Artemis Bouzaki).

In parallel, we have launched the Bone Health in Oncology Dataset (BOnD) within our BRC theme. This prospective study is recruiting patients attending the Metabolic Bone Clinic at the Christie, incorporating clinical assessments, polygenic risk scores and omics profiling to better understand fracture susceptibility in cancer patients.

FRACTURES AND CANCER OUTCOMES

Fractures have a measurable and concerning impact on cancer outcomes. For example, men with prostate cancer receiving ADT who sustained skeletal fractures had reduced median survival compared with those without fractures.[9] Similarly, fractures in patients with breast cancer have been linked to increased hospitalisation and mortality.[10] A large Canadian registry study also reported that fracture-related mortality remains elevated for years after the initial event.[11] These data underline the importance of targeted prevention strategies.

The rising number of cancer survivors calls for a shift in how we view long-term care. While preventing recurrence remains a priority, we must also recognise and address survivorship-related complications, such as fragility fractures. Current guidelines offer practical approaches to fracture risk management, including BMD measurement, FRAX® assessment, and pharmacological treatments, such as bisphosphonates or denosumab. However, they may fall short in complex or rapidly evolving oncology settings.

There is growing momentum behind research into bespoke risk-prediction models and interventions that reflect cancer type, treatment history, co-morbidities and broader factors, such as frailty. Integrating fracture risk assessments into oncology care pathways, supported by clinician education and cross-disciplinary collaboration, will be vital.

Ultimately, supporting bone health in cancer survivors is not just about preventing fractures – it is about improving survival, maintaining independence and enhancing quality of life. With growing survivorship, recognising fractures as a preventable consequence of cancer and its treatment can help shape more holistic, proactive care.

CLAIRE E HIGHAM and VICTORIA CHATZIMAVRIDOU GRIGORIADOU
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The BOnD study and CPRD fracture risk work are being delivered through the NIHR Manchester BRC (NIHR203308). The views expressed are those of the author(s) and not necessarily those of the NIHR or the Department of Health and Social Care. This work is also supported by the MRC (grant number MR/T024887/1; fellowship number MR/Z504166/1).

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FERTILITY IN CHILDHOOD CANCER SURVIVORS
A NURSING PERSPECTIVE

WRITTEN BY CECILIA FOLLIN

Due to advances in cancer treatment, survival rates among childhood cancer patients have improved significantly. Survival now exceeds 80% in many childhood malignancies. However, survivors may face a range of reproductive complications, including abnormalities in pubertal development (e.g. precocious puberty, or delayed or absent puberty), menstrual dysfunction, endocrine disorders, infertility, and complications in pregnancy or labour. Fertility-related concerns are consistently ranked among the top five unmet needs during the survivorship period.[1]

INCREASED RESPONSIBILITY FOR NURSES

As the survival rates of childhood cancer continue to rise, the nursing responsibility to address issues relating to quality of life will be more important, including fertility preservation and reproductive health counselling. Nurses who care for survivors of childhood and adolescent cancer must understand and anticipate long-term, treatment-related complications, such as gonadal insufficiency and impaired fertility.

The risk of infertility is influenced by the location of the cancer, as well as the type, dose and combination of cytotoxic therapies used. Additionally, factors such as the patient's age at treatment, sex, and genetic predispositions further affect the risk of permanent infertility.

Survivors are particularly vulnerable to infertility following radiotherapy and treatment with alkylating agents, both of which have dose-dependent gonadotoxic effects. Specific treatment thresholds associated with a significantly elevated infertility risk include:

- testicular radiation >4Gy
- ovarian radiation >5Gy
- hypothalamic–pituitary radiation >22–30Gy (in females)
- total body irradiation (TBI).

These exposures are known to contribute to premature gonadal failure and permanent infertility.[2,3] Radiation impacting the ovaries and uterus has also been linked to pregnancy complications, such as spontaneous abortion, preterm labour, fetal malposition and low birth weight.[4]

MEETING THE NEED

Fertility concerns can greatly affect the long-term quality of life of survivors of childhood cancer. While international guidelines for fertility preservation exist, there remains a lack of specific recommendations on how to deliver fertility counselling to adolescents. For nurses (who often serve as consistent points of contact and sources of emotional support), this gap presents both a challenge and an opportunity.

Barriers to fertility discussions include the urgency of initiating cancer treatment, poor prognosis, and cultural or religious considerations. Nurses are in a key position to assess emotional readiness, provide developmentally appropriate education, and advocate for patient and family needs within the multidisciplinary team.

Nurses hold a pivotal position in the continuum of care for survivors of childhood cancer, particularly when it comes to addressing fertility-related concerns. As trusted healthcare providers who often build long-term relationships with patients and their families, nurses are uniquely equipped to provide education, support and advocacy throughout the cancer journey and beyond. Nurses play a vital role in delivering clear, age-appropriate information about the potential impact of cancer treatments on fertility. They help demystify complex medical information, ensuring that patients and caregivers understand the risks and available fertility preservation options.

THE IMPORTANCE OF TIMELY INFORMATION

By providing this education early in the treatment process, nurses empower families to make informed decisions in a timely manner. Early discussions about fertility are crucial, yet often overlooked due to the urgency of initiating treatment. Nurses are well-positioned to advocate for and initiate timely fertility counselling before treatment begins.

They also help co-ordinate long-term follow-up, reinforcing the importance of monitoring reproductive health as part of survivorship care. Nurses can advocate for policies and practices that promote equity in access to fertility clinics. This includes raising awareness within healthcare teams, and working for changes that integrate fertility care into routine follow-up after childhood cancer.

INFORMING BEST PRACTICE

As frontline providers, nurses have valuable insights into patient needs and care gaps. By engaging in or supporting research, nurses contribute to a growing body of evidence that can inform best practice in fertility preservation. Their involvement helps to shape guidelines, improve care delivery and, ultimately, enhance survivorship outcomes. Additionally, nurses can advocate for equitable access to fertility preservation services. This includes raising awareness among healthcare providers, promoting institutional changes, and working towards the integration of fertility care into standard survivorship protocols.

Nurses in oncology care also bring valuable insights into patient needs and systemic gaps. Their participation in clinical research contributes to a growing body of evidence that informs best practice and supports the development of standardised fertility care guidelines. Ultimately, such involvement enhances survivorship outcomes and supports high quality, patient-centred care.

Fertility-related psychological distress can be profound. Through compassionate nursing interventions, including emotional support, active listening and timely referral to mental health services, nurses can reduce anxiety and create an environment where patients and families feel more open to discussion. This foundation of trust and support is essential for delivering personalised survivorship care that addresses both physical and emotional well-being.

As the population of childhood cancer survivors grows, so too does the imperative for nurses to lead efforts in promoting long-term reproductive health and overall quality of life.

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IDENTIFYING PITUITARY ENDOCRINOPATHY AFTER CHECKPOINT INHIBITORS

WRITTEN BY DANIEL L MORGANSTEIN

Prior to 2010, hypophysitis was a rare condition, mostly seen in the post partum period or due to IgG4-related disease. However, that changed with the introduction of ipilimumab, a cytotoxic T-lymphocyte antigen-4 (CTLA-4) inhibitor, also termed an immune checkpoint inhibitor, which was among the first treatments to show survival benefit in melanoma.

SIDE EFFECTS OF CHECKPOINT INHIBITORS

It rapidly became apparent that ipilimumab also caused a presumed immune-mediated hypophysitis in up to 10% of patients. This frequently presents with headache, occasionally with pituitary enlargement significant enough to impact the optic chiasm, and with near-universal hypopituitarism.[1] It is now well recognised that pituitary dysfunction can also frequently occur without clear-cut evidence of pituitary inflammation.

Checkpoint inhibitors target naturally occurring immune checkpoints – co-regulators of T-cell function that drive self-reacting T-cells to anergy, for example CTLA-4 interacting with B7-1 expressed on antigen-presenting cells. Blocking this leads to enhanced T-cell activation against the cancer cells, but also to a risk of immune-related adverse events (Figure below).

Subsequent developments led to drugs blocking the interaction between programmed cell death-1 (PD-1) and programmed cell death ligand-1 (PD-L1) on more mature T-cells. This pathway is exploited by many cancer cells that over-express PD-L1 to avoid immune detection.[2] Drugs targeting this PD-1/PD-L1 interaction are now widely used in many different cancers, including in adjuvant treatment, or increasingly (as in breast cancer) in the neo-adjuvant setting prior to surgery.

PD-1/PD-L1 inhibitors show a different pattern of pituitary dysfunction, with actual hypophysitis being rare, but isolated adrenocorticotrophin (ACTH) deficiency developing in up to 2% of treated individuals.[3] However, as these drugs are now much more widely used than ipilimumab, this is translating to ever-increasing numbers of affected patients. Transient secondary hypothyroidism and hypogonadism are described less commonly than with ipilimumab, and may recover with both drugs. The striking finding, however, is that ACTH deficiency is permanent, leading to a life-long need for glucocorticoid replacement.[4]

RECOGNISING THEIR ENDOCRINE IMPACT

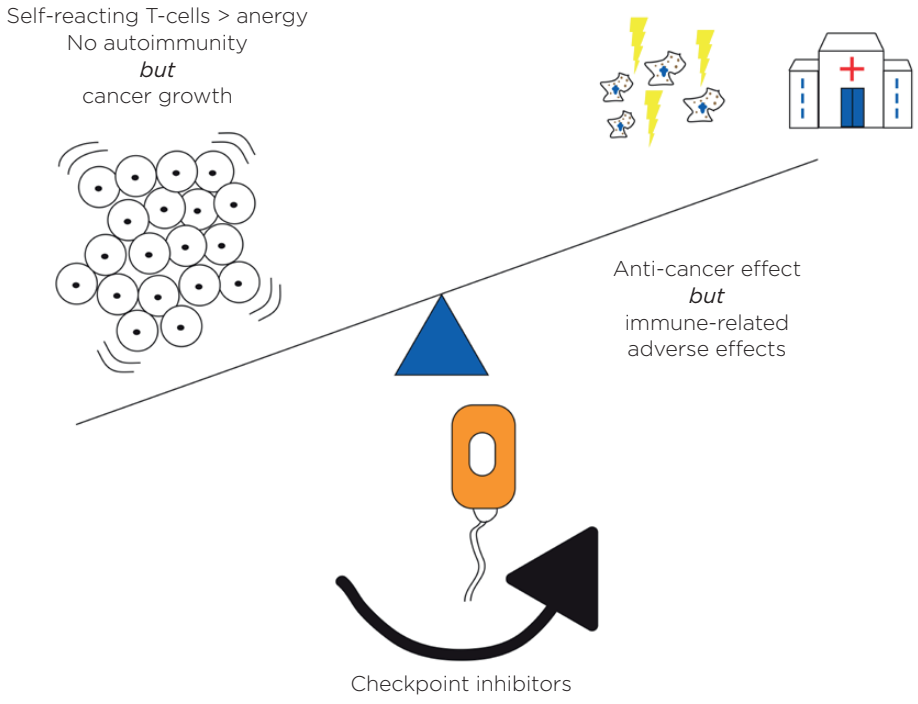
The oncology literature frequently reports all pituitary abnormalities as hypophysitis, which can make it challenging to truly understand the impact of checkpoint inhibitors on the pituitary. In fact, from the viewpoint of an endocrinologist, there are three distinct presentations: hypophysitis (which usually, but not always, leads to hypopituitarism), hypopituitarism and isolated ACTH deficiency. A recent position statement, endorsed by the Society for Endocrinology, has suggested an endocrine classification on this basis (see Table on **page 10**). Using this classification will ensure correct endocrine management.[5]

A key practice point, therefore, is that whilst some people will present with symptoms of an actual hypophysitis, with headache, pituitary dysfunction and an enlarged pituitary on magnetic resonance imaging, many more will only have symptoms of adrenal insufficiency due to isolated ACTH deficiency.

'From the viewpoint of an endocrinologist, there are three distinct presentations: hypophysitis (which usually, but not always, leads to hypopituitarism), hypopituitarism and isolated ACTH deficiency.'

Recognition is made more challenging by two points. The first is that symptoms of adrenal insufficiency, especially fatigue, are common in people with advanced cancer even in the absence of endocrine dysfunction, so a high degree of clinical suspicion is needed to ensure prompt measurement of morning cortisol when there are even subtle changes in symptoms.

The relationship between anti-cancer effects of checkpoint inhibitors and immune-related events, including endocrine dysfunction. Reproduced from Anderson & Morganstein[8] under CC BY-NC 4.0 licence. ©The Authors 2021.



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Proposed diagnostic classification and criteria for immune checkpoint inhibitor-induced pituitary endocrinopathy. Reproduced from Percik *et al.*[5] under **CC BY 4.0 licence**. ©The Authors 2023.

Pathology/description	Proposed diagnostic criteria
1. Hypophysitis: active pituitary inflammation	1. Pituitary enlargement on MRI with or without headache, or combination of headache and new onset hypopituitarism (see point 2).
2. Hypopituitarism: deficiency of two or more pituitary axes, with or without evidence of hypophysitis	2. Deficiency of two or more pituitary axes: deficiency defined as: • 08.00–10.00h cortisol below assay-specific reference range with non-elevated ACTH, in the absence of exogenous glucocorticoids† • Free thyroxine below reference range with non-elevated thyrotrophin • Morning testosterone (males) below reference range with non-elevated gonadotrophins on more than one occasion • Secondary amenorrhoea with oestradiol <100pmol/l and non-elevated gonadotrophins (females premenopause) • Prolactin above or below reference range can support a diagnosis.
3. Isolated ACTH deficiency usually without hypophysitis*	3. 09.00h cortisol below assay-specific reference range with non-elevated ACTH, in the absence of exogenous glucocorticoids.† Other pituitary axis intact.

*Exclude use of exogenous glucocorticoids prior to diagnosing isolated ACTH deficiency, reconsider if failure of adrenal recovery after the standard withdrawal approach.

†In those with ongoing symptoms of adrenal insufficiency but 09.00h cortisol within the reference range, consider an insulin tolerance test to confirm or rule out hypothalamic-pituitary-adrenal axis dysfunction if no contraindications.

The second is that steroids are frequently used in oncological practice. A third of patients treated with a checkpoint inhibitor will need high-dose steroids to treat non-endocrine adverse events.[6] Checkpoint inhibitors are increasingly used in combination with conventional chemotherapy, where dexamethasone is frequently used as an anti-emetic. Thus, careful assessment is required when assessing cortisol levels to ensure correct diagnosis of ACTH deficiency as opposed to adrenal suppression. This is especially important when the checkpoint inhibitor is used as part of a neo-adjuvant treatment approach, to ensure that adrenal insufficiency is not missed prior to elective surgery.

'Prompt assessment of cortisol levels at the onset of symptoms remains vital to ensure those developing ACTH deficiency are treated with glucocorticoid replacement before the onset of adrenal crisis, wherever possible.'

CLINICAL MANAGEMENT

All guidelines and the Summary of Product Characteristics for checkpoint inhibitors recommend monitoring for changes in thyroid function before and during treatment. This leads to the question of whether there is clinical utility in monitoring cortisol levels during treatment to allow earlier detection of ACTH deficiency. One study suggested that, when it was practical to monitor early morning cortisol levels, it was possible to diagnose ACTH deficiency earlier. When detected it can safely be managed as an outpatient with prompt glucocorticoid replacement. However, more recent data show that the onset of adrenal insufficiency is rapid, with a gap from the last normal cortisol to the onset of adrenal insufficiency sometimes as short as a few days.[7]

This suggests that screening is unlikely to be practical, and prompt assessment of cortisol levels at the onset of symptoms remains vital to ensure those developing ACTH deficiency are treated with glucocorticoid replacement before the onset of adrenal crisis, wherever possible. This can be particularly challenging when morning cortisol levels are indeterminate, as synacthen tests will not be reliable, given the rapid onset of ACTH deficiency, and many patients with cancer will not be able to have insulin tolerance tests. Therefore, in this situation, repeat testing after a few days may be most appropriate, covering with glucocorticoid replacement if unwell (for example with a fever). It remains unclear whether partial ACTH deficiency can occur and, in the case of persistent indeterminate cortisol levels, individual clinical assessment is required.

In conclusion, whilst immunotherapy with checkpoint inhibitors represents a major advance in cancer treatment, it is also posing new challenges to endocrinologists who need to be ready to diagnose and follow up this rapidly increasing cohort of patients with life-long iatrogenic endocrine dysfunction.

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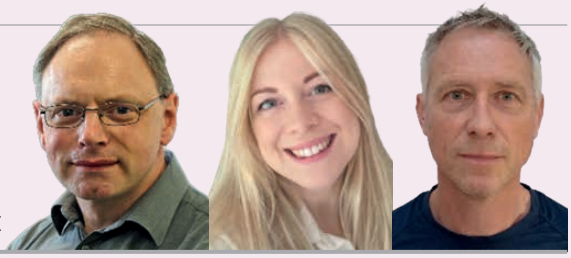
COMBINATORIAL DRUG STRATEGIES FOR THYROID CANCER

WRITTEN BY MARTIN L READ, KATIE BROOKES AND CHRISTOPHER J McCABE



The sodium–iodide symporter (NIS) is the sole known conduit of iodide into human cells. Researchers from the University of Birmingham have identified new combinatorial drug strategies to stimulate NIS activity. These findings have potential clinical application for improving radionuclide-based therapies and imaging across multiple cancer settings.

The first clinical use of radioiodide therapy in 1946 for thyroid cancer was a huge milestone in nuclear medicine and oncology.[1] Since then, β-emitting radioiodide (^{131}I) has become a standard treatment and is widely used to safely, efficiently and specifically destroy remaining thyroid cancer cells post-surgery and to target metastases.

Despite the effectiveness of radioiodide therapy, there is a worrying prediction that the total number of deaths due to thyroid cancer will increase over the next 25 years.[2] Globally, the total mortality for thyroid cancer has been projected to rise by 91.2% by 2050, and this figure is even higher for the continents of Asia (102.4%) and Africa (169.4%).

Poor tumoural uptake of ^{131}I is fundamental to the failure of radioiodide therapy, and is typically diminished in 25–50% of thyroid cancer patients. For example, patients with radioiodide-resistant thyroid cancer, particularly those with metastatic disease, have a life expectancy of only three to five years.[3]

THE NIS: A KEY DRUGGABLE TARGET

To address this clear unmet medical need, several research groups have investigated the NIS as a key druggable target to enhance radioiodide therapy.[4] This is because the NIS is the sole transporter responsible for specific cellular iodide uptake at the plasma membrane, and hence critical for ^{131}I internalisation. It is also well-recognised that any alterations in NIS activity caused by diminished NIS expression and/or mislocalisation away from the plasma membrane can lead to tumoural radioiodide refractoriness (Figure 1).

The inherent complexity of how NIS activity is dysregulated in cancer presents a major hurdle for the thyroid research community to overcome. For instance, the NIS is regulated by a plethora of mechanisms, including transcription factors, post-translational modifications, histone acetylation, DNA methylation, hormonal signalling, autophagy, oxidative stress and microRNAs/long non-coding RNAs.[4] Many of these pathways are altered in thyroid tumour cells. which adds to the complexity of developing NIS-targeting therapeutic strategies.

IDENTIFYING NIS TARGETABLE PATHWAYS

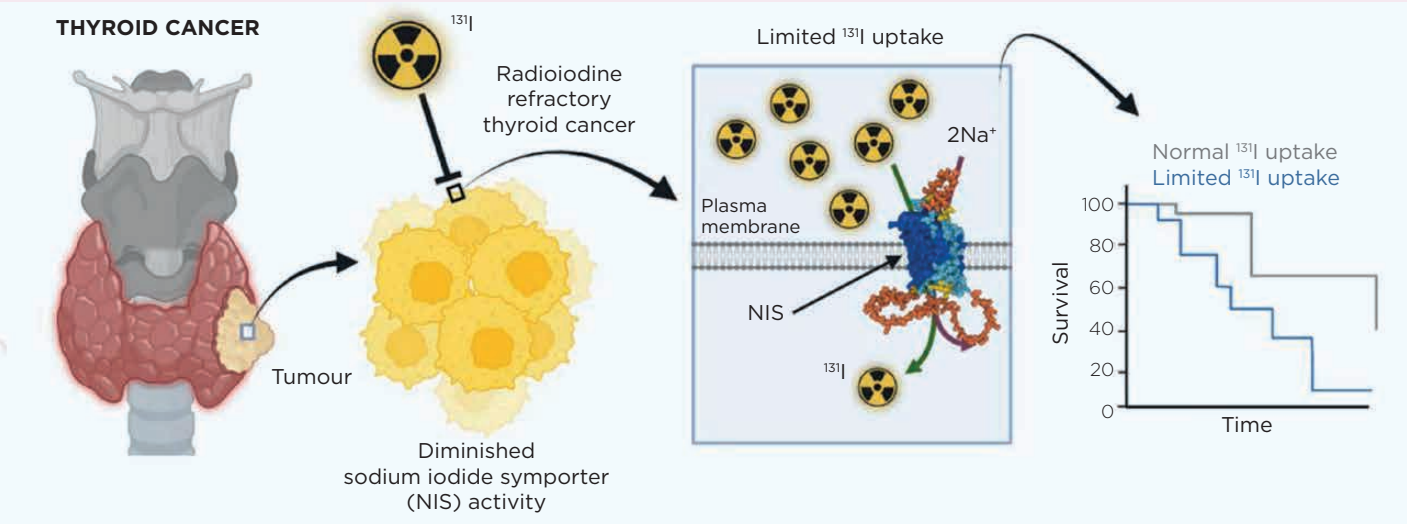
We recently undertook a series of drug screening studies to identify the key targetable pathways modulating NIS activity in thyroid cancer cells.[5–7] One important finding was the identification of cellular processes capable of modifying radioiodide uptake outside the canonical pathways (e.g. BRAF/MEK signalling) of NIS processing.

For example, we conducted a high-throughput screening of compounds approved by the US Food and Drug Administration.[6] In this study, we enhanced NIS activity and subsequent radioiodide uptake by targeting the ubiquitin–proteasome system using the anti-alcohol drug disulfiram, as well as valosin-containing protein (VCP) inhibitors, including the antihistamine carebastine and antifungal clotrimazole.[6]

An under-studied aspect of NIS processing in thyroid cancer cells is its trafficking to, and retention at, the plasma membrane. In earlier work, we showed that the proto-oncogene PTTG1-binding factor acts as an NIS-interacting protein capable of inducing NIS endocytosis when overexpressed in cancer cells, leading to diminished radioiodide uptake.[8] More recently, we characterised AP2 subunit genes as NIS interactors, implicating the AP2 complex in clathrin-dependent endocytosis of NIS.[7]

Based on these observations, we were then able to successfully demonstrate that the antimalarial drug chloroquine was effective at blocking NIS endocytosis, thereby identifying a promising non-canonical approach to retain NIS at the plasma membrane and enhance radioiodide uptake.[7]

Figure 1. NIS activity is compromised by diminished expression and mislocalisation away from the plasma membrane, which contributes to the failure of ^{131}I as a therapeutic strategy and poor survival rates, particularly in aggressive and metastatic disease. NIS structure reproduced from the **AlphaFold Protein Structure Database** under **CC-BY 4.0 licence.** ©EMBL-EBI and DeepMind Technologies Ltd 2022. Figure created using **biorender.com**.



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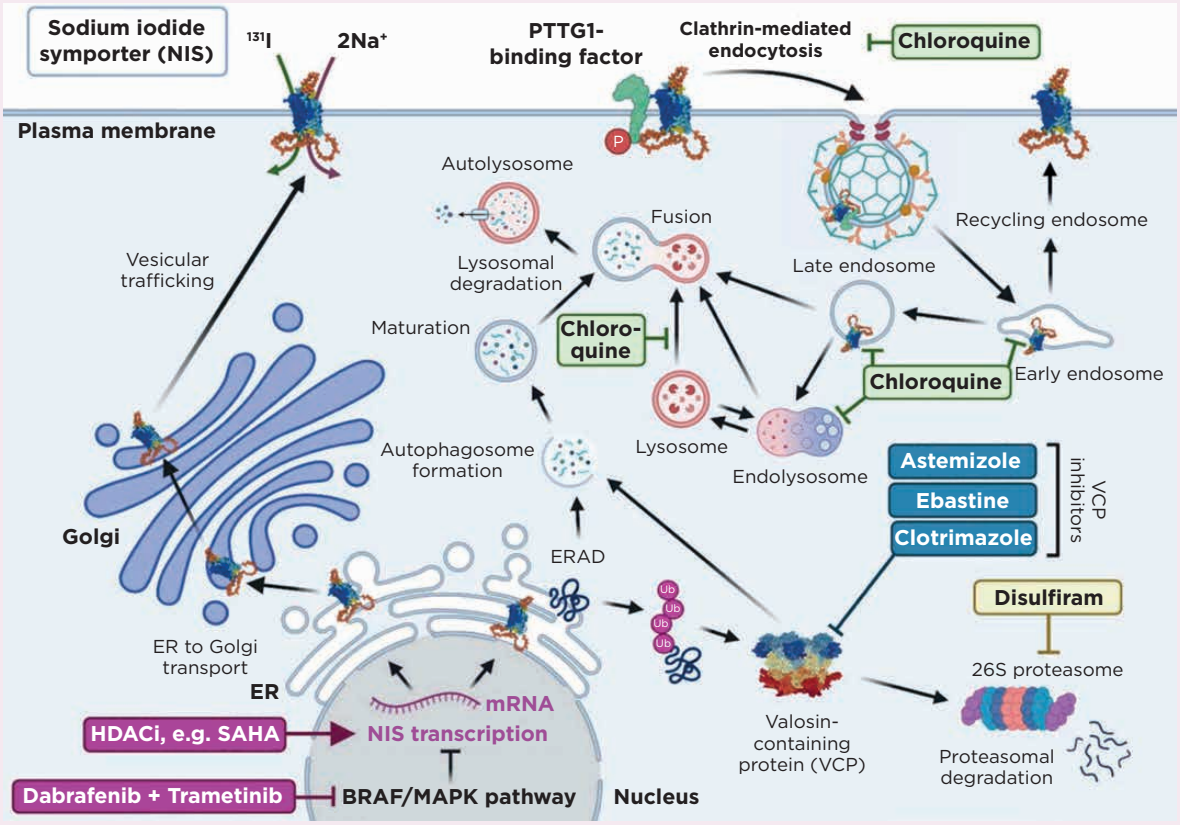

Figure 2. Targetable processes to enhance NIS function and radioiodide therapy. Valosin-containing protein is a critical component of ubiquitin–proteasome and autophagy–lysosome pathways. Chloroquine inhibits fusion of autophagosomes and lysosomes, endolysosomal degradation and endocytosis. Histone deacetylase inhibitors (HDACi; e.g. SAHA) promote NIS transcription. Disulfiram inhibits proteasomal degradation. ER, endoplasmic reticulum; ERAD, endoplasmic reticulum-associated degradation. NIS structure reproduced from the **AlphaFold Protein Structure Database** under **CC-BY 4.0 licence.** ©EMBL-EBI and DeepMind Technologies Ltd 2022. Figure created using **biorender.com**.

COMBINATORIAL DRUG STRATEGIES

Combinatorial drug strategies offer many advantages, including enhanced efficacy, broader targeting and synergistic effects. To date, a wide range of combinatorial drug strategies to target NIS function have been evaluated. In particular, combined targeting of MAP kinase pathways which generally drive tumourigenesis in thyroid cancer (e.g. BRAF/MEK inhibitors, such as dabrafenib and trametinib) has shown some promise clinically in overcoming radioiodine refractoriness.[9] However, issues of drug resistance and adverse events with BRAF/MEK inhibitors remain, and new alternative strategies targeting NIS function are urgently needed.

Due to the complexity of NIS regulation, we recently combined drugs that target distinct cellular processes to maximise the induction of NIS activity (Figure 2). We showed, for instance, that co-treatment of thyroid cancer cells with the histone deacetylase inhibitor SAHA, to induce NIS mRNA, and VCP inhibitors (such as ebastine, astemizole or clotrimazole) led to significant increases in radioiodide uptake compared with SAHA alone.[6] Similarly, combining SAHA with the endocytosis inhibitor chloroquine led to effective and additive increases in radioiodide uptake in thyroid cancer cells *in vitro*,[6] as well as in thyroid glands of wild type BALB/c mice *in vivo*.[7] Encouragingly, we again recently showed that combining SAHA with a metabolite of disulfiram gave robust induction of NIS function in an *in vivo* orthotopic model of breast cancer.[10] Together, our findings indicate that new combinatorial strategies targeting NIS function might represent a feasible option to enhance radioiodide therapy.

FUTURE CONSIDERATIONS

There has been considerable progress in identifying new combinatorial drug strategies capable of inducing NIS function in thyroid cancer cells refractory to radioiodide uptake. The next significant challenge will be to thoroughly examine the biological impact of these new drug combinations on NIS activity in animal models of thyroid cancer.

Of critical importance will be to study the translatable potential of these drugs to enhance radioiodide ablation, and whether further drug refinement by rational design or reformulation is required to achieve sufficient tumour accumulation. It is envisaged that, if progress can be maintained, then better therapeutic options will become available to the clinician, in order to facilitate radioiodide ablation of thyroid cancer in the near future.

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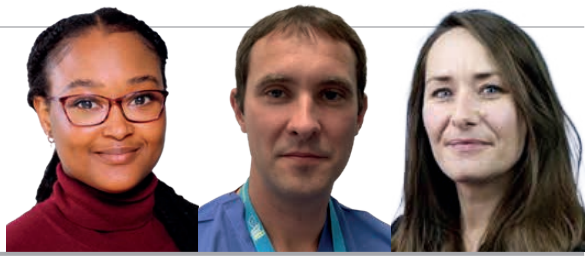
3D BIOPRINTING FOR PRECLINICAL MODELS OF BREAST CANCER

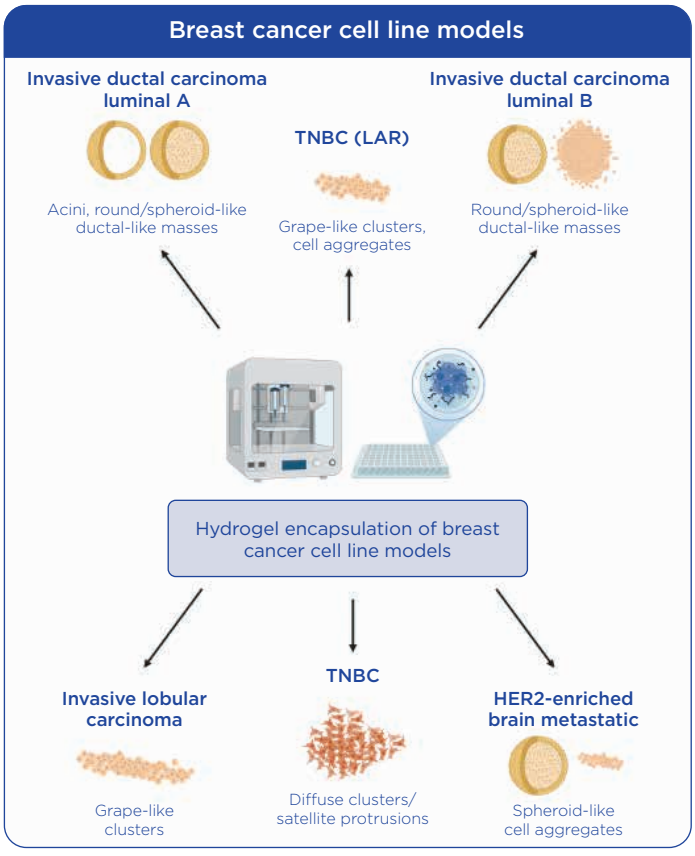
WRITTEN BY STEPHANIE AGBANA, MICHAEL O'REILLY AND MARIE McILROY



Our understanding of the significance of sex hormones as drivers of endocrine-related cancer originates from key historical studies by Beatson (oophorectomy) and Huggins (adrenalectomy) in the treatment of patients with advanced disease.[1,2] Since the late 20th century, there have been tremendous pharmacological advances in targeting the endocrine drivers of the two most common endocrine-related cancers: breast and prostate cancer. Today, patients can expect much more favourable outcomes.

However, resistance to endocrine therapies is inevitable, with approximately 30% of endocrine-treated breast cancer patients experiencing disease progression. Consequently, uncertainties remain regarding the precise steroid drivers of the disease.[3,4] There is, therefore, still much to be understood in the areas of tumour intracrinology, the influence of body composition on steroid production, and cellular responses to complex mixtures of steroid hormones. Of particular relevance in breast cancer research are the poorly understood consequences of menopause hormone therapy use, both by survivors of breast cancer and by individuals at increased risk of developing the disease.[5]

Schema illustrating the various structures formed by breast cancer cell line subtypes when bioprinted in 3D hydrogel. LAR, luminal androgen receptor-positive; TNBC, triple-negative breast cancer. Created with BioRender



A NEW TOOL FOR INVESTIGATING ENDOCRINE-RESISTANT DISEASE

Breast cancer research has traditionally been conducted using a wide range of patient-derived cell lines, reflecting various subtypes. Over the past decade, there has been a rapid expansion in the development of patient-derived xenograft and organoid models of disease. These sophisticated models provide the opportunity to evaluate the tumorigenic potential and tumour cell interactions within the *in vivo* microenvironment. However, the low tumour take-rate and the long duration (>18 months) required for tumours to grow to a sufficient volume are major limitations of these models, especially for hormone receptor-positive breast cancers.

Our group has been exploring the utility of a synthetic, xeno-free hydrogel 3D bioprinting approach to provide a more robust, time-efficient model system for the 3D propagation of breast cancer cell lines and primary breast tumours. Modern drop-on-demand bioprinting systems offer a highly reliable, sterile and reproducible platform for generating 3D tumour structures that accurately recreate the growth patterns of breast cancer subtypes within a matrix that mimics the stiffness of normal breast tissue. This approach enables the modelling of cell–cell interactions, cell–extracellular matrix interactions, and potentially, cell–drug interactions.

'Modern drop-on-demand bioprinting systems offer a highly reliable, sterile and reproducible platform for generating 3D tumour structures that accurately recreate the growth patterns of breast cancer subtypes within a matrix that mimics the stiffness of normal breast tissue.'

Using this platform, we have developed a comprehensive panel of 3D bioprinted breast cancer cell line subtypes, including luminal A and B, lobular, luminal androgen receptor-positive, triple-negative breast cancer and brain metastatic subtypes (see Figure). The structures formed have been characterised to reflect not only hormone receptor status (oestrogen receptor-α, progesterone receptor, androgen receptor) but also the degree of inter-epithelial and epithelial–extracellular matrix interaction (ZO-1, epithelial cell adhesion molecule, F-actin). In our hands, 3D bioprinting of primary tumours closely mirrors the breast cancer subtype (invasive ductal carcinoma or lobular), matching the gold-standard mammary intraductal model.[6]

SEX STEROIDOGENESIS: A UNIQUELY HUMAN CONDITION

Human endocrinology is unique even among mammals, with only humans and certain species of whale undergoing menopausal transition. Importantly, after menopause, a considerable proportion of steroid hormones are of adrenal origin, in stark contrast to rodents, whose sex hormones are all gonadally driven. Thus, *in vivo* models involving oophorectomy and steroid supplementation do not accurately recapitulate the aged, postmenopausal breast cancer environment.

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With this in mind, we have specifically utilised 3D bioprinted models of breast cancer to explore the often-neglected endocrine environment, which is crucial to understanding resistance to oestrogen/oestrogen receptor-targeting therapies. We and others have highlighted the tumour-potentiating role of the postmenopausal androgenic steroid milieu, particularly in the setting of aromatase inhibitor resistance. Androgen excess is well established as perturbing metabolism in women, and renewed interest in adrenal-derived subclasses, such as the 11-oxygenated androgens, as drivers of pathology makes them of particular interest in postmenopausal breast cancers, which significantly associate with metabolic syndrome and obesity.[7]

While we have extensive clinical data highlighting the link between androgens as drivers of disordered metabolism, our understanding of the exact mechanisms remains incomplete. Understanding how the patient's steroid hormone profile impacts tumour behaviour is often neglected. Moreover, typical analysis of steroid hormone impact is often limited to the most potent, yet least abundant, steroids, and focuses on genomic action within the tumour cell.

'Establishing humanised endocrine models of breast cancer will enhance our understanding of both genomic and non-genomic sex steroid actions.'

Studies on the impact of androgen excess on health and disease in women suggest that more abundant, lower affinity ligands are often more significantly associated with the disease state.[8,9]

To address this, we have utilised conditioned medium from primary tumour-associated adipocytes exposed to pro-hormone androstenedione, and subsequently profiled by liquid chromatography tandem mass spectrometry, to evaluate the impact in 3D bioprinted models of disease. The development of more complex endocrine models of resistant breast cancer utilising this platform may help elucidate the interplay amongst the cognate steroid receptors when exposed to physiologically relevant levels of hormones arising in the mammary microenvironment.

CONCLUSION

Establishing humanised endocrine models of breast cancer will enhance our understanding of both genomic and non-genomic sex steroid actions. With the recent 2025 Food and Drug Administration guidelines ushering in a new era of drug discovery, which does not rely on expensive and often ineffective *in vivo* studies, integrating these more physiologically relevant endocrine models could become a reliable and cost-effective new approach methodology in drug development.[10]

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An interview with… RICHARD ANDERSON

RESEARCHING FERTILITY AFTER CANCER TREATMENT

Richard Anderson is Professor of Clinical Reproductive Science at the University of Edinburgh, and a consultant at the Royal Infirmary of Edinburgh. He also provides a specialist endocrinology service to the Edinburgh Gender Identity Clinic. He was founding Co-ordinator of the European Society of Human Reproduction Embryology Special Interest Group in Fertility Preservation, and is a member of the Human Fertilisation and Embryology Authority (HFEA) Scientific and Clinical Advances Advisory Committee.

What inspired you to study reproductive health?
My introduction to scientific research came about through an honours year as a medical student in the Department of Pharmacology in Edinburgh. During that time, I did a project in the MRC Brain Metabolism Unit, then directed by George Fink. This was eye-opening, and I was asked by George if I wanted to stay on to do a PhD, which I did under the supervision of Rory Mitchell. We studied the potential role of GABA in neuroendocrine regulation within the hypothalamus and pituitary, which was a great introduction to how the reproductive system is controlled.

Going back to third year medicine was a bit of a shock after that, but my attachment in obstetrics and gynaecology gave me a first glimpse of what reproductive medicine might be like. I decided to try to make that my career, taking up a SHO post in the Royal Infirmary and the Simpson Memorial Maternity Pavilion. This allowed interactions with the work at the then Centre for Reproductive Biology (CRB). I went on to do a two-year post as a Research Fellow with Fred Wu in the MRC Reproductive Biology Unit, working on a World Health Organization clinical trial in male contraception. That area (and indeed male reproductive endocrinology) has continued to be a significant focus of my work, recently with a trial using testosterone/nestorone gel run by the National Institutes of Health and the Population Council.

During that time, I got to know David Baird and subsequently undertook subspecialty training in reproductive medicine as a lecturer in obstetrics and gynaecology within the CRB, supervised by David. Those were great years, very busy at work with training in reproductive medicine, on-call obstetrics and gynaecology and research work, as well as family life with the birth of my two daughters. I subsequently spent a year in Sam Yen's lab in San Diego (the highlight was living in south California), before returning as a consultant in the MRC Unit, and starting my current post in the University in 2005.

Tell us about your research on cancer treatments and female fertility
Working with David was an introduction to the early days of ovarian tissue cryopreservation for fertility preservation for cancer patients. We developed this clinically from the mid-1990s, with great support from Professor Hamish Wallace in paediatric oncology.

The field of fertility preservation has become an increasing focus of my work since then, leading the ovarian tissue cryopreservation programme. This led to the first birth in the UK for the first woman whose ovarian tissue we replaced, after 11 years in cryostorage. That was a wonderful occasion, and I went to see David to tell him about it. His characteristically gruff response was, 'What took you so long?' (despite his obvious pleasure at the news).

Working with Hamish, analysing long-term reproductive outcomes of girls across south-east Scotland who had had cancer, allowed us to examine the criteria for offering ovarian tissue cryopreservation. We had initially developed these in the absence of any evidence, so it was very reassuring to see how this translated into identifying the right patients.

'I hope that those starting their careers will have opportunities to test the research waters, as combining research with clinical practice has been a huge privilege, and great fun.'

In parallel with this, I was working with David Cameron at the Edinburgh Breast Clinic, exploring the potential value of anti-Müllerian hormone to predict and assess the ovarian toxicity of cancer treatments, and we continue to refine this. The initial study we undertook was a five-year prospective study in women newly diagnosed with breast cancer, so it took a long time to generate data. This was possible with support from being in the MRC Unit.

What are the biggest challenges in this area?
A key aspect that we have particularly focused on is identifying the right patients who may need a fertility preservation intervention. One way we have approached this is by using Scottish national databases for cancer and maternity to identify, on an unbiased population basis, the impact of cancer and its treatment on whether (or not) women subsequently have a baby. This has also allowed us to show that having a baby after breast cancer was not associated with increased mortality, even in women with oestrogen receptor-positive disease.

What exciting developments are happening in the field?
It has also been very rewarding working with Evelyn Telfer in her work on *in vitro* follicle growth. The next step is whether the mature oocytes that are generated can be successfully fertilised, and we have an application for this under review by the HFEA at the moment.

What advice would you have given your younger self?
I consider myself enormously lucky to have had the opportunity to work in two MRC units, and particularly to have worked in the CRB under its various names. This environment, where it is completely natural for clinicians and discovery scientists to be rubbing shoulders daily, has been very rewarding, and I continue to lead some lab-based research. The decision to take four years out of my medical undergraduate studies is one that I have never regretted, although I suspect my parents rather despaired that I would ever complete my medical degree. I hope that those starting their careers will have opportunities to test the research waters, as combining research with clinical practice has been a huge privilege, and great fun.

An interview with…
RACHAEL GUENTER
DEVELOPING MODELS AND THERAPIES FOR NETs

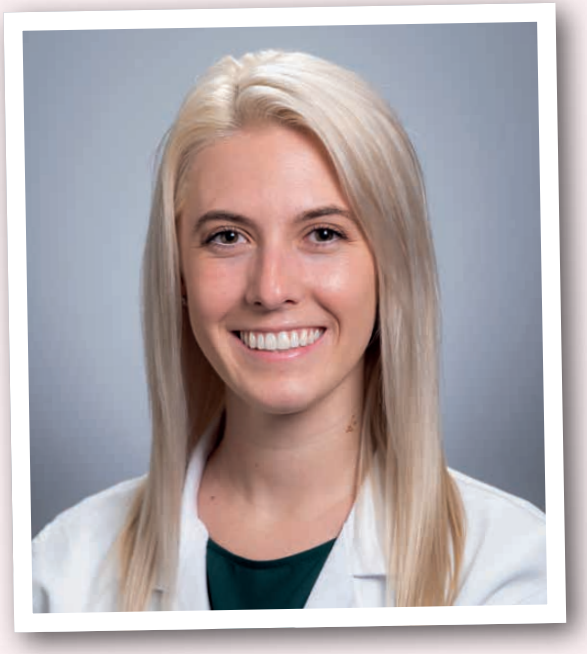
Rachael Guenter has been a faculty member of the Department of Surgery in the University of Alabama at Birmingham (UAB), USA, since 2023. She investigates the molecular mechanisms of neuroendocrine cancer and pancreatic ductal adenocarcinoma to develop novel therapeutic approaches, and has published strategies to improve the detection of neuroendocrine tumours (NETs).

Currently, Rachael is focusing on the development of novel theranostic agents for pancreatic cancers and understanding the role of Notch signalling in gastroenteropancreatic NETs. She is particularly interested in developing new treatments for patients with unresectable, advanced disease. She also advocates to increase awareness of neuroendocrine cancer and aims to provide accurate information for patients and the general public.

Could you tell us about your career so far?
As I was working towards my undergraduate degree in chemical and biomolecular engineering at North Carolina State University in 2017, I was fortunate to participate in research. I worked in two labs: one that studied the genetics of plant pathology and one that studied novel systems for water purification. Being exposed to research in college led me to decide to pursue a career in research.

'I am also excited about the growing number of young scientists who are dedicating their careers to studying neuroendocrine cancer…'

After completing my PhD in cancer biology at UAB in 2021 with Herbert Chen, I undertook a postdoctoral fellowship, and subsequently became a faculty member to run a lab alongside J Bart Rose, who was my mentor for my fellowship. We lead a basic science lab that studies neuroendocrine cancer and pancreatic ductal adenocarcinoma. I enjoy the complexity and diversity of our research projects. The other exciting part of my career is mentoring younger scientists and inspiring them to pursue careers in research.



What inspired you to choose science?
My Dad (a brilliant mechanical engineer!) sparked my inspiration for science, and my undergraduate research experiences solidified the decision to become a scientist.

What do you enjoy most about your work?
I love the process of using the scientific method to achieve a discovery which generates knowledge that no one else yet knows.

Tell us about your current project(s)?
I have two main projects. The first is understanding how the Notch signalling pathway aids the survival and growth of pancreatic NETs. Notch is a developmental pathway that cancer cells can take advantage of to survive. The second project involves attaching radioactive molecules to a nanobody in order to detect and target a specific protein, called calreticulin, on the surface of cancer cells.

What inspired you to lead the NET Models consortium?
I have studied NETs in the laboratory for over eight years, and so have experienced the difficulties associated with a lack of appropriate preclinical models. It is difficult to understand the molecular mechanisms of a tumour when we cannot properly model it in the laboratory. I believe future discoveries require more accurate research models.

What are the biggest challenges in this field?
Lack of NET-specific funding, lack of disease awareness, and lack of appropriate preclinical models. Awareness will help build funding, which will then help build research tools.

What exciting developments are underway?
As a leader of the NET Models consortium, I of course believe that what this group is doing is incredibly exciting. We are driving an international effort to expand the horizon of NET research by supporting the development of NET models. I am also excited about the growing number of young scientists who are dedicating their careers to studying neuroendocrine cancer, and the integration of patients into research decision-making.

How will the consortium help to continue these developments?
The consortium is building a movement to advance NET models. Through this movement, we are fostering international collaborations, as well as the careers of NET scientists at all stages. The consortium prioritises a diverse set of minds and experiences, and so we integrate not only NET scientists, but also patients, physicians, patient advocates, nurses and veterinarians. By working together, we can solve some of the biggest challenges in the field of NET research.

What are you most proud of in your career, so far?
I am most proud of the knowledge that my research team and I have contributed to cancer research, especially neuroendocrine cancer.

What advice would you give your younger self?
When I was younger, I wanted a career that would fund my expensive hobby of horse riding. As it turns out, research is NOT that career – but, the discoveries I get to make are worth it. I would tell myself to trust in timing, and that everything does happen for a reason.

Attend the Society's annual **OBESITY UPDATE**

Stay up to date with the latest research and clinical techniques in obesity therapy

Monday 1 December 2025

Crowne Plaza, Nottingham

REGISTER TODAY

Obesity **UPDATE**

Introducing the third edition of the **COMPETENCY FRAMEWORK FOR ADULT ENDOCRINE NURSING**

The Society for Endocrinology has unveiled the third edition of its ***Competency Framework for Adult Endocrine Nursing***, marking a significant advancement in the professional development of endocrine nurses.

The updated framework reflects the evolving landscape of endocrine nursing. It aims to enhance patient care by providing a structured pathway for nurses and nursing support professionals at all levels of expertise.

A DECADE OF PROGRESS

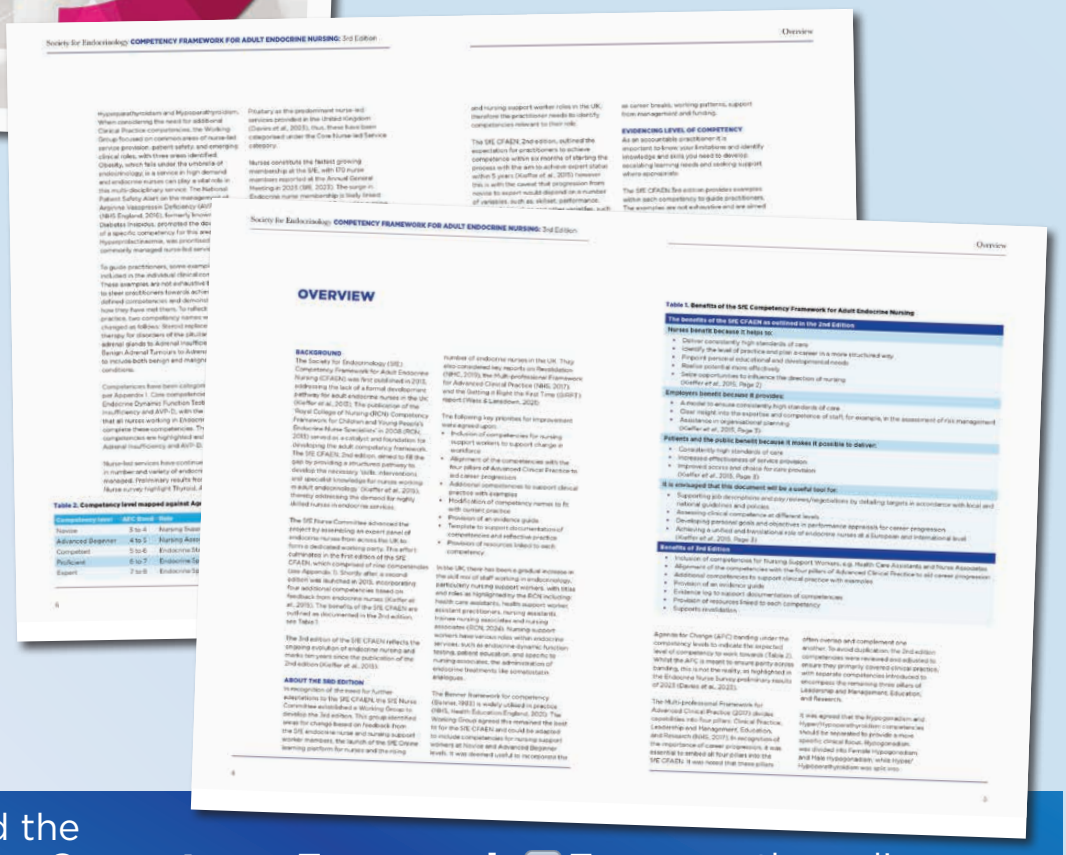
Originally developed in 2013, the framework was designed to outline the core knowledge, skills and interventions specific to adult endocrine nursing. The second edition was released in 2015, and expanded the framework to include competencies in managing conditions such as benign adrenal tumours, hypo- and hyperparathyroidism, osteoporosis and polycystic ovary syndrome.

The third edition builds upon this foundation by incorporating the latest clinical practice and addressing emerging areas in endocrine care.

2 ADDITIONAL COMPETENCY LEVELS

A notable enhancement in the third edition is the adoption of the Benner 'novice to expert' model, which spans five levels of clinical competence. This stratification allows for a more tailored approach to nurse development, accommodating individuals from entry level positions to seasoned professionals, providing a flexible and personalised roadmap for career development. The new levels 'novice' and 'advanced beginner' extend the existing levels as follows:

- Novice
- Advanced beginner
- Competent
- Proficient
- Expert.



8 NEW COMPETENCIES

The updated framework introduces eight new competencies, reflecting the broadening scope of endocrine nursing. The inclusion of these areas ensures that the framework remains aligned with current clinical demands and patient needs. The new areas include:

- Arginine vasopressin deficiency (AVP-D)
- Hyperprolactinaemia
- Obesity management
- Late endocrine effects of cancer treatment
- Transition from paediatric to adult endocrine services.

NEW DIGITAL PLATFORM FOR NURSE MEMBERS

The Society recognises the importance of accessible resources, and has launched an online platform to accompany the framework, which is available to all nurse members. Access this interactive tool for:

- Downloadable framework and competency documents
- The full set of 21 competencies
- Editable evidence logs to track development
- Real-life examples to guide application of the competencies
- Networking and mentorship opportunities within the endocrine nursing community.

You can download the **Third Edition of the Competency Framework**. To access the online platform ensure that you have an up to date membership for 2025.

Have your say in the STATE OF ENDOCRINOLOGY 2025 SURVEY

The State of Endocrinology 2025 survey is set to generate recommendations to help meet endocrinology's future clinical and research needs.

The initiative is led by the European Society of Endocrinology (ESE), in collaboration with the national endocrine societies that form the ESE Council of Affiliated Societies, which include the Society for Endocrinology. The recommendations will support advocacy efforts aimed at national and European healthcare decision makers.

The survey is structured around different sections that relate to roles within clinical endocrinology and endocrine science. It will take on average 20 minutes to complete, depending on your professional profile. You can decide to save it part-way through and return to finish it later. You will need to do that from the same device.

The questions are in English, but you can use any other language in your open text replies, as they will be translated.

Take the survey before Tuesday 15 July (23.59 CEST).

Taking flight HYPOPARA BUTTERFLY LAUNCHED ON AWARENESS DAY!

To recognise World Hypopara Day on 1 June 2025, we're delighted to have launched the completed **Seeds of Change Hypopara Butterfly** – working in partnership with Ascendis Pharma UK and Parathyroid UK.

Held annually, World Hypopara Day is an important milestone dedicated to raising awareness of the impact of hypoparathyroidism on those living with the condition and the professionals who care for them.

Earlier this year, many of you contributed to the creation of the Hypopara Butterfly, a mural capturing artistic representations of stories and experiences from across the hypopara community. It incorporates insights gathered from endocrinologists who support people living with hypopara: the challenges they face in managing the condition and the hopes for the future of patient care.

The first iteration was revealed at the SfE BES conference 2025 in Harrogate, and we were excited to see the completed mural on show on 1 June.

The Butterfly officially launched on Parathyroid UK's **World Hypopara Day Party Facebook page.** The hypopara community and everyone else can view the final mural there, along with the insights built into it.

Go to **Seeds of Change** to find out more, including how to make a bespoke #MyHypoparaButterfly image and how to repost the original with a personalised caption, to raise awareness.

Seeds of Change is a disease awareness initiative developed by Ascendis Pharma UK Limited and supported by Parathyroid UK and Society for Endocrinology. The words, statements and images on the Hypopara Butterfly have been shaped by lived experiences submitted by the hypopara community.

Wondering how to **GET MORE INVOLVED WITH THE SOCIETY?**

The Society for Endocrinology is the UK home of endocrinology. We bring together the global endocrine community to share ideas and advance our discipline. The Society for Endocrinology's mission is to enable endocrinology's potential to advance science and health.

As a membership organisation, we support a thriving member community of around 3000 scientists, clinicians and nurses who work with hormones throughout their careers. We do this by providing funding, networking opportunities, training events, educational resources and more. We also engage policymakers, journalists, patients and the public with hormone science to encourage informed health decisions, and to demonstrate the value of endocrinology to the wider world.

There are many ways you can connect and get involved with the Society from events to publishing, and everything in between.

BENEFIT FROM BEING A MEMBER

Membership is at the heart of the Society and the key way to engage with us. Membership enables you to stay at the forefront of endocrinology, collaborate with those in the field, apply for grants and benefit from other career development opportunities. To join the Society, you simply select your membership category and complete the application form on our website. As soon as you receive confirmation from us, you will be ready to make the most of your member benefits. We're happy to help you out with any queries – email **members@endocrinology.org** for more information and support.

PUBLISH IN A SOCIETY JOURNAL

We publish cutting-edge research and best practice in the field of endocrinology through seven official journals and conference abstracts. Whether you are a scientist, a clinician or a nurse, you can engage with the Society by submitting your papers to one of our many publications ranging from *Clinical Endocrinology* to *Endocrine Connections*.

CONNECT AND COLLABORATE

The Society hosts a variety of events throughout the year, catering to healthcare professionals, researchers and trainees in the field of endocrinology. These events include annual conferences, training courses, networking events, workshops and masterclasses.

There are plenty of opportunities for professionals to connect, collaborate, and share best practice in endocrinology.

SUPPORT THE FUTURE OF HORMONE HEALTH

Another way you can get involved with the Society is by offering support in the form of a donation. Donations help fund vital research, support early-career endocrinologists, and promote accurate public understanding of hormones and endocrine disorders. Whether through direct contributions, fundraising, purchasing merchandise, or leaving a legacy, every gift strengthens the endocrine community.

Find out more at **www.endocrinology.org**

Society for Endocrinology
UPCOMING EVENTS

HORMONE DEPENDENT CANCER 2025

17–18 November 2025 York

Learn from experts at this exciting all-new event for autumn, as we introduce Hormone Dependent Cancer 2025. This meeting is dedicated to fostering collaboration and discussion across all fields working on hormone-dependent cancers.

Aimed at early-career participants, Hormone Dependent Cancer is a key opportunity for you to explore the latest advancements in research, clinical techniques and therapeutics, while networking with world-leading experts and peers.

The meeting will feature thought-provoking and exciting sessions, and aims to promote resource sharing within the community, to drive scientific progress.

You will be able to engage in career development workshops, present your own research and dive into topics such as genomics, immuno-oncology and innovative cancer models.

Find out more and register today

"We are delighted to launch the inaugural UK Hormone Dependent Cancer meeting. This early career-focused event is an unmissable opportunity for PhD students, post-doctoral researchers, research assistants, fellows and junior group leaders in breast, prostate and other hormone-linked cancers to hear about the latest cutting-edge developments in the field, network with research leaders, attend tailored careers workshops and showcase their research to other delegates. We hope to see you in York!" Hormone Dependent Cancer founding group

NATIONAL TRAINING SCHEME FOR THE USE OF RADIOIODINE IN BENIGN THYROID DISEASE

23 September 2025, Birmingham

This established, one-day workshop is on the use of radioiodine in the treatment of benign thyroid disease. The course is an essential component of the national training scheme, aiming to allow application for ARSAC certification for iodine-131 administration for the treatment of benign thyroid disease.

Find out more and register today

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ENDOCRINE ACADEMY 2025

Endocrine Academy comprises Thyroid Ultrasound, Clinical Update and Endocrine Nurse Update. These exciting training events will help you continue your learning and development this autumn. Each has been designed to enhance your skills and keep you up to date with the latest developments in endocrinology.

THYROID ULTRASOUND
2 November 2025, Stratford-upon-Avon

This one-day course combines knowledge and hands-on experience of thyroid ultrasound. It is aimed at all endocrine specialists from registrar level to experienced consultants.

Previous attendees have found the practical sessions highly valuable, allowing them to apply theoretical concepts to real-life scenarios.

> *The Thyroid Ultrasound event offered an outstanding combination of hands-on practice, interactive learning and expert-led discussions. The structured approach, from theory to practical application, provided a deeper understanding of ultrasound techniques and clinical decision-making. A truly invaluable experience for attendees',* 2024 attendee

CLINICAL UPDATE
3–5 November 2025, Stratford-upon-Avon

Clinical Update offers three days of gold-standard endocrine training and essential updates for consultants. It is delivered as lectures and interactive workshops which, over a three-year cycle, cover the endocrinology topics of the national curriculum in endocrinology and diabetes, issued by the Joint Royal Colleges of Physicians Training Board.

The interactive nature of the sessions and opportunity to get questions answered is what makes this event a must-attend for new clinicians, with a focus on real-life cases. In summary, 'Clinical Update provided an outstanding opportunity for interactive learning, practical case discussions and meaningful networking. The workshops were engaging, the expert panels insightful, and the atmosphere both welcoming and inspiring. It was a truly enriching experience for all involved.'

ENDOCRINE NURSE UPDATE
4–5 November 2025, Stratford-upon-Avon

Aimed at both established and new-to-post nurses, this two-day event focuses on best practice and the latest developments in endocrine nursing. The event will feature interactive sessions, lectures, workshops and networking opportunities.

This event offers attendees the chance to connect with colleagues, exchange best practice and build a strong professional support network, concluding, 'The Endocrine Nurse Update provided an invaluable opportunity for professional development, fostering meaningful connections among colleagues and experts. Engaging discussions, insightful presentations and a welcoming atmosphere made learning both enriching and enjoyable. It was a truly inspiring experience for all involved.'

Registration for Endocrine Academy 2025 is open now! **Find out more**