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The Endocrinologist

THE NEWSLETTER OF THE SOCIETY FOR ENDOCRINOLOGY • ISSUE 90 WINTER 2008/09

SPECIAL ISSUE
Comparative Endocrinology

EDITORIAL

Welcome to the winter edition of *The Endocrinologist*. This issue has a special focus on comparative endocrinology, as you may have noticed from the farmyard scene on the cover. How many endocrine conditions can you spot? The trouble with animals is that their disorders can present rather differently from the human forms of the disease – so some caricatures are a mixed metaphor to help you along! More hints can be found if you turn to pages 8-9 where you will find a series of 'Did you know…..?' snippets about the endocrinology of fish, birds and domestic animals as well as a couple of in-depth articles about thyroid disease in cats and 'Cushing's syndrome' in dogs on pages 10-12. It's fascinating stuff. This issue also carries reports on grant funding (page 6), and a letter from Richard Dyer about the NO ('New Organisation') in bioscience (page 7). Definitely worth a read.

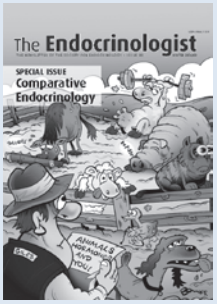
I am very excited about the new Society for Endocrinology Archive. This contains papers from *Journal of Endocrinology* all the way back to issue 1, as well as a full archive of the other journals published by the Society. It is a fantastic resource, which will be available free of charge until January 2009. You can find out more on page 4. It has long been a complaint of more 'senior' endocrinologists that the early work is simply ignored by each new generation of researchers. There really will be no excuse, now that it is so easy to access the archive. Well done to all the staff and Council for having the vision to undertake this mammoth project!

Communicating science features strongly in this issue. On page 13, we learn about the Voice of Young Science, a group organised by Sense About Science, who organised a media workshop to bring young researchers together with journalists. Meanwhile, page 16 looks at the Society's activity in communicating science to a wider audience, at an event on obesity at the recent BA Festival of Science at Liverpool. Hotspur also relates his personal experience of communication problems at conferences on page 15!

This is my last issue as Editor. It has been a lot of fun. We didn't quite achieve my aim of getting *The Endocrinologist* featured as the guest publication on 'Have I Got News for You', but the competition was extremely fierce. I am very grateful to all the staff in the Bristol office, and especially Andy Lowe for his gentle but persistent nagging. John Newell-Price takes over the helm in January and I can only wish him as much fun in the role as I have had.

Very best wishes for the New Year to you all.

JOY HINSON
(j.p.hinson@qmul.ac.uk)



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Deadline for news items for the Spring 2009 issue: **9 January 2009**. Please send contributions to the above address.

Improved preprint service

We use our accepted preprints service to post authors' manuscripts online immediately they are accepted for publication, before copy editing, page layout and proofing. This places them in the public domain well in advance of final publication.

Our new, much improved, accepted preprints system means that preprints will now go online within 24 hours of acceptance and, as soon as they appear, they will be indexed on PubMed, with DOIs (Digital Object Identifiers) assigned and metadata lodged with CrossRef, so that they can be immediately located and cited.

When the definitive formatted version is published in a Society journal, the article is automatically removed from the list of accepted preprints and placed in the table of contents for the relevant issue.

2010 MEDALLISTS

Following a call for nominations, and a subsequent ballot by the Awards Committee, the following medallists have been selected for 2010. All will give their lectures during the Society's BES 2010 meeting.

Dale Medal Professor S O'Rahilly, Cambridge

Transatlantic Medal Professor S Melmed, Los Angeles, CA, USA

Society for Endocrinology Medal Professor W Arlt, Birmingham

Hoffenberg International Medal (previously known as the Asia & Oceania Medal and International Medal) Professor T Yoshimura, Nagoya, Japan

European Medal Professor B Allolio, Würzburg, Germany

Young Endocrinologist prize lectures

The 2009 winner of the clinical prize lecture is Dr Thomas Barber (Oxford) for 'In search of the genetic basis of polycystic ovary syndrome and its metabolic consequences', and the basic science prize winner is Ms Georgia Papacleovoulou (Edinburgh) for 'An anti-inflammatory role of IL-4 in the human ovarian surface epithelium'. Both will present their lectures during the Society BES meeting in Harrogate next March.

All Society for Endocrinology Clinician-in-Training and Scientist-in-Training Members can apply for these awards, the honorarium for which is £2500. Don't miss the next call for abstracts for these awards in mid-2009.

CLINICAL UPDATE

Clinical Update 2008 was the second in a series of residential events providing essential clinical training for all trainees and new consultants in endocrinology.

The event, held in Bristol in October, attracted 192 delegates. Programme Co-ordinator Dr Steve Ball was delighted, remarking, 'Yet again we have generated a collegiate atmosphere to give delegates an effective forum for networking with peers and established endocrinologists.'

The 7 didactic lectures and 16 topics were taken from the PMETB/SAC curriculum for diabetes and endocrinology. Over a 3-year period, the programme covers the entire curriculum and is regarded as the UK's premier national training event. Workshops covered each topic and consisted of a seminar by a leader in the field and case presentation by a delegate.

You will find details of Clinical Update 2009 (Manchester, 2-4 November) on the Society's website shortly.

WITH REGRET

We are sorry to announce the recent death of Professor I MacIntyre. In the 1960s, Professor McIntyre conducted pioneering work on calcitonin in relation to bone. We hope to publish an obituary in the next issue.

Endocrine retreat success

The Society's first Autumn Endocrine Retreat took place in October at Milton Hill Hall in Oxfordshire. Its aims were to foster scientific interactions between trainee members of the Society in a friendly environment, to stage keynote lectures from established endocrine researchers on topical subjects, and to provide an informal setting for presenting data and discussing approaches to research. The event was a huge success, achieving every aim with flying colours. We hope to bring you a fuller report in the next issue.

Congratulations

We are delighted to announce that the following Society members have been awarded Chairs: Karim Meeran of Imperial College London; Marta Korbonits of Barts and the London School of Medicine and Dentistry; Karen Chapman of the University of Edinburgh; and Megan Holmes, also of the University of Edinburgh.

SOCIETY CALENDAR

10 February 2009
Society for Endocrinology Clinical Cases Meeting
(in association with the RSM)
London

16-19 March 2009
Society for Endocrinology BES Meeting
Harrogate

7-9 September 2009
Society for Endocrinology National Endocrine Nursing Conference
Newcastle upon Tyne

2-4 November 2009
Society for Endocrinology Clinical Update
Manchester

DIPLOMA IN CHARITY ACCOUNTING

We congratulate Pat Barter, Finance and Administration Director based in the Bristol office, who has obtained her Diploma in Charity Accounting. The Diploma acknowledges specialist expertise and experience in charity accounting and finance at the highest level.

New Society archive: search back to 1939!

▶ The Society for Endocrinology is pleased to launch its new online archive, available from January 2009, charting the history of endocrinology for the past 70 years.

The Society for Endocrinology Archive is a new online collection of material published from 1939 to late 1997. It is hosted by online provider HighWire Press on the same platform as our more recent articles, and the PDFs are text-searchable using Acrobat.

The archive includes papers from *Journal of Endocrinology* from volume 1 in 1939 to September 1997, from *Journal of Molecular Endocrinology* from volume 1 in 1988 to August 1997 and from *Endocrine-Related Cancer*, the newest journal, from 1994 to 1997. There are over 10 726 articles, or 93 000 pages, in this one archive.

All the papers published from 1997 up to 12 months before the current issues will continue to be available free to all users. These papers had already been published electronically and so required no extra investment from the Society.

Why did the Society digitise the back volumes? As Sue Thorn, the Society's Executive Director, says, 'The Society realised that, by making the full archive available online, we would be providing an important service to endocrinologists worldwide.' The Society is dedicated to the advancement of scientific and clinical education and research in endocrinology. The archive will help fulfil this mission and provide the endocrine community with a valuable archive of key papers that are relevant to endocrinologists today.

Key papers within the archive will be available online free until January 2009. To find out more go to www.endocrinology-journals.org. The Society invested heavily in the digitisation project and so, from January 2009, access will be on payment of a reasonable fee. Access can be purchased outright by institutions for perpetual use, subscribed to annually or purchased with the Society for Endocrinology three-journal package at a discounted rate.

This project has been carried out in collaboration with HighWire and with support from libraries at the University of Bristol, Roslin Institute and AstraZeneca, who supplied the copies of the early volumes.

It is a major achievement for the Society for Endocrinology, a small not-for-profit publisher, who is now able to rival larger publishers in providing the community with a superb online archive. Selected papers and highlights from the archive will follow in the spring issue of *The Endocrinologist*.

Clinical Endocrinology archive

As part of a major digitization project involving 500 scholarly journals, Wiley InterScience made the full *Clinical Endocrinology* archive from volume 1 issue 1 1972 live earlier this year. Clinical Endocrinology is the official clinical journal of The Society for Endocrinology and is published by Wiley-Blackwell.

The full archive is available on the Wiley InterScience portal, available at http://www3.interscience.wiley.com/journal/117998163/home

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Grants update

For information on all the Society's grants and awards see www.endocrinology.org/grants.

Undergraduate Essay Prize 2009

This annual prize is awarded to the best essay on any topical aspect of endocrinology. It must be the original work of the candidate and written within the current academic year by an undergraduate student. It is open to all undergraduate students currently registered at an institution of higher education. The closing date for applications is 2 March 2009.

Lab Visit Grant for Young Endocrinologists

Miss Joanne McNelis has been awarded £1980 to visit Institute de Pharmacologie, Moléculaire et Cellulaire, CNRS, Valbonne, France.

Sponsored Seminar Grants

Dr Jenny Pell of the Babraham Institute, Cambridge was awarded £2000 for a seminar held on 26/27 June 2008.

Conference Grants

At the August 2008 deadline, members were invited to apply for a grant to attend ICE 2008 in Rio de Janeiro. We are pleased to say that over 30 grants were awarded for this meeting alone. Paid members earning less than £50 000 per annum are eligible to apply for our conference grants; the next deadline is 15 April 2009. The Society has recently altered the ruling for these grants. Members can now apply for an overseas conference grant once every calendar year (previously it was once every 12-month period).

Grant reports

The recently re-established North West Endocrine Society was delighted to receive a **Sponsored Seminar Grant** from the Society for Endocrinology to support our first meeting in Manchester last May. We had a multidisciplinary approach, inviting endocrinologists, specialist nurses, basic scientists, clinical biochemists, pituitary and endocrine surgeons. The topics were carefully chosen to appeal to all disciplines and included symposia on Cushing's disease and parathyroid disease. With the Society's support, we allocated one prize to the best oral presentation and two prizes for poster presentations. About 60 people attended and feedback was extremely positive. Attendees favoured the multidisciplinary approach, the mixture of external and local speakers, and the opportunity to network with local colleagues.

TARA KEARNEY

The **Small Grant** *received was invaluable in supplementing an ongoing PhD project. The project was designed to study whether dendrites in magnocellular neurones contain functionally distinct vesicle pools. To look at vesicle dynamics in live cells, we used transgenic vasopressin-eGFP rats. Vasopressin-eGFP was visualised using the confocal imaging system. The award was used towards the costs of these transgenic animals and for imaging time on the confocal system. Even though this work is still in progress, the first results were presented at this year's British Society for Neuroendocrinology meeting, and my PhD student also presented preliminary findings at the Japan-Partnership Awards meeting in Kitakyushu, Japan. The techniques involved are a significant step forward in molecular biology, with the sight of* in vivo *transduction and live imaging of neurones. The study of vesicle pool segregation in magnocellular dendrites will also contribute to the understanding of the mechanism of vesicle release in peptide-secreting neurones, which is a major interest in our centre.*

MIKE LUDWIG

The Society for Endocrinology funded a **Lab Visit** to Dr Marian Ludgate's laboratory at the University Hospital of Wales in Cardiff. My aim was to learn the culture, transfection and growth in 3D culture of FRTL-5 cells. These cells have strict culture conditions, are slow-growing and take longer to adhere than many commonly used cell lines. Since my visit, I have managed to gain transfection efficiencies suitable for stable cell line selection. The technical skills for the 3D culture would have been difficult to grasp without Dr Ludgate's experience. I also enjoyed interacting with other endocrinologists, which is not usually possible as I am based at the Institute of Human Genetics in Newcastle. I had the chance to give short talks on my research in the weekly lab meetings, which gave me useful feedback. Neither 3D culture or its imaging has been attempted within my Institute; this lab visit has allowed me to bring back, develop and pass on these skills. The techniques will allow me to further my study of autoimmune thyroid disease and the role of autoantigen localisation in the autoimmune process.

DAVID MCDONALD

The **Small Grant** *enabled me to finish a larger project that had run out of funding, and would have otherwise been impossible. The crucial outstanding experiments helped us to understand a novel aspect of our research into secretory vesicle trafficking, and to complete a manuscript. Publication of these results will enable us to attract more funding. There are also benefits for the host institution, the Centre for Integrative Physiology, as the grant furthered networking between different disciplines within the Centre. My colleague, Dr Ulrich Wiegand, who was also working on this project, has now joined the Society for Endocrinology!*

GARETH LENG

The Society's generous support meant that, for the first time, we could hold a **Sponsored Poster Session** during the Welsh Endocrine and Diabetes Society (WEDS) meeting last June. WEDS seeks to foster improved links in education across endocrine and diabetes units within the principality. The session attracted 32 high quality abstracts from undergraduate medical students, research scientists and junior doctors through to SpRs based at all of the regional hospitals within Wales. We achieved our aim of fostering enthusiasm for endocrinology amongst these groups, and it was pleasing to award three prizes for the best quality submissions. The session lasted for one and a half hours and had more than 100 attendees, who universally agreed it was an enormous success. The WEDS Executive Committee have agreed that the venture should be repeated annually.

ALED REES

THE NEW FACE OF BIOSCIENCE

In this abridged version of the full prospectus, Richard Dyer examines the creation of a new organisation by the Institute of Biology (IOB) and the Biosciences Federation (BSF).

The excitement of modern biology is palpable to all. Uniquely important issues are frequently discussed by the media and the public. How many in this country are not aware of the debates about stem cells, loss of species through global warming and modern agriculture, the teaching of biology in schools or what our diet is doing to our bodies?

But how do the biologists join these debates about biology? If they do at all, it is through a myriad of possible routes and representing too many organisations. It is the exciting diversity of the biosciences that has led to many special interest groups where individuals with shared interests productively work together. There are probably more than 200 independent organisations. This landscape is unique to the biosciences: physics, chemistry, mathematics and engineering are represented by very few organisations, which are wealthy and influential.

Whilst special interest groups undoubtedly bring advantages of focus to research or fundraising, the fragmentation can lead to huge disadvantage, for example, in outreach to schools or in representing biology to governments and funders. The need for a unified voice has never been greater.

So the councils of the IoB and the BSF proposed that a New Organisation (NO) should be created to embrace the activities and strengths of both BSF and IoB, and add new activities that will benefit UK biosciences and provide greater value to the membership. This proposal was unanimously endorsed by a joint meeting of the IoB and BSF at the Royal Society in May 2008.

To move this ambitious plan forward, the BSF and IoB have established a joint implementation group (IG) chaired by Prof Sir Brian Heap. Other members of the IG are Dame Bridget Ogilvie, Prof Dame Nancy Rothwell, Prof Malcolm Press, Prof Keith Gull, Prof David Coates, Mr Alan Johnston, Dr Alan Malcolm and Dr Richard Dyer. Members of the IG are unanimously agreed on the following key issues.

1. Membership of the NO will be open to both individuals and organisations from any sector of the biological sciences.
2. The council of the NO shall comprise a chair and 12 members, 4 of whom will be elected by the individual membership, another 4 from the institutional membership and the final 4 nominated by council to ensure that there is a good balance of representation and that the council is fit for the purposes expected of a charity in the 21st century.
3. The NO shall have a royal charter and continue to offer chartered status and fellowship to individual members.

Currently the IG is preparing the papers that will need to be put to the memberships of the BSF and IoB for necessary approval for these proposals to be implemented.

The NO can be launched successfully with the present combined incomes of the BSF and IoB. In 2007, the corporate subscription income for BSF was £235k and the membership income for IoB was £860k. For 2009 the total subscription income for the two organisations will be about £1.2m. In addition, the IoB raises about £200k from other sources and the BSF about £100k per annum from member organisations for identified projects. This 'à la carte' funding is an important element for future development.

A full business plan for the NO has been produced. The IG is not focused on the detail of the structures within the NO. That will be a responsibility of the first council and new chief executive. However, the IG is determined to suggest some overarching principles for its organisation: in particular, that it has a flat structure with the ability to make rapid decisions.

The immediate goals for the NO will focus on membership, outreach and policy work.

There will be an immediate drive to increase the number of individual and institutional members. Currently some large areas of the biosciences are poorly represented in both the BSF and IoB. The NO will retain a royal charter and offer chartered status to its individual members. An immediate goal will be to strengthen the standing of this qualification by introducing a structured career and professional development programme. The aim will be to increase the esteem of all qualifications, including fellowship.

Structured outreach to schools and to the public will be built through the regional groups that already exist in the IoB. There is considerable scope for exciting new ventures. The NO will also focus on increasing outreach to the membership as a whole through regional scientific meetings and high quality lectures and debates. The leadership, both executive and non-executive, will ensure that the activities of NO are not solely based in London or south east England. Finally, there is the prospect of substantially increasing the effectiveness of outreach through the media, both directly and by partnering with the Science Media Centre.

Both the BSF and IoB have been largely reactive to policy initiatives from governments and funders. It is equally important to be proactive to set the national agenda (national meaning all four countries of the union).

Both the IoB and BSF could undertake many of the activities that are proposed for NO, but they would not engage all the heartlands of the biosciences. To meet the challenges of today, biology needs a single voice. Our subject needs an organisation like the Royal Society of Chemistry or the Institute of Physics which has the respect of the community as a whole, and where individuals are proud to be members because of the standards maintained and the quality of project delivery. With your support, the NO can achieve this status rapidly: this is urgently needed and the current opportunity must be embraced.

RICHARD DYER

The full version of the prospectus, containing more information on the finance, structures and immediate goals of the new organisation, is available at www.bsf.ac.uk.

COMPARATIVE ENDOCRINOLOGY CE

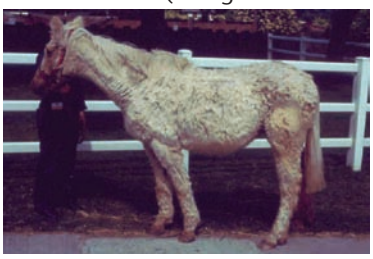
What every endocrinologist

Interesting tales from the world of comparative endocrinology.

Horses can get Cushing's

▶ Cushing's is found in humans and dogs, but it is also the most common endocrine disorder to affect middle-aged and geriatric horses. It is found so often that some believe it is part of the natural ageing process.

Equine Cushing's syndrome is generally caused by hypertrophy, hyperplasia or adenoma formation in the pituitary gland, although it can also be caused by adrenal tumours. Horses tend to develop pituitary adenomas that originate from the pars intermedia, whereas in humans the adenoma can also originate from the pars distalis. In horses, the most obvious clinical finding is hirsuitism (a long coat that fails to shed), but other clinical signs may include polydipsia, polyuria, hyperglycaemia, muscle wastage and laminitis (failure of the bond between the hoof wall and the bone in the foot). The symptoms are controlled through changes in management and/or drug therapy (most commonly with pergolide.



Metabolic syndrome can affect horses as well as humans. Human metabolic syndrome is characterised by obesity, insulin resistance, hypertension and dyslipidaemia. Peripheral Cushing's syndrome or equine metabolic syndrome is characterised by the combination of obesity, insulin resistance and laminitis in mature horses. The effectiveness of insulin signalling at insulin-sensitive target cells is often found to be impaired in native pony breeds, particularly in obese animals, and insulin resistance is thought to be a risk factor for laminitis. It has also been suggested that chronic insulin resistance can predispose an animal to Cushing's syndrome. There is an increasing body of evidence that suggests that certain animals may have a genetic and phenotypic predisposition to the development of equine metabolic syndrome.

LIANE CROWTHER, THE HORSE TRUST

GH boosts commercial milk production

▶ Growth hormone (GH), also known as bovine somatotropin (BST), is used commercially in the USA and elsewhere to increase the milk yield of dairy cows.

The increase is about 10-20%. GH is a homeostatic re-partitioning agent, which means it re-directs nutrients away from body tissues (adipose tissue and muscle) and towards the mammary gland, where they are synthesised into milk. It works exquisitely to increase the lifespan and synthetic capacity of the milk secretory cells, and the blood flow through the mammary gland, and to reduce the rate of uptake of nutrients at other tissues. The yield-enhancing effects of GH occur within a matter of days. Over a period of weeks the appetite of the dairy cow is also increased; in the meantime the energy balance of the cow is reduced such that the additional milk comes from body reserves. GH is administered commercially once every 2 weeks as a slow-release subcutaneous injection.

These effects were first identified before World War Two. Extracting GH from the pituitary glands of culled cattle was considered as a way of increasing the UK's milk supply during the war. But the amount that could be produced in that way would have had a negligible effect on the milk supply of the country. It was the advent of recombinant DNA technology in the 1980s that led to a method of producing copious amounts of GH and enabled its commercialisation during the 1990s.

Use of GH in this way is highly controversial. Its use in the EU and elsewhere is prohibited because of possible (though unlikely) adverse health effects on human consumers of milk, and because of the real adverse health effects it has on the cows. Meta-studies of BST use have shown increased rates of mastitis and lameness in dairy cows, as well as an incidence of infections at the injection site. Even in the USA there are now increased calls for this synthetic hormone to be banned.

MICHAEL ROSE, ABERYSTWYTH UNIVERSITY

Spawning salmon may die from 'Cushing's'

▶ With Jamie Oliver on the food revolutionary path again, this time in Rotherham, you may have seen him cajoling novices into creating healthy food in front of a large audience in the town square. Pan-fried salmon was on the menu.

There is little debate that limited intake of salmon and other fish is good for you, as part of a balanced diet. But you may not be so familiar with data from 50 years ago, demonstrating the endocrine mayhem and ill health that the Pacific salmon appears to suffer during migration and spawning.

This amazing fish, the picture of health at sea, migrates hundreds of miles to spawning grounds, only then to die. Post-mortems of spawning fish show very advanced coronary artery disease, and vacuolation of striated muscle. The change in physical appearance from sea to spawning ground is striking, with the appearance of an almost 'buffalo hump' (excuse the cross-species analogy), whilst internally the intra-renal gland increases dramatically. It might not then be such a surprise to find very elevated cortisol levels in the spawning fish. Is the cause of demise Cushing's syndrome? The clinical, anatomical, histological and biochemical data are rather compelling!

JOHN NEWELL-PRICE, UNIVERSITY OF SHEFFIELD

should know...

Hyperthyroidism is the most common endocrinopathy in cats

Feline hyperthyroidism is both clinically and histopathologically very similar to toxic nodular goitre (HTNG) in humans. While in humans HTNG is more prevalent in females, the condition affects male and female cats equally. It results in debilitating disease in a significant percentage of middle-aged and older cats.

In both cats and humans, hyperthyroidism is caused by TSH-independent overactivity of one or more benign hyperfunctioning adenomatous thyroid nodules. This leads to high circulating concentrations of thyroxine and tri-iodothyronine, which cause multisystemic clinical signs including weight loss, increased appetite, tachycardia and polyphagia.

Most HTNG patients exhibit a gain-of-function TSH receptor gene mutation. Many of the receptor gene mutations are directly comparable between feline hyperthyroidism and HTNG. The most common somatic mutation detected in cats (a Met-452>Thr mutation) is analogous to the human Met-453>Thr observed in sporadic human hyperthyroidism.

ANDREW LOWE, FROM WATSON ET AL. *2005,* JOURNAL OF ENDOCRINOLOGY ***186****, 523-537*

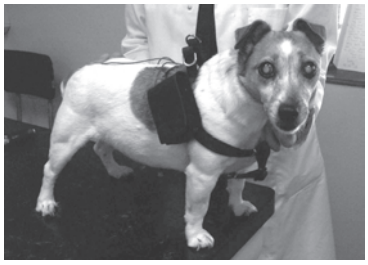
Dogs get diabetes too

It often surprises our medical colleagues to learn that veterinary surgeons diagnose and treat diabetes in companion animals in much the same way as they do in human patients. Comparative research into diabetes in dogs might offer opportunities that are not possible in rodent models.

Canine diabetes is diagnosed on the basis of clinical signs of polyuria and polydipsia, persistent hyperglycaemia and glucosuria. Virtually all diabetic dogs are insulin-deficient and are dependent upon insulin therapy. It is difficult to use the classification system for human diabetes in dogs, since the underlying cause of the beta cell loss or dysfunction is not usually investigated. However, it is clear that canine diabetes is not a single disease entity and several types of the disease occur.

Neonatal diabetes is seen in particular breeds (primarily labradors in the UK) but is rare and seems to be due to congenital beta cell aplasia. Most diabetic dogs are diagnosed in middle age (between 5 and 12 years old). Although there is no sex predisposition, female dogs can develop diabetes during dioestrus, which is comparable with human gestational diabetes.

There are clear breed differences in susceptibility to diabetes with samoyeds and tibetan and cairn terriers at an increased risk, whereas golden retrievers, german shepherd dogs and boxers are relatively resistant. This suggests that there is a genetic component to diabetes susceptibility in dogs, and recent work has implicated MHC and some other immune response genes.



A diabetic Jack Russell Terrier showing diabetic cataracts and wearing a Minimed Continuous Monitoring System

There is little evidence that obesity is a major risk factor for diabetes in dogs, which is in contrast to the situation in cats. Thus, canine type 2 diabetes does not seem to exist. Since most dogs suffer from insulin deficiency, it has been suggested that the disease is most similar to the type 1 form of the disease. Although there is evidence for circulating beta cell autoantibodies (primarily against GAD65) in a proportion of diabetic dogs, most are autoantibody negative. Furthermore, the age of onset suggests that if the beta cell loss is immune-mediated, this process might be more comparable with that seen in latent autoimmune diabetes of the adult (LADA) rather than juvenile-onset type 1A diabetes. Chronic subclinical pancreatitis is also believed to contribute to beta cell loss or dysfunction in some cases.

Much remains to be investigated in terms of the genetic and environmental factors that contribute to canine diabetes susceptibility and the mechanisms that lead to beta cell dysfunction. However, veterinarians aim to contribute to the research effort into this disease alongside basic science and medical colleagues.

BRIAN CATCHPOLE, ROYAL VETERINARY COLLEGE, UNIVERSITY OF LONDON

Birds may show developmental responses to stress

The long term effects of developmental stress have been studied in mammalian models for many years, to understand not only the underlying mechanisms, but also to determine the consequences for human health.

These studies have shown that exposure to glucocorticoid stress hormones during development can permanently alter the reactivity of the HPA axis. Treatment can also have significant effects on adult behaviour, cognitive ability and important indicators of diseases such as cardiovascular disease and diabetes. However, the continued physiological link between mother and offspring during development constrains the ability to determine the direct effects of stressors on subsequent physiology and behaviour.

Researchers at the University of Glasgow are now using birds to understand the role of glucocorticoid programming in shaping adult phenotypes. Here, there is only a brief window of opportunity for a mother to invest glucocorticoid hormones into each egg, and no direct maternal input of hormones during postnatal development. This therefore allows precise quantification of exposure levels and the scope for controlled experimental manipulation of glucocorticoid levels at several developmental stages.

Although currently in the early stages, this model could provide an important tool in understanding the basic mechanisms underlying the long term effects of developmental stress in humans.

KAREN SPENCER, UNIVERSITY OF GLASGOW

COMPARATIVE ENDOCRINOLOGY CE

Feline hyperthyroidism

With perhaps 10% of the ageing cat population developing this endocrine disease, Hattie Syme tells us more.

▶ Which endocrinopathy would you consider most likely if a patient presented to you with weight loss, behaviour change, tachycardia and goitre? Would it change if the patient wasn't human, but an elderly cat like the one in Fig.1?

Well, by far the most likely diagnosis would be hyperthyroidism. Other endocrine causes of weight loss do occur in cats, notably diabetes mellitus, but hyperthyroidism is the most common endocrinopathy in the ageing cat. In fact, the prevalence of feline hyperthyroidism is incredibly high. In our clinics in central London, about 10% of cats older than 9 years have hyperthyroidism, and the prevalence increases further with advancing age. This differs markedly from the situation in dogs, where hyperthyroidism is extremely uncommon, only occurring occasionally in association with functional thyroid carcinoma. Dogs do, however, get hypo-thyroidism, which cats (in general) do not.

Figure 1. *Hyperthyroid cat*
(PICTURE COURTESY OF DR SCOTT-MONCRIEFF, PURDUE UNIVERSITY)

The recognition of hyperthyroidism is often not difficult. The classical signs are weight loss in association with a good appetite, although 'apathetic' forms of the disease occur where the appetite may be reduced. Gastrointestinal signs like vomiting and diarrhoea are relatively common, and the patient may develop an unkempt or greasy coat. Tachycardia and goitre are the most consistent findings on physical examination. Cats have two thyroid lobes that normally lie either side of the trachea, midway down the neck. Goitre may, therefore, be palpable unilaterally or bilaterally, and is usually about the size of a small pea (although larger masses do occur).

Increasingly, hyperthyroidism is diagnosed early during its clinical course and signs are not as marked as they were when the condition was first described. The diagnosis is confirmed by elevated total thyroxine. Although feline TSH has been cloned and sequenced, no assay for TSH has proved to be sufficiently sensitive for the diagnosis of hyperthyroidism.

The pathological changes that develop in feline hyperthyroidism are similar to those of toxic adenomatous goitre in humans. Cats do not develop antibodies to the TSH receptor as occurs in Graves' disease.

There is much speculation regarding the cause of hyperthyroidism in cats. Although this condition was first recognised in the late 1970s, the prevalence of hyperthyroidism has increased to epidemic proportions today. This has been variously attributed to an increase in feline longevity, increased willingness of owners to seek treatment for their pets, improved diagnosis by veterinary surgeons and a true increase in disease prevalence.

It seems likely that all are true to some extent. Epidemiological studies have found associations of hyperthyroidism with increasing age, a protective effect in certain pure breeds (notably the Siamese) and associations with certain lifestyle factors, most notably consumption of canned cat food.

Theories abound as to why canned food should increase the risk of developing hyperthyroidism. It could be a marker for cats that are most likely to enjoy a protected indoor existence and so most likely to live to an advanced age. However, its significance in multivariable models that include age as a risk factor make this less likely to be the sole explanation. The iodine content of cat food is extremely variable, and often up to ten times that recommended. It has been suggested that this could contribute to the development of thyroid disease. Alternatively, one study found that the association with hyperthyroidism was stronger when the cats were fed from 'pop-top' cans rather than the traditional type, leading to the hypothesis that the plasticiser used might be responsible.

The polyphenolic compound bisphenol A, a weakly oestrogenic endocrine disruptor, is used as a plasticiser in can linings and has been detected in canned food. The cat may be particularly susceptible to any 'endocrine-disrupting' effects of bisphenol A since it is eliminated in other species by glucuronidation, a metabolic pathway that is relatively deficient in the cat. In a similar vein, a recent publication found that feline serum samples contained high levels of polybrominated diphenyl ethers (PBDEs) which, it was postulated, could be associated with the increasing prevalence of hyperthyroidism (although no direct association was actually shown). The authors speculated that, in addition to dietary sources of PBDEs, cats may ingest these compounds through grooming, as high concentrations are found in dust.

Treatment of hyperthyroidism in cats is not dissimilar to treatment of humans, although humans may be better at taking tablets. Broadly speaking, there are three options: medical management with methimazole or carbimazole, surgical thyroidectomy or treatment with radioactive iodine. Each has advantages and disadvantages.

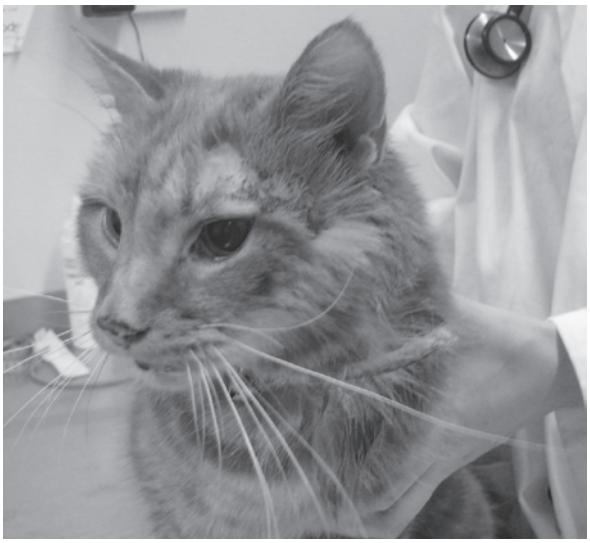


Figure 2. *Typical cutaneous reaction to methimazole/carbimazole*

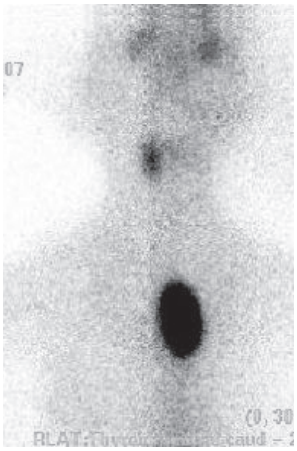


Figure 3. *Technetium scan demonstrating intrathoracic thyroid tissue*

Medical treatment is relatively cheap but requires continued monitoring at significant cost. If the cat is fairly young and healthy then the total cost of medical management may be greater than that of a definitive therapy. Side-effects are relatively common, but generally these are mild (gastrointestinal upset) and may diminish with time, though more serious side-effects can include cutaneous adverse drug reactions (Fig. 2) and occasionally blood dyscrasias, which may be life-threatening if they are not recognised and treated promptly.

Surgical thyroidectomy is associated with a high risk of recurrence if only a unilateral thyroidectomy is performed, because the disease is bilateral in at least 70% of cases, although it is often asymmetrical with one gland being notably larger than the other. A technetium scan before surgery would aid in treatment planning (Fig. 3), since if the hyperfunctional tissue is intrathoracic then non-surgical treatment options are preferred. However, few practices have access to a gamma camera so this is usually not possible. Hypoparathyroidism is a potential complication of bilateral thyroidectomy. Radioactive iodine therapy is offered by a few referral institutions, but the relatively high cost and the long isolation period for cats after treatment (2-4 weeks) mean that it is chosen by relatively few owners, although it is very effective.

One problem encountered when treating cats for hyperthyroidism is the unmasking of chronic kidney disease (CKD). Feline CKD is very common; it is second only to neoplasia as a cause of death in this species. Hyperthyroidism increases glomerular filtration rate (GFR) so CKD is usually not detected before treatment, but up to half of all cats develop azotaemia as they become euthyroid. In spite of this, the cats' condition tends to improve and they gain weight with treatment, probably because the azotaemia is often mild.

The survival time of cats that develop azotaemia does not appear to be noticeably shorter than that of those that do not, although cats that are azotaemic before treatment starts do not fare so well. Similar changes in GFR in the hyperthyroid state have been reported in humans, but because the prevalence of renal disease is much lower it has received much less attention.

In summary, hyperthyroidism is a very common endocrinopathy in the cat, with close parallels to toxic nodular goitre in humans. Diagnosis and treatment of the condition are relatively straightforward. What is not known is why the condition develops. Various nutritional and environmental risk factors have been suggested and it has been proposed that the cat is acting as 'a canary in the mine-shaft', alerting us to the potential danger to humans from exposure to environmental pollutants.

HATTIE SYMES, ROYAL VETERINARY COLLEGE

COMPARATIVE ENDOCRINOLOGY CE

A case of canine hyperadrenocorticism

Methods and results

George is a 10-year-old, entire male Yorkshire terrier. He was presented to his veterinary surgeon with the complaint that he was eating and drinking excessively, putting on weight and losing his hair.

The dog was panting excessively and seemed to have difficulty rising. On physical examination, the veterinary surgeon found the dog to have an enlarged and rounded abdomen, palpable hepatomegaly with bilateral alopecia evident on the flanks (Fig. 1). On the ventral abdomen, the veins were very prominent and the skin seemed thin and was hyperpigmented, with many comedones evident.

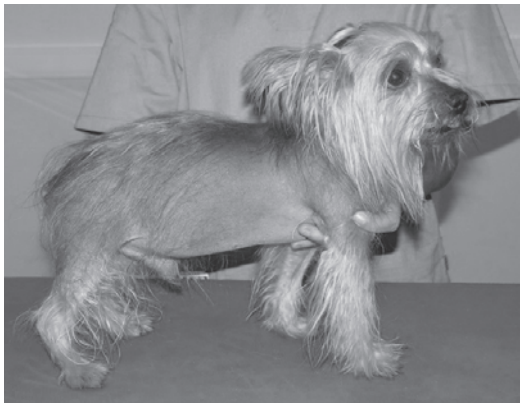


Figure 1. *George at presentation*
(FIGURES PROVIDED COURTESY OF DECHRA VETERINARY PRODUCTS)

Routine haematology identified the following abnormalities: mature neutrophilia, monocytosis, lymphopaenia, eosinopaenia, and thrombocytosis. Clinical chemistry identified elevated levels of liver enzymes alkaline phosphatase and alanine aminotransferase, hypercholesterolaemia, and hyperlipidaemia. Urinalysis found that the urine was very dilute (SG 1006), but no other abnormalities were found.

These findings were very suggestive of canine hyperadrenocorticism (HAC). The clinical and laboratory findings made other causes of excessive drinking and weight gain, such as diabetes mellitus, less likely.

An ACTH stimulation test was used to screen for HAC. Blood samples were assayed for cortisol levels before and 1 hour after the intravenous injection of synthetic ACTH, with the following results: pre-ACTH cortisol: 176 nmol/l; post-ACTH cortisol: 968 nmol/l. A normal response is a rise to up to 450 nmol/l. This is an exaggerated response, suggestive of pituitary-dependent HAC.

A high-dose dexamethasone suppression test was carried out to try to differentiate between pituitary- and adrenal-dependent disease. Blood samples were assayed for cortisol levels before, 3 hours after and 8 hours after intravenous injection of 0.1 mg dexamethasone/kg, with the following results: pre-dexamethasone cortisol: 164 nmol/l; 3-hour post-dexamethasone cortisol: 38 nmol/l; 8-hour post-dexamethasone cortisol: 59 nmol/l.

Persistent suppression of cortisol levels after the injection of a high dose of dexamethasone is again highly suggestive of pituitary-dependent HAC. Failure to suppress is more suggestive of adrenal-dependent HAC, although a proportion of dogs with pituitary-dependent HAC fail to suppress with this test.

CONTINUED ON PAGE 12

A case of canine hyperadrenocorticism *continued from page 11*

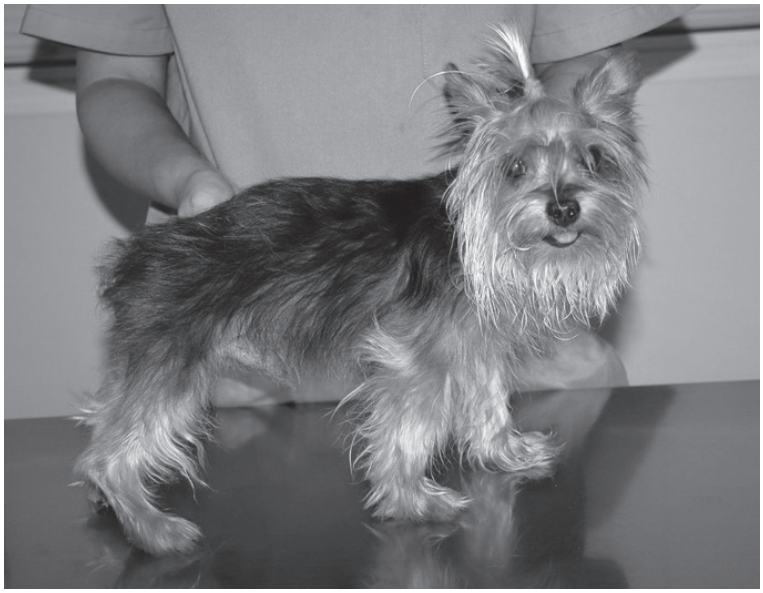


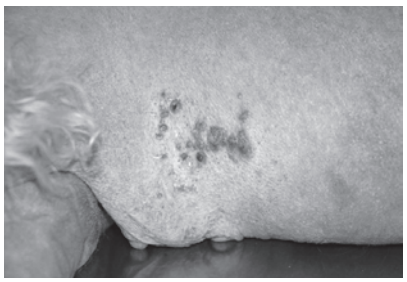
Figure 2. *George after several months of therapy*

A diagnosis of pituitary-dependent HAC was made and treatment with trilostane initiated. Within 6 weeks the polydipsia and polyphagia had largely resolved. The dog was more active and panting much less. Some months later the hair coat had also regrown (Fig. 2).

Discussion

Approximately 85% of naturally occurring cases of canine HAC are pituitary-dependent (usually due to a functional microadenoma in the pars distalis) and 15% adrenal-dependent. However, a significant number of iatrogenic cases occur each year as a consequence of using systemic and topical glucocorticoid therapy, particularly for treatment of pruritic skin and ear disease.

Figure 3. *Calcinosis cutis in another dog with HAC*



The presenting signs are characteristic and would be very suggestive of a diagnosis of canine HAC, although affected dogs may show very few typical clinical signs. Other presenting signs might include: bruising and poor tissue healing; muscle-wasting; calcinosis cutis (Fig. 3); recurrent pyoderma, urinary tract or other infections; respiratory distress as a consequence of pulmonary thromboembolism. Less commonly one might see: systemic hypertension (usually manifesting as sudden-onset blindness); calcium oxalate urolithiasis; refractory diabetes mellitus; mental dullness, pacing, other CNS signs with some pituitary macroadenomas.

Imaging techniques are also often used in the diagnosis of canine HAC. Abdominal radiography may identify an adrenal mass, particularly if it is calcified. Abdominal ultrasonography may identify a unilateral adrenal mass in patients with adrenal adenomata, or bilateral adrenal enlargement in patients with pituitary-dependent HAC. Computed tomography or magnetic resonance imaging may identify a pituitary macroadenoma.

As well as the ACTH stimulation test, other screening tests which cannot differentiate between pituitary- and adrenal-dependent disease include: low-dose dexamethasone stimulation tests (a screening test often used instead of ACTH stimulation test to confirm the presence of HAC); urine corticoid:creatinine ratio; 17-hydroxyprogesterone levels measured pre- and post-ACTH.

Given the unreliability of the high-dose dexamethasone suppression test, new tests which can discriminate between pituitary- and adrenal-dependent disease are being assessed and the measurement of endogenous ACTH levels is now considered to be the most useful test for this purpose.

Alternative treatment options include: surgical adrenalectomy or hypophysectomy as appropriate (technically difficult and rarely performed); Lysodren (o,p'-DDD; results in selective necrosis of cortisol-producing cells in the adrenal cortex); ketoconazole (inhibits adrenal enzymes responsible for synthesis of cortisol); radiation therapy for pituitary macroadenomata.

Most affected dogs live approximately 2 years, although some may live 5 years post-diagnosis.

MALCOLM COBB, KATE GRIFFITHS, UNIVERSITY OF NOTTINGHAM

Voice of Young Science

The Voice of Young Science (VoYS) media workshop took place on 6 June at the Institute of Biology in London with sponsorship from the Society for Endocrinology. It was open to early career researchers like myself, hosted by Sense About Science, a registered charity aiming to promote good science and evidence for the public.

The day began with a discussion on the changing image of science in the media, and the problems that can arise when research leaves your hands and enters the public domain. After lunch, a panel of journalists discussed how they handle research stories and how to avoid problems that lead to bad reporting. We then had a presentation about the media training and support available to researchers, and what we can do to promote good science. Speakers included Dr Azra Ghani, Reader in Infectious Disease Modelling at the London School of Hygiene and Tropical Medicine, Mike Swain from the *Daily Mirror* and Lewis Smith from *The Times*.

I must admit to having a rather negative view of journalists before attending the workshop, but that was about to change. What surprised me and several of the other students was that, despite some bad press (including a nasty grilling on Newsnight), the scientists on the panel were overwhelmingly positive about the media. They highlighted the change over the last decade to a more positive view of science, describing it as the 'golden age of science reporting'.

The panellists were quick to point out that bad reporting is simply not in a journalists interests and that they have no say in what headline is attached to their story. Many of the problems that appear to arise are due to miscommunication between the journalist and researcher, especially when they are trying to condense a discovery into as little as 50-100 words. As such, the panellists stressed the importance of a clear and concise press release.

I would encourage anyone embarking on a career in research to attend one of these workshops, which are run throughout the year. It was invaluable to learn of the existence of the Science Media Centre and Sense About Science, to whom you can turn for advice and training in all aspects of media relations.

I would also urge early career researchers to join the VoYS network, which encourages its members to become involved in public scientific debate. The network also works to challenge bad or misleading uses of science, as in their well publicised booklet 'There goes the science bit…'. In the booklet, members challenged companies to justify the supposed science behind their product claims, including holistic CDs to cancel out computer radiation and patches to suck out impurities through your feet!

For more information or to request a copy of the booklet visit www.senseaboutscience.org.uk, or to find out about the Science Media Centre see www.sciencemediacentre.org.

VERONICA LINDOP

Specialty Certificate Examinations (SCE) in Diabetes and Endocrinology

20 MAY 2009

The Federation of Royal Colleges of Physicians of the UK, in association with the Society for Endocrinology, Association of British Clinical Diabetologists and Diabetes UK, have developed a programme to deliver SCEs in Diabetes and Endocrinology (formerly known as Knowledge Based Assessments). A specialty SCE is now a compulsory component of assessment for Certificate of Completion of Training (CCT) for all UK trainees whose specialist training began in or after August 2007.

The first diet will take place 20 May 2009 - see www.mrcpuk.org/KBA/pages/home.aspx for further information.

FAMOUS LIVES:
Andrew Dasburg

The remarkable story of a born survivor of Addison's disease.

A betting man might have hesitated to place cash on the chances of the young Andrew Dasburg surviving to adulthood, let alone becoming a nonagenarian and an acclaimed artist, who outlived three spouses and outwitted two deadly diseases. Poverty, joint pain, ill-health and despair were to dog him for most of his life. Art was the compulsive passion that kept him going.

Dasburg was born in Paris in 1887. When he was 5, his seamstress mother emigrated to the USA with her illegitimate only son. They lived with Andrew's aunt in Hell's Kitchen, a crowded New York tenement district. Somewhere along the way, the boy acquired tuberculosis which attacked his hip joint. As an adult, he would need two canes to walk. As a child, he wore leg braces and was sent to a New York school for crippled children. The teachers there identified a talent for sketching and he went on to attend the Art Students' League and the New York School of Art on scholarship places.

A summer scholarship to Woodstock Art School introduced him to his first wife, sculptor Grace Johnson, who accompanied him to Europe. The marriage produced his only son, Alfred, but ended some 13 years later. Dasburg was by then spending much of his time in New Mexico. He settled there permanently in 1930 with his second wife, Nancy Lane, after he lost his savings in the 1929 crash. In 1933 he married again, to the poet Marina Wister. He was a leading modernist artist, described as charming, opinionated, ambitious and a ferocious worker.

The Great Depression had arrived, and Dasburg was about to become very sick. He was 49 when his Addison's disease was diagnosed in early 1937. The nearest hospital, in Albuquerque, was not aware that treatment was possible and sent him home to die. But a handful of leading North American hospitals were keeping their adrenal patients alive on a mix of ground-up beef glands and teaspoons of salt. Marina's family connections in Philadelphia gained him access to a new, experimental treatment: intravenous and granulated animal adrenal cortex extracts, pioneered by Dr WW Swingle at Princeton, and used by Dr Leonard Rowntree at the Mayo Clinic for his private patients.

At considerable expense, Dasburg was to give himself daily injections of Swingle's aqueous extract for 25 years. He would reluctantly switch to synthetic cortisone tablets in 1962, when Dr Swingle retired. But he plunged into depression even before he experienced his first adrenal crisis in 1939, after catching flu. He did not paint again until the end of World War Two.

Despite a succession of health problems and recurrent hospitalisations, Dasburg then painted and drew intensively for over 30 years, producing some of his finest work. He lived frugally in a remote part of New Mexico, with medical bills often eating up most of his meagre income. For some years he sustained a romantic liaison with a young art student who was 40 years his junior, during his wife's extended absence in Philadelphia. It was largely due to Marina's determined advocacy that he had survived. Sadly, she was less successful in dealing with her own alcohol dependency and spent her final years institutionalised by her family.

In 1953, the Royal Society of Medicine held a symposium on the diagnosis and treatment of Addison's disease. Introducing the proceedings, Professor DM Dunlop reported that of the 30 Addison's patients he had treated before 1938, only two survived long enough to be treated with the deoxycortone acetate pellets which became available in 1939. He had a further 30 patients during 1939-1951, of whom fewer than half survived to see the availability of cortisone tablets in 1952. As at 1953, the longest time that one of his patients had survived post-diagnosis was 13 years. Most of his patients had died during an adrenal crisis; he commented that better understanding of the use of intravenous cortical extract and intravenous fluids during crisis, and more careful and regular supervision of the patients, contributed to improved results during the 1940s.

Following him, Dr Leonard Simpson observed that he had treated 20 Addison's patients with implanted deoxycortone pellets from 1939-1951. The longest any had survived was 10 years, with hypoglycaemic crisis the main cause of death. He commented that the 1952 advent of readily available synthetic cortisone, which could be injected in extremis, had minimised the dangers and terrors of Addisonian crisis.

By 1953, Dasburg had already survived several adrenal crises and had outlived any treated UK patient by a good 3 years, thanks to his access to Dr Swingle's injected aqueous cortex extract. In fact, Swingle's extract was well known to UK endocrinologists and had been trialled by Dr Simpson at the London Hospital in the early 1930s. Its use was reserved for intravenous response to adrenal crises as the price was judged prohibitive for maintenance therapy. In today's money, the annual cost would have been about £20 000.

Dasburg was to live on for a further 26 years before dying of old age. He passed his driving test at the age of 81 and took up lithographs at the age of 88. The afflictions of old age - a stroke, arthritis, shoulder bursitis, cataract surgery - constrained his ability to produce large-scale canvasses but did not deflect him from a disciplined daily work schedule. In 1979, at the age of 92, he died peacefully at his home in Talpa, New Mexico.

KATHERINE G WHITE

The Addison's Disease Self-Help Group (www.addisons.org.uk) would be interested to hear of any outstanding achievements among the Addison's patients of UK physicians - be it longevity, artistic, academic or sporting accomplishments. Please contact kgwhite@addisons.org.uk.

Failures in the human endocrine communication system

Sometimes the medium is the message, as Hotspur relates.

A medical lecturer's responsibility to the audience has not changed over the years: educate, stimulate, inform and breathe life into the subject matter. The most gifted teachers are those with the ability to communicate in abundance.

However, the medium through which they transmit information has changed over the last 30-40 years. From the chalk and blackboard, we moved to the overhead projector and transparencies, and subsequently the slide projector with slides. Throughout most of my working life, slides and a slide projector were the tools of the trade but, of course, now they are obsolete, replaced by Powerpoint presentations.

Any of these processes can go wrong. The overhead projector was vulnerable to lightbulb failure, with the fear that no spare bulb would be located. The slide projector, however, lent itself to a significantly greater range of problems.

I had the misfortune to give a lecture when the slide projector was set to automatic, with a predetermined, fixed interval between slides. I had to adjust my comments on each slide to fit into a fixed time, irrespective of slide content or interest. The audience only became aware of my predicament when I appeared to develop acute-onset asthma.

On another occasion I was talking at one of several parallel workshops taking place in adjoining rooms. Much to my horror, the audience were laughing at my presentation by slide number 3. This threw me, as such a response to my data did not usually occur until slide 6. The laughter was precipitated because my slides were 'moving on' haphazardly, even though I was not touching the remote control. It transpired that the remote control of the speaker in the next room was 'working' my slide projector.

The number of slides to be shown for optimal communication is also critical, and there have been times when I have been anxious about indulging in information overload. This anxiety manifests itself bizarrely when you end up discussing the issue with someone who probably has no idea what the word 'endocrinology' even means. I am referring to the slide projectionist.

On one such occasion, in a state of panic, I reached the projection room and muttered to the projectionist, 'I'm on after the next speaker. I'm concerned that I have too many slides. I have 29 slides for a 20-minute talk.'

Despite his lack of endocrine qualifications, the projectionist's reply calmed me immediately. 'I shouldn't worry, sir; the next speaker (who happened to be an extremely senior eminent member of the Society for Endocrinology) is showing 80 slides in 20 minutes.'

But the most unsettling experience occurred at an overseas national endocrine conference. I had placed my unnumbered slides in the slide tray myself. During the chairman's introduction, when I was already standing at the podium, I noticed - to my horror - that the projectionist was lifting individual slides out of the tray, holding them up to the light for cursory examination and replacing them, apparently at random. I sensed disaster; why was he behaving like this? Couldn't he curtail his enthusiasm a little longer for the pearls of wisdom about to be bestowed?

Well, the lecture started and I asked for the first slide: number 22 appeared. I asked for the second slide and number 38 appeared. The only possible action was to halt the talk. The chairman ordered an early coffee break and I repositioned my slides. It transpired that the projectionist had spilled the slides, and replaced them in an order he felt to be appropriate. I resisted the temptation of suggesting that if he knew the order, then why didn't he give the lecture himself? In reality no harm was done, and the early coffee break proved very popular with the audience...

In the modern computer era, less tends to go wrong with the process. There are, however, variations on the theme, which involve terminology new to me. Thus a few months ago I was delighted - but a little apprehensive - to be invited by a pharmaceutical company to give a webcam lecture at a satellite symposium attached to an international meeting in Europe. It was for a group of endocrinologists from a country thousands of miles away, who were unable to attend the meeting. I was unsure about the exact format of a webcam performance, and assumed the pharmaceutical company would provide guidance.

The initial request asked for the subject matter of my talk. I proposed the highlights of the international endocrine meeting. Hard work for me to cover, but it would inform those unable to attend the meeting about the latest developments and areas of interest. The pharmaceutical company agreed and said it was an excellent suggestion.

About 3 weeks before the meeting, I was asked for Powerpoint slides of my highlights presentation. I replied that I could not provide slides, as the meeting and lectures had not yet taken place. Despite this, the request was repeated, so I informed the company that, for my webcam performance, I would read my notes taken at the lectures, and there would be no slides whatsoever.

The company was nonplussed and insisted that a webcam lecture had to have visual aids, other than my face. With less than 2 weeks to go I was becoming increasingly anxious. I decided on an alternative strategy and offered the company a choice of two lectures that I had recently prepared on topics to be discussed at the meeting.

The company accepted my offer with alacrity and gratitude. With only a few days to go, I sent them my Powerpoint presentation of the chosen lecture, and breathed a sigh of relief. Their email reply arrived almost instantaneously, 'We think the idea of this lecture is excellent and we are most grateful for your Powerpoint presentation. By the way, when will you be sending the slides of the highlights?'

'HOTSPUR'

Fat of the land or land of the fat?

The public enjoyed learning more about another topical area of endocrinology at a recent Society event.

▶ This was the title of the Society's latest highly successful event to engage the interest of the general public in endocrinology.

Part of the BA Festival of Science in Liverpool in September, the event saw experts in the field of obesity examine how to solve the problem of our nation's ever-expanding waistlines. It included a series of scientific talks and a panel debate, which were delivered to a packed lecture theatre of over 80 members of the public. The animated speakers successfully engaged the public in learning about their research, which sparked lively discussion amongst members of the audience.

Ian Macdonald provides background

Quentin Cooper (of BBC Radio 4's Material World) was our host and did a fantastic job of capturing the audience's attention throughout the session. He opened the proceedings with a witty introduction to the current obesity crisis that left the audience wondering whether a spoon full of sugar helps the medicine go down, or a spoon full of medicine helps the sugar go down.

'Starters' were then dished up by Prof Ian MacDonald (University of Nottingham), who provided a general background on the contributing factors and prevalence of obesity.

The first of three scientific talks was presented by Dr Giles Yeo, a geneticist from the University of Cambridge. Dr Yeo charmed the audience with a captivating talk on genetic predisposition to obesity, aptly entitled 'Are my genes to blame when my jeans don't fit?' Dr Yeo explained how genes play a crucial part in an individual's susceptibility to obesity and how the heritability of body fat is between 40 and 70% - similar to that of height.

Next to take the floor was Dr Helen Budge (University Hospital Nottingham), who described how nutrition in the womb and early infancy influences our risk of developing obesity in later life. The audience learnt how too little nutrition during early fetal development increases the risk of obesity, whereas too few calories in the last months of pregnancy can lead to high blood pressure and diabetes.

Dr Rachel Batterham (University College London) rounded off the first session with an engaging talk on how our gut hormones play a crucial role in controlling our hunger and weight. Dr Batterham spoke about ghrelin, dubbed by the press as the 'hunger hormone', and described how our gut hormone system was not designed for today's 'obesogenic' environment.

After a refreshment break to satisfy our elevated ghrelin levels, we were treated to a panel debate on how to tackle the issues of obesity. Kept in check by Quentin

The audience quiz the expert panel

Cooper, a panel of four experts debated over whether surgery, dietary intervention, exercise or lifestyle changes were the solution to the nation's obesity crisis. This informative and entertaining session was opened by Dr Carel le Roux (Imperial College London) who presented the case for surgery as the only proven way to lose weight and live longer. He explained to the audience how obesity surgery improves a patient's health by enabling them to consume fewer calories to satisfy their hunger.

Gill Fine (Food Standards Agency) then presented the case for dietary intervention through food labelling and government initiatives. She explained how food labelling schemes such as the 'traffic light system' have been set up by the Government to clearly identify the salt and saturated fat content of products, in a bid to tackle the obesity problem.

The case for physical activity was given by Prof Ron Maughan (Loughborough University) who presented exercise as the cheapest, easiest and safest option for avoiding the unwanted consequences of excess body fat.

Dr Alexandra Johnstone (Rowett Research Institute) concluded the panel talks by using a 'weight suit' to illustrate her case for lifestyle interventions. The suit was an excellent tool for demonstrating the strain excess weight can put on your body. Dr Johnstone explained how weight loss needs to be a gradual process and is best achieved and maintained by diet and lifestyle changes.

Following these interesting talks, the floor was opened for questions and the audience put the panel through their paces in a thought-provoking debate session.

We were delighted that one of our speakers, Dr Giles Yeo, was selected to take part in the BA x-change: a daily round-up of the 'best of the fest', where festival goers are encouraged to share their opinions on the hot topics of the day. Dr Yeo delivered a fantastic summary of his talk to a packed bar, and expertly fielded questions from a lively audience.

This event formed part of the biology section at the Festival and was jointly organised by the Society for Endocrinology, Nutrition Society and Biochemical Society, with sponsorship from the Biosciences Federation.

Overall this was a fantastic morning of talks which clearly engaged the public in the science of obesity. We were delighted that our event received excellent press coverage, with prominent articles in *The Guardian*, *The Daily Telegraph*, *Daily Mail*, and on BBC News Online. The Society extends its thanks to all the speakers involved in this successful event and we look forward to increasing the number of public events we hold at science festivals in the near future. If you would like to get involved in a public event, email rebecca.ramsden@endocrinology.org.

REBECCA RAMSDEN

Society for Endocrinology

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- The entrant must promise that the article is their original work. If it contains material such as a previously published illustration or table, the material must be clearly identified and acknowledged within the text.
- The essay must have been written in the current academic year by an undergraduate student at an institution of higher education in the United Kingdom or Ireland.
- The Society reserves the right to withhold the prize if the standard of the essays submitted is not of sufficient merit.
- The Society reserves the right to verify undergraduate credentials.
- The essay must be no more than 1500 words.
- The closing date for submission is 2 March 2009.
- If your essay is not amongst the prize winners, all rights revert back to the author.

HOT TOPICS

The latest research from the Society's journals brought to you by Andrew Lowe, Claire Eudall and Laurie Mousah.

TAMs in thyroid cancer

Infiltrating inflammatory cells are major constituents of tumour microenvironments, of which tumour-associated macrophages (TAMs) may make up as much as 50% of the tumour mass. TAMs secrete a rich repertoire of chemokines and growth factors, which may be involved in tumour cell growth, spread, mobility, migration and invasion.

Clinical studies in human breast, prostate and cervical cancer support a role for TAMs as tumour promoters. Ryder and colleagues have examined TAMs in several histological thyroid cancer grades, and observed quite clearly that those thyroid cancers that are heavily infiltrated with macrophages strongly correlate with aggressive properties and a higher mortality.

This suggests that TAMs promote thyroid cancer progression, a novel paradigm that has major clinical and possible therapeutic relevance, as many of the therapies that are currently being developed target cell-intrinsic pathways activated by oncogenic kinases. The effectiveness of these therapies may be compromised if tumour progression is driven via inflammatory mediators produced by innate immune cells. **AL**

(See the full article in Endocrine-Related Cancer ***15****(4), December 2008)*

Sex steroid regulation of mitochondrial fusion and fission

Astrocytes and steroid hormones like oestrogen and progesterone can act together to promote cell survival under pathological conditions, by regulating processes such as mitochondrial fusion and fission in energy metabolism, apoptosis, and proliferation.

In this study by Arnold and co-workers, the effects of oestrogen and progesterone on cell viability were studied in comparison with mitochondrial fusion and fission gene transcription in primary cortical astrocytes from female and male mouse brains. The oestrogen- and progesterone-treated female astrocytes showed an increase in cell numbers, while oestrogen-treated male astrocytes showed no change. Progesterone-treated male astrocytes showed a decrease in cell numbers, possibly as a consequence of greater stimulation of apoptosis.

The data show for the first time that sex steroids can influence fusion and fission in the mitochondria of astroglia, and suggest that the effects of oestrogen and progesterone in cortical astrocytes are gender-specific. The interaction of these steroids with mitochondria may represent a possible cause for gender differences in cellular pathology in the CNS, such as the higher incidences of neurodegenerative diseases in males. **CE**

(See the full article in Journal of Molecular Endocrinology ***41****(5), November 2008)*

Metformin maintains weight loss in PCOS

Polycystic ovary syndrome (PCOS) is a heterogeneous syndrome of hyperandrogenic anovulation that is typically due to intrinsic ovarian dysfunction. Adults with PCOS are at increased risk of metabolic syndrome and related cardiovascular disease, so encouraging weight loss is important in the clinical management of obese PCOS patients.

Metformin can reduce central adiposity in obese PCOS patients, but does not enhance overall weight reduction. Rimonabant has been shown to reduce weight, free androgen index (FAI) and insulin resistance in obese PCOS patients compared with metformin. However, serious weight regain can occur following cessation of rimonabant treatment.

Sathyapalan and colleagues have investigated whether subsequent metformin treatment after rimonabant would maintain the improvement in weight, hyperandogenaemia and insulin resistance. The study found that a mean weight loss of 6.2 kg associated with 3 months of rimonabant treatment was maintained by 3 months of metformin treatment, combined with the improved FAI and insulin resistance scores, when compared with 6 months of metformin treatment alone. **AL**

(See the full article in Clinical Endocrinology ***70****(1), January 2009)*

Adult stem/progenitor cells in the human thyroid

The pluripotent nature of embryonic stem cells has been widely reported, sparking much debate about their potential for use in tissue regeneration. However, less is known about stem cells found in adult tissues. The significance of the regenerative properties of adult stem cells remains largely unclear. This is partly due to their rarity and difficulties in locating and identifying their progeny.

Fierabracci and colleagues have devised a new method to isolate stem cells from the human thyroid that includes digesting specimens using enzymes and culturing the residual fragments with EGF and bFGF. Their data demonstrate that spheroids with self-replicative potential were collected from all thyroid specimens and that stem/progenitor cells are located in the thyroid. Moreover, the thyroid is not terminally differentiated; it has the ability to generate thyroidal cells and the potential to produce non-thyroidal cells.

The authors suggest that adult stem cells could replace the use of embryonic stem cells in regenerative medicine. **LM**

(See the full article in Journal of Endocrinology ***198****(3), September 2008)*

British Pharmacological Society: Joint Symposium of the Obesity and Metabolic Diseases Special Interest Group and Clinical Pharmacology Section

17 December 2008, Brighton, UK.

Contact: Sarah Mackay, British Pharmacological Society (Email: sm@bps.ac.uk).

Fertility 2009

7-9 January 2009, Edinburgh, UK.

Contact: Jessica Canfor, Profile Productions Ltd, Northumberland House, 11 The Pavement, Popes Lane, London W5 4NG, UK (Tel: +44-20-88327311; Fax: +44-20-88327301; Email: fertility@profileproductions.co.uk; Web: www.fertility2009.org).

Type 2 Diabetes and Insulin Resistance

20-25 January 2009, Banff, Alberta, Canada.

Contact: Keystone Symposia (Email: info@keystonesymposia.org; Web: www.keystonesymposia.org/Meetings/ViewMeetings.cfm?MeetingID=996).

Obesity: Novel Aspects of the Regulation of Body Weight

20-25 January 2009, Banff, Alberta, Canada.

Contact: Keystone Symposia (Email: info@keystonesymposia.org; Web: www.keystonesymposia.org/Meetings/ViewMeetings.cfm?MeetingID=997).

Expanding Boundaries of Endocrinology

27 January 2009, London, UK.

Contact: Jo Summers, Royal College of Physicians (Email: conferences@rcplondon.ac.uk; Web: www.rcplondon.ac.uk/event/details.aspx?e=1379).

Ion Channels as Therapeutic Targets 2009

5-6 February 2009, Horsham, UK.

Contact: British Pharmacological Society (Email: meetings@bps.ac.uk; Web: www.bps.ac.uk).

Advanced Endocrinology

6-8 February 2009, Singapore.

Contact: Sarah Ten (Email: mice2@themeetinglab.com; Web: www.endometab.com).

5th Asia-Oceania Conference on Obesity

6-8 February 2009, Mumbai, India.

Contact: Euan Woodward, Obesity International Trading Ltd, 231 North Gower Street, London NW1 2NR, UK (Tel: +44-20-76911900; Email: ewoodward@iaso.org; Web: aiaaro.com/aiaaro2009).

Society for Endocrinology Clinical Cases Meeting in association with the RSM

10 February 2009, London, UK.

Contact: Conference Team (Email: conferences@endocrinology.org; Web: www.endocrinology.org/meetings/2009/cc2009).

Complications of Diabetes and Obesity

24 February-1 March 2009, Vancouver, BC, Canada.

Contact: Keystone Symposia (Email: info@keystonesymposia.org; Web: www.keystonesymposia.org/Meetings/ViewMeetings.cfm?MeetingID=998).

52nd Symposium of the German Endocrine Society

4-7 March 2009, Giessen, Germany.

Contact: Reinhard G Bretzel, Third Medical Department, University Hospital Giessen and Marburg, Rodthohl 6, D-35392 Giessen, Germany (Tel: +49-641-9942750; Fax: +49-641-9942759; Email: reinhard.bretzel@uniklinikum-giessen.de; Web: www.giessen2009.de).

6th Annual ENETS Conference

5-7 March 2009, Granada, Spain.

Contact: ENETS (Email: enets.office@charite.de; Web: www.neuroendocrine.net/rel).

Diabetes UK Annual Professional Conference 2009

11-13 March 2009, Glasgow, UK.

Contact: Conferences Team, Macleod House, 10 Parkway, London NW1 7AA, UK (Tel: +44-20-74241000; Fax: +44-20-74241081; Email: conferences@diabetes.org.uk; Web: www.diabetes.org.uk/apc2009).

15th Annual Meeting of the International Society for Clinical Densitometry

11-14 March 2009, Orlando, FL, USA.

Contact: International Society for Clinical Densitometry (Tel: +1-860-5867563; Fax: +1-860-5867550; Email: iscd@iscd.org; Web: www.iscd.org).

Society for Endocrinology BES 2009

16-19 March 2009, Harrogate, UK.

Contact: Shirine Borbor, Society for Endocrinology, 22 Apex Court, Woodlands, Bradley Stoke, Bristol BS32 4JT, UK (Tel: +44-1454-642210; Fax: +44-1454-642222; Email: conferences@endocrinology.org; Web: www.endocrinology.org/meetings/2009/sfebes2009).

ECCEO 9 (European Congress for Clinical and Economic Aspects of Osteoporosis and Osteoarthritis)

18-21 March 2009, Athens, Greece.

Contact: YP Communication, Boulevard G. Kleyer 108, B-4000 Liège, Belgium (Tel: +32-4-2541225; Fax: +32-4-2541290; Email: yolande@piettecommunication.com; Web: www.ecceo9.org).

2nd Joint Meeting of the International Bone and Mineral Society and the Australian and New Zealand Bone and Mineral Society

21-25 March 2009, Sydney, NSW, Australia.

Contact: International Bone and Mineral Society (Web: www.ibms2009.com).

Cell Death Pathways

22-27 March 2009, Whistler, BC, Canada.

Contact: Keystone Symposia (Email: info@keystonesymposia.org; Web: www.keystonesymposia.org/Meetings/ViewMeetings.cfm?MeetingID=970).

European Association for the Study of Obesity Bjorntorp Symposium

24-27 March 2009, Prague, Czech Republic.

Contact: EASO, 231 North Gower Street, London NW1 2NR, UK (Email: enquiries@easo.org; Web: www.easo.org).

3rd International Meeting on Skeletal Endocrinology

27 March 2009, Brescia, Italy.

Contact: Secretariat (Email: skeletal@euroconventions.it; Web: www.skeletal-endocrinology.org/presentazione.asp).

43rd Annual Scientific Meeting of the European Society for Clinical Investigation

1-4 April 2009, Frankfurt/Main, Germany.

Contact: Nicola Bock-Schildbach, Congress and Promotion, Amselweg 7, Königstein 61462, Germany (Tel: +49-6174-933595; Fax: +49-6174-933596; Email: Bock-Schildbach@esci2009.com; Web: www.esci2009.com).

IASO Stock Conference: Obesity and Inflammation

3-6 April 2009, Cairns, Qld, Australia.

Contact: IASO (Tel: +44-20-76911900; Email: stock@iaso.org; Web: www.iaso.org/stock2009.asp).

Annual Meeting of the American Society for Biochemistry and Molecular Biology

18-22 April 2009, New Orleans, LA, USA.

Contact: ASBMB, ASBMB Meetings Office, 9650 Rockville Pike, Bethesda, MD 20814-3996, USA (Email: meetings@asbmb.org; Web: www.asbmb.org/Page.aspx?id=146).

20th National Meeting of the British Neuroscience Association

19-22 April 2009, Liverpool, UK.

Contact: British Neuroscience Association (Email: bna2009@liv.ac.uk; Web: www.bna.org.uk/bna2009).

3rd Focused Meeting on Cell Signalling

20-21 April 2009, Leicester, UK.

Contact: BPS (Email: meetings@bps.ac.uk; Web: www.bps.ac.uk).

11th European Congress of Endocrinology

25-29 April 2009, Istanbul, Turkey.

Contact: European Society of Endocrinology, c/o Sezen Elagoz, TeamCon Congress Services Worldwide, Halaskargazi Caddesi Alp Palas Apt. No:79/1, Harbiye, Istanbul 34371, Turkey (Tel: +90-212-3438003; Fax: +90-212-3438023; Email: secretariat@ece2009.com; Web: www.ece2009.com).

American Association of Endocrine Surgeons Annual Meeting 2009

2-5 May 2009, Madison, WI, USA.

Contact: AAES (Email: cartyse@upmc.edu; Web: www.endocrinesurgery.org/mtgs/future_meetings.cfm).

17th European Congress on Obesity - ECO 2009

6-9 May 2009, Amsterdam, The Netherlands.

Contact: ECO 2009 (Email: eco2009@easoobesity.org; Web: www.easoobesity.org/eco2009).

18th Annual Meeting and Clinical Congress of the American Association of Clinical Endocrinologists

13-17 May 2009, Houston, TX, USA.

Contact: AACE, 245 Riverside Ave, Suite 200, Jacksonville, FL 32202, USA (Tel: +1-904-3537878 ; Email: asanders@aace.com; Web: www.aace.com/meetings/calendar/calendar.php).

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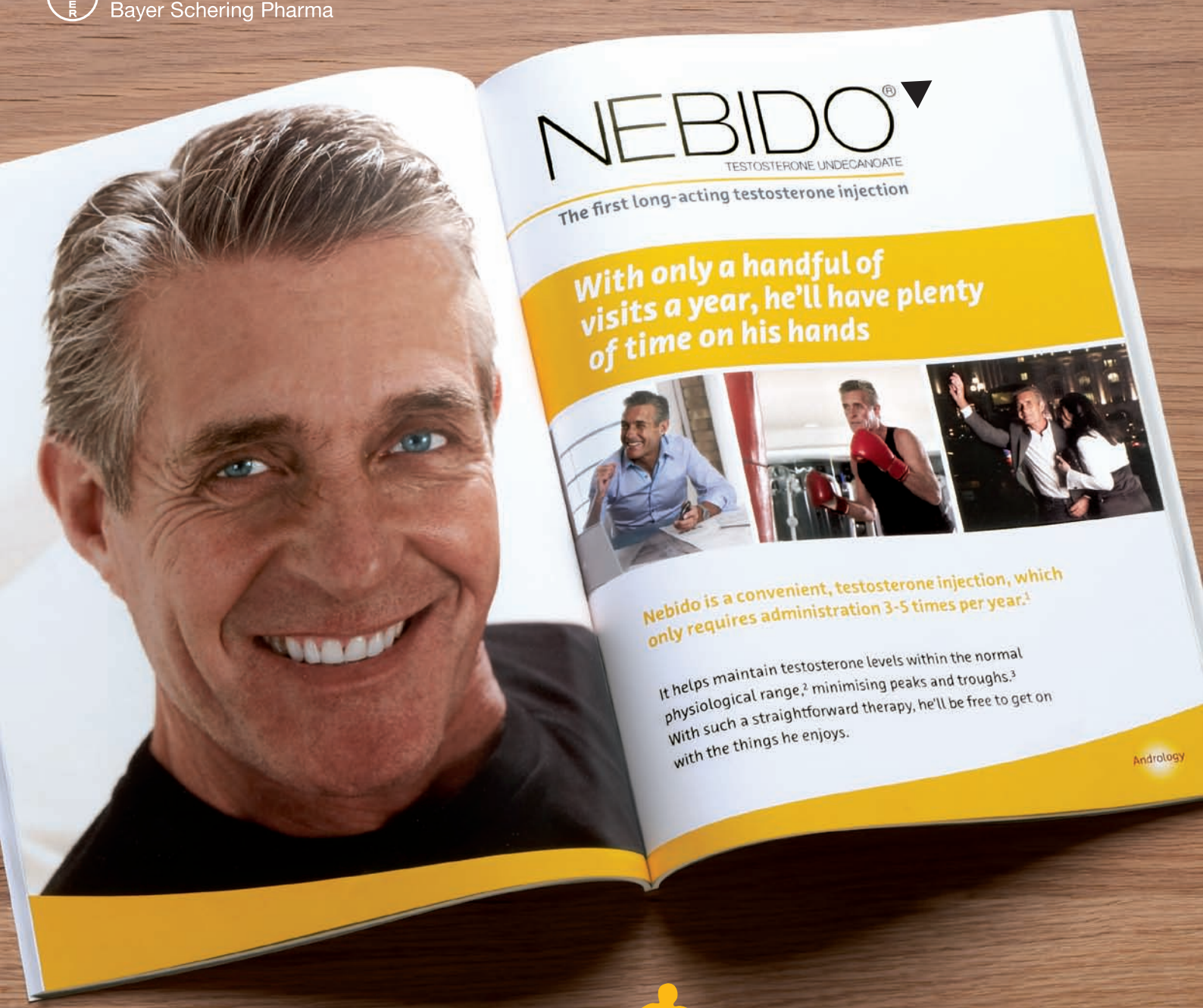

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PRESCRIBING INFORMATION Nebido® (testosterone undecanoate) Presentation: Ampoule with 4ml solution for injection containing 1000mg testosterone undecanoate. **Uses:** Testosterone replacement therapy for male hypogonadism when testosterone deficiency confirmed by clinical features and biochemical tests. **Dosage:** One ampoule (1000mg) injected intramuscularly every 10 to 14 weeks. *Starting treatment*: Measure serum testosterone levels before start and during initiation of treatment. If appropriate, first injection interval may be reduced to a minimum of 6 weeks. *Maintenance*: Injection interval within 10 to 14 week range. Monitor serum testosterone regularly; adjust injection interval as appropriate. **Children:** Not for use in children. Not evaluated clinically in males under 18. **Contra-indications:** Androgen-dependent prostate cancer or breast cancer. Past or present liver tumours. Hypersensitivity to testosterone or any of the excipients. **Warnings and precautions:** Limited experience in patients over 65. Before therapy exclude prostate cancer. Examine prostate and breast at least annually, or twice yearly in elderly or at risk patients (clinical or familial factors). Periodically check testosterone concentrations, haemoglobin, haematocrit, liver function. Androgens may accelerate the progression of sub-clinical prostate cancer and benign prostatic hyperplasia. Monitor serum calcium concentrations in cancer patients at risk of hypercalcaemia (and hypercalcinuria). Rarely, liver tumours have been reported. Nebido may cause oedema with or without congestive cardiac failure in patients with severe cardiac, hepatic or renal insufficiency or ischaemic heart disease. In this case, stop treatment immediately. Use with caution in patients with renal or hepatic impairment, epilepsy, migraine or blood clotting irregularities. Improved insulin sensitivity may occur. Irritability, nervousness, weight gain, prolonged or frequent erections may indicate excessive androgen exposure requiring dose adjustment. Withdraw treatment if these symptoms persist or reappear. Pre-existing sleep apnoea may be potentiated. Testosterone may produce a positive reaction in anti-doping tests. Not for use in women. Not suitable for developing muscles or increasing fitness in healthy individuals. Inject Nebido extremely slowly to avoid the coughing or respiratory distress reactions that occur rarely with injection of oily solutions. Interactions reported with oral anticoagulants (requires dose monitoring), ACTH or corticosteroids, and thyroxin binding globulin in laboratory tests. **Side-effects:** Most common reactions are injection site pain (10%). Also reported are: diarrhoea; leg, breast or testicular pain; arthralgia; dizziness; increased sweating; headache; respiratory, skin or prostate disorders; acne; gynaecomastia; pruritus; subcutaneous haematoma at injection site. Other known reactions to testosterone containing preparations are: polycythaemia (erythrocytosis); weight gain; electrolyte changes; muscle cramps; nervousness, hostility, depression; sleep apnoea; very rarely jaundice and liver function test abnormalities; skin reactions; libido changes; increased frequency of erections; interruption or reduction in spermatogenesis; priapism; prostate abnormalities; prostate cancer (inconclusive data); urinary obstruction; water retention; oedema; hypersensitivity. **Basic NHS Price:** £76.70 per 1 x 4ml **Legal Classification:** POM **Product Licence Number:** 0053/0350 **Product Licence Holder:** Bayer plc., Bayer House, Strawberry Hill, Newbury, Berkshire RG14 1JA **Nebido® is a registered trademark of Bayer Schering Pharma AG (formerly Schering AG). PI revised:** 1 May 2008

References: 1. Nebido Summary of Product Characteristics. **2.** Von Eckardstein S *et al. J Androl* 2002; **23**(3): 419-425. **3.** Gooren LJG and Bunck MCM. *Drugs* 2004; **64**(17): 1861-1891. 8NEBI26a May 2008

Information about adverse reaction reporting in the UK can be found at www.yellowcard.gov.uk Alternatively, adverse reactions can be reported to Bayer plc by email: phdsguk@bayer.co.uk