The potential benefits of saturated fat in people’s everyday diet

Autophagy: An Unsung Hero

Levels of obesity are now affecting large numbers of the population along with its associated health risks. What could our future hold if we looked back at our ancestors to help understand lessons from the past to ascertain the context of what an optimal diet for us could look like?

Conventional wisdom has told us that saturated fat equals poor risk however, what if this is not the case. In this article we examine a different view based on a thought provoking talk ‘Insurance and the Rise of Evolutionary Medicine’ given at the LUCID conference in Brighton.

In recent years there have been major advances in our understanding of very specific cellular processes, and how they work to keep cells healthy. Pharmaceutical research scientists are working hard, attempting to harness them, to develop commercially viable drugs. On the other hand, simple non-commercial methods of activating them get little attention. In this article we will discuss a very ancient (yet recently discovered) cellular repair pathway and its connection to diet.

Apoptosis, which was first described in 1842, has occasionally made medical headlines. The word, derived from Greek, roughly translates to “falling off”. Apoptosis is defined as “the process of programmed cell death that may occur in multicellular organisms”. Unlike cell death by necrosis, apoptosis is a relatively civil affair. Cellular signaling calls in macrophages to clean up the mess, leaving no trace of the cell and causing none of the damage that necrotic cells do. It is estimated that in the average adult between 50 and 70 billion cells quietly die each day by apoptosis! That’s quite a few funerals.

Apoptosis plays many roles. For example, the differentiation of fingers and toes in a developing human occurs because cells between the fingers apoptose. It also plays a role in preventing cancer: There are mechanisms that detect fatal genetic errors in cells. To protect the rest of your multicellular republic, such defective cells exhibit the apoptotic response and commit suicide. Clearly, the inactivation of apoptosis is central to the development of cancer. In fact, numerous cancer therapies attempt to reactivate the apoptotic response in cancerous cells.

It turns out that apoptosis has a cousin that hasn’t had its share of the limelight: Autophagy. It too translates from Greek, and means “self eating”. Despite sounding sinister, autophagy plays a very important role in maintaining cellular order. Whilst we can trace its roots back more than 3 billion years, we’ve only been able to understand its mechanisms for about 15. Where apoptosis terminates delinquent cells, autophagy plays a role in preventing them from becoming ungovernable in the first place. From a multicellular organism’s perspective both autophagy and apoptosis promote the health of the cellular republic. From a citizen cell’s perspective autophagy, unlike apoptosis, promotes life rather than death.

Autophagy can be defined as “the basic catabolic mechanism that involves cell degradation of unnecessary or dysfunctional cellular components through the actions of lysosomes.” It is an
evolutionarily conserved stress response that is present in all living cells. Nutrient stress (ie a shortage of nutrients) is one of its most potent activators. Part of its role is to scavenge cells, in search of damaged proteins and organelles for recycling, thus putting the building blocks of life to better use. This has the obvious health benefit of eliminating damaged cell components that may go on to later impair the functioning of the cell and lead to disease in the republic of cells. From the scientific literature we find that autophagy protects against genomic instability giving it a key role in preventing diseases such as cancer, neurodegeneration, cardiomyopathy, diabetes, liver disease, autoimmune diseases and infections. Sounds important!

The evolutionary purpose of a cellular self-repair and recycling mechanism is obvious: During periods of protein deficiency, the cell will nevertheless require some basic amino acid building blocks to ensure it can continue to function. Selectively degrading misfolded proteins and damaged organelles for this purpose makes sense, serving a double benefit: It removes cellular junk, and provides much needed nutrients during times of nutrient shortage. Nature would long ago have weeded out cells that indiscriminately tried to recycle any random cellular component, even if it still functioned well. At least two systems are involved in this recycling process: Autophagy (which we discuss here) and the ubiquitin-proteasome system (which we don’t).

Autophagy explains quite elegantly why caloric restriction has life extension benefits in all model animals that have been studied. Reducing caloric intake is now known to activate autophagy, and based on the discussion above would be expected to clean out cellular garbage, so to speak, and promote health. Indeed, a fascinating 2007 paper titled “Autophagy is required for dietary restriction-mediated life span extension in C. elegans” puts forward that caloric restriction does not work without autophagy. This suggests that autophagy is caloric restriction’s primary mechanism of action.

What then is the primary regulator of autophagy? The pharmaceutical industry is naturally working hard to find novel patentable compounds that regulate it and why would they do otherwise? They are beholden to their shareholders. It turns out that one widely unnoticed cellular signalling pathway is a major regulator of autophagy. This pathway is called mTOR, or the Mechanistic Target of Rapamycin. This ancient pathway is common to all living cells and can be traced right back to the LECA or the Last Eukaryotic Common Ancestor, mother to most living cells today.

Bio-prospecting is defined as the search for plant and animal species from which medicinal drugs and other commercially valuable compounds can be obtained. In 1964 a group of Canadian scientists went bio-prospecting to the Easter islands. They brought back a soil sample containing a bacterium. This bacterium produced an antifungal molecule, later called Rapamycin, that proved to have a range of fascinating and profound biological effects in a variety of cell types. These effects lead to the question: What is the target of rapamycin? When they found it, they unimaginatively named it the Target of Rapamycin (TOR). In the intervening years we have come to appreciate that the TOR complex has much more to it than just Rapamycin, and in that sense the name is misleading and unfortunate.

In the last 15 years or so TOR research has exploded. In the same way that the Insulin/IGF cell signalling pathway is the primary nutrient sensing pathway for glucose, TOR is the primary nutrient sensing pathway for amino acids. It turns out that an abundance of amino acids up-regulates TOR which reduces autophagy while simultaneously encouraging cell division. This makes sense: Cells should divide more readily when there is an abundance of nutrients.
Autophagy promotes good health at a cellular level by basically taking out the garbage. And autophagy is largely regulated by mTOR. So the crucial question is: What regulates mTOR? As is alluded to above, mTOR is controlled predominantly by diet. Specifically the protein and carbohydrate content. A diet high in either of these will up-regulate mTOR and reduce healing autophagic activity. Interestingly, fat in the diet does not affect mTOR. Ron Rosedale, MD and internationally renowned expert in aging and metabolic disease, has for years been advocating a health promoting diet. Rosedale, Westman & Konhilas have a paper out titled “Clinical Experience of a Diet Designed to Reduce Aging”. In this paper they show that people who follow Rosedale’s diet develop a metabolic profile that is similar to that of centenarians.

He has had astonishing results in his patients, treating them only with diet: Specifically a high fat / adequate protein / very low carbohydrate diet. Rosedale has been promoting this way of eating since as far back as Atkins has. The crucial difference being that Atkins ignored the perils of excess protein. Developments in aging research over the last two decades have largely vindicated Rosedale’s original work, which now looks almost prophetic. People on his diet experience all the benefits of caloric restriction, without actually restricting calories: They consume unlimited amounts of healthy fats, leading to delicious food and great satiety.

If you are curious, and would like to know more about autophagy, mTOR, and Rosedale’s ideas that discuss them then the web is a great resource. His essays can easily be found and PubMed literally has thousands of papers discussing autophagy and mTOR. Sadly, the tone of most of them ignore diet’s influence and focus on the quest for patentable compounds. As if a deficiency in such compounds caused the health problems in the first place.

Jordi Posthumus
Vice President – Longevity Trade
Hannover Life Re Bermuda

Following our recent Underwriting and Claims Seminar, Time and Tide wait for no man, we are pleased to share further talks from the day. In our June issue we covered diseases of childhood and adolescence. Here we will be focusing on the Multiple Sclerosis (MS) sessions sharing three different perspectives.

**A personal insight into Multiple Sclerosis**

Hannover Re UK Life Branch Claims Assessor Theresa Taylor offered an informative and moving account of her own experience of diagnosis and living with MS. She described the stages of her disease and diagnosis, highlighting the lessons for those assessing MS claims. As both a claims assessor and a critical illness policyholder, Theresa has a unique insight into the claims and underwriting issues around MS.

The first symptoms suggesting something might be seriously wrong appeared to come when Theresa experienced pins and needles in her tongue and lips and a tremor in her right hand in May 2010. In retrospect, however, after consulting her own medical notes, she realised there had been early indications as far back as 2007. Theresa’s GP ascribed these early symptoms to the side effects of medication she was taking for an unrelated minor illness.

The symptoms of May 2010 subsided and life went on as usual until October that year, when Theresa experienced tingling in the right side of her face – followed by pins and needles, pain and tightness in her right leg. This left her with weakness, altered sensation and clumsiness in the leg for around a month. Two months later, the symptoms returned accompanied by severe bouts of fatigue. By January 2011 she was having difficulty even lifting her limbs. On hearing of these symptoms, HR UK Medical Officer Dr Emile de Sousa, urged Theresa to go back to her GP, who arranged an urgent neurology appointment.

Following an MRI brain scan, Theresa received a call from the...
neurologist asking her to return for an urgent appointment and a second scan. The GP, Theresa discovered when she saw her notes, had referred her with a suspected brain tumour, but lesions in her periventricular white matter and in her brain stem lead the neurologist to diagnose a Clinically Isolated Syndrome. He advised Theresa there was a risk of developing MS, but that, for now, she should continue with her life, watch and wait.

She did not have long to wait. Theresa had her first major relapse during a visit to her parents in Somerset when she experienced heavy limbs and difficulty swallowing. She narrowly made it home down the motorway before a major attack set in. This left her with slurred speech, facial droop, difficulties swallowing and the loss of use of her right arm and leg for several days.

Further investigations followed in March 2011, including another MRI brain scan and a scan of her cervical and thoracic spinal cord, which found a couple of lesions, as well as a lumbar puncture, which Theresa had previously declined. This came back positive for oligoclonal bands. After Lyme’s Disease, Lupus and other inflammatory diseases had been ruled out, she was diagnosed with Relapsing & Remitting MS in May 2011.

Theresa spoke movingly about the effect of her diagnosis on her family and of strong support from friends, family, her MS nurse and from her colleagues at HR UK. She then described her experience with disease modifying drugs. After having an adverse reaction to Copaxone, she was initially reluctant to try Tysabri because of the risk of contracting a rare but potentially fatal brain infection called Progressive Multifocal Leukoencepha Iopathy (PML).

After going a year without disease modifying drugs – during which she was relapsing every three to four months – Theresa was finally persuaded that it would be better to take the plunge and give herself a better chance of continuing to function at a high level for longer.

Theresa spoke with stoicism about the future, noting that she hoped the Tysabri would prove effective in warding off further relapses. If not, she said, she would probably stop taking it because of its daunting side effects. Theresa thanked her extended support network warmly and said she hoped to benefit from some of the newer treatments now coming into use and expressed gratitude at not having a life-threatening condition.

Looking back on the history of diagnosis through to claim on her CI policy, which Theresa had held since she was 18, she read in her notes that she had been to the GP with a number of symptoms that might have been early signs of MS, about which she had completely forgotten. These included a burning sensation in her right thigh as far back as July 2007, breathlessness and fatigue in April 2008, and memory and concentration problems in February 2009. She had also forgotten the occasion on which in December 2009 she had complained of muscle spasms in her leg plus sensory changes in the thigh and had been told (as the GP’s notes recorded) that the cause was wearing boots that were too high and too tight!

None of these symptoms or consultations came to mind when she was asked about her past medical history by the neurologist and why would it. After all, she had been reassured each time that nothing sinister was going on.

As a claims assessor, Theresa wondered if we are running the risk of being biased when deciding the category of non-disclosure. The relevance of her symptoms and consultations took on a different meaning once she had sight of the medical notes and a diagnosis of MS. Unless the GP had already made the links and disclosed his concerns with his patient, the claims assessor must resist the consultations/symptoms collectively. Instead consider each one in isolation and in conjunction with the specific questions on the application form, so they have a better understanding of the possible misrepresentation – the benefit of hindsight is a wonderful thing.

We should also be aware that patients will not necessarily be aware of everything that appears in their medical notes. After all, Theresa had no idea her initial GP referral had been for cancer (the suspected brain tumour) until she read it in her notes long afterwards. The definition for MS requires six months’ continuous symptoms. Theresa noted that, if her steroid treatment had worked, she would only have had five months continuous symptoms! Theresa also stressed that GPs and neurologists are likely to record only those symptoms about which they are directly concerned in their notes, making it important to check MS nurses’ notes as well to get a fuller picture of a claimant’s condition.
A GP’s perspective on MS

HR UK Medical Officer Emile de Sousa’s talk focused on the roles of GPs and specialists in the diagnosis and treatment of MS. Since GP’s notes are a key source of information for those evaluating MS claims, he suggested, an insight into the GP’s perspective would be extremely helpful to claims assessors. His presentation included three brief case histories that illustrate the respective roles of GPs and specialists in the diagnosis and treatment of MS.

The first involved a 23 year old woman who attended Dr de Sousa’s surgery complaining of stumbling and of altered sensation in various parts of her body. He examined her and found her cranial nerves and cerebellum to be normal, but identified alterations in sensation in areas of skin supplied by different nerve roots and localised muscular weakness, suggesting a provisional diagnosis of Demyelination.

Three weeks later the neurologist found there had been a progression in her symptoms, with evidence of optic neuritis and transverse myelitis. Her Visual Evoked Potentials showed a delayed right eye response and a lumbar puncture revealed oligoclonal bands. This enabled the neurologist to confirm a diagnosis of MS.

Over the course of 51 appointments over four and a half years, the patient was treated for an incidental vitamin B12 deficiency picked up in the initial blood tests and for severe cystic acne (for which she subsequently referred her to a specialist dermatologist) as well as discussing pregnancy and the associated risks. Over the course of 30 appointments, the neurologist treated her first with methylprednisolone and then, as her symptoms progressed, with Copaxone and subsequently Avonex. This currently appears to be working, Dr de Sousa said, and the patient is “doing well”.

A second patient, a 45 year old nurse, came to Dr de Sousa, with a burning sensation in her right hand and on examination exhibited a weak triceps reflex. He referred her for an MRI which shed no light. She was reluctant to have a lumbar puncture. The symptoms subsided and no more was heard about it until ten months later when the patient reported sudden acute visual loss, leading Dr de Sousa to suspect an embolism. She was referred to a cardiologist who found no evidence of a vascular event and referred her on to an ophthalmologist. The latter suggested she had optic neuritis – although no evidence of this was seen when she was examined.

Ten months later, she reported an unpleasant burning sensation in her lower left leg and pain in her right arm. A colleague of Dr de Sousa’s prescribed amitriptyline for neuropathic pain, which appeared to work. On a third examination she had reduced sensitivity to pinprick at the left L3 and L4 dermatomes, a brisk right ankle reflex and a depressed left ankle reflex. She was referred to a neurologist. In this instance, however, the tests came back normal and the precise cause of her symptoms remains unknown.

The third case study was that of a 47 year old woman with mild anxiety and hypochondriasis who came to see Dr de Sousa in 2005 with weakness in both legs. On examination she exhibited brisk reflexes and an MRI scan found transverse myelitis. No action was required as it was resolving on its own. Four years later, in May 2009, she came back reporting easy fatigability in her right leg and foot and clumsiness. On examination, however, she appeared normal.

By the following month she was experiencing marked weakness and was having difficulty getting out of her chair and on examination this time, she appeared to be
is found to have lesions in their brain and/or spinal column that look like MS, moving on to Clinically Isolated Syndrome (CIS), where they have had one attack, Relapsing-Remitting MS (RR MS) where there has been more than one attack, Transitional MS, an intermediate stage between RR MS and progressive disease, Secondary Progressive MS (SP MS), Primary Progressive MS (PP MS), and finally ‘Advanced MS’.

He stressed, however, that these categories are not fixed or inherent, but a question of judgement or interpretation on the part of the neurologist. A person will often be found to have had a number of silent lesions in their brain before they experience their first attack. Dr Silber likened the difference between silent lesions and ‘eloquent’ lesions (i.e. those that result in noticeable symptoms), to that between the impact of an accident in a quiet London side street and one that closes Tower Bridge.

The pathology of MS involves white blood cells, as they cross the blood brain barrier, misidentifying myelin as foreign and attacking it, with repeated attacks over time causing a gradual accumulation of nerve damage and consequent dysfunction. Following a first attack, a patient is likely to continue having further attacks on average 0.8 times per year (although the intervals vary widely and unpredictably between individuals). The body repairs nerve damage to a greater or, subsequently, lesser extent following each attack, but over time the damage becomes permanent. Dr Silber likened this process to darning a jumper affected by moths, which can allow you to continue wearing it until holes start fraying at the edges and can no longer be repaired.

MS is a clinical disease that cannot be diagnosed on the basis of a scan alone in the absence of an attack. Sclerosis means scarring, and multiple sclerosis refers to the disease’s distinctive pattern of damage that is disseminated both in space and time, with lesions affecting different areas of the central nervous system at different times and hence resulting in a variety of physical and mental symptoms. There is no absolute test for MS. Many of those who think they have it do not, and many people have it without realising.

The fact that MS is so hard to diagnose creates difficulties for GPs, neurologists and, in particular, their patients, who often have to live with debilitating uncertainty and worry for
many years. Dr Silber outlined the case history of one female patient who suspected she might have MS, as her mother had previously, and was told in the 1970s on the basis of normal scans but a slightly unusual visual evoked potentials (VEP) score that she might have ‘benign’ MS. Subsequently, after the disease had not developed over 20 years and her scans continued normal, she was told fairly categorically that she did not have MS. Her response was that the medical profession had ruined her life, which she had effectively put on hold all that time.

Clearly there is a danger of over-diagnosis, but equally many people who have been told ‘we are not sure’ go on to develop MS – sometimes very suddenly. Others who may appear to be at a very early stage, can see their conditions deteriorate suddenly and dramatically, as with one woman who was told she had ‘benign’ MS (a term Dr Silber said he prefers not to use) but then went from running half marathons to barely walking in the space of six months. The earlier MS can be identified, the better the chances of intervening effectively to delay its progress and keep patients functioning at a higher level for longer.

These diagnostic challenges give added significance to the critical diagnostic criteria applied. A detailed account of how these criteria have developed over time was provided since the formulation of the Schumacher Criteria of 1965, on which all subsequent criteria continue to be founded.

Discussion moved onto the following:

- Poser Criteria of 1982, which introduced para-clinical tests as an aid to diagnosis
- The Barkhof MRI Criteria, where Dr Silber outlined the use and interpretive significance of oligoclonal bands in cerebrospinal fluid from a lumbar puncture (which provide evidence of the production of antibodies on the CNS side of the blood brain barrier)
- visual evoked potentials (which can identify asymptomatic damage to the optic nerves and hence dissemination in space)
- enhanced and non-enhancing lesions on MRI scans (which distinguish between old and new scarring of white matter and hence help establish dissemination in time)

The McDonald Criteria of 2000, revised in 2005, introduced greater sensitivity by abandoning the insistence in previous definitions on an interval of three months between scans detecting new lesions and allowing diagnosis based on a single scan showing the simultaneous presence of asymptomatic lesions at any time. Dr Silber illustrated this sharing MRI evidence of a person suffering optic neuritis who also shows enhancing lesions in unrelated areas of the brain, indicating (asymptomatic) distribution in time.

Research indicates that those with a clean scan (aside from evidence of that first attack) have an average 80% chance of having no further attacks within 15 years. Those whose scans show evidence of previous scarring have only a 20% chance of escaping further attacks over the same period. The more widespread the scarring, the greater the risk of disability. A clinically isolated attack with evidence of previous scarring is effectively the same as having RR MS.

Further observations were illustrated on the various stages in disease progression, depicting scans and other test results and potted case histories of some of the individuals concerned. Around half of those in whose brains MRI-like lesions are detected without having experienced an attack will go on to develop further lesions over the next five years and a smaller proportion to experience a clinical event. I tell such people, Dr Silber said, “that their overall risk is pretty low, arguing that evidence of an RIS should not be construed as sufficient grounds for not offering a person insurance.” (See Graph 1)

A range of conditions can be mistaken for MS. Other inflammatory diseases like Lupus can result in inflammation of the blood vessels, manifesting in hard signs and making a person systematically unwell in a way that can look like MS. Devic’s Disease (Neuromyelitis Optica) causes lesions exclusively in the optic nerve and spinal cord. Although Devic’s can be considered part of the MS spectrum, it is a more serious condition, causing very large spinal cord lesions, with patients recovering poorly from relapses. There is a specific blood test for Devic’s. Minor strokes can also manifest in a flurry of attacks and recoveries that could suggest MS.

Others report a multitude of symptoms, both physical and cognitive, but test normal and are likely to have problems that are predominantly psychogenic in nature. These people, Dr Silber stressed, are often genuinely disabled by their conditions.
and can benefit from neuropsychiatric input – although funding for this is hard to get within the NHS today.

There are also some degenerative diseases whose symptoms can resemble MS, including cervical degeneration and Hereditary Spastic Paraplegia. Another possible source of misidentification is B12 deficiency, which is curable if caught early.

Early symptoms of MS include weakness, spasticity and Ataxia in the legs, weakness, incoordination and tremor in the arms, visual problems, difficulty speaking or swallowing, sexual, bowel and bladder problems, altered sensation and pain in many different regions, depression, anxiety and cognitive problems, as well as a fatigue that can be utterly debilitating.

The management of MS was discussed, beginning with helping patients through the succession or combination of classic Kübler-Ross Grief Cycle responses (denial, anger, depression, bargaining, acceptance) that typically follow a diagnosis of MS. Dr Silber said “he favoured oral steroids as the best means of speeding recovery after an attack, as this can avoid keeping patients in hospital.” He also examined the role of Botox in symptom management and of Cannabis derivative Sativex in patients with more severe disability and spasticity, describing the encouraging results of current trials.

In terms of disease modifying therapies that can potentially prevent further relapses, there is a window of opportunity for treating MS. Referring to the Expanded Disability Status Scale (EDSS), which runs from 0 (normal) to 10 (death from MS), it was noted the results of an Italian study that looked at patients with Relapsing-Remitting MS and Primary Progressive MS over a ten year period and found that around 50% of those with RR MS had reached EDSS 4.0 (at which point they will still be able to walk 500 metres unaided, despite relatively severe disability) compared with virtually 100% of those with PP MS. Beyond EDSS 4.0, however, patients with RR MS and PP MS deteriorate at the same rate. At that point the window has been missed, and ‘the moths’ are in control.

Given the huge array of disease modifying drugs currently available or in the pipeline: how do you choose? Mapping the more common drugs graphically against X and Y axes for safety
and efficacy, Dr Silber discussed the relative merits of first line therapies such as Copaxone and Beta Interferons as well as more aggressive drugs including Fingolimod, Natalizumab and Alemtuzumab. Natalizumab (aka Tysabri), he said is the current gold standard, reducing attacks by around two thirds, but increasing the risk of contracting an often fatal infection called Progressive Multifocal Leukoencephalopathy (PML) caused by the JC virus, which is present in roughly 50% of the population. A detailed decision-making flow diagram, can be used to discuss patients’ treatment options with them. (See Graph 2)

A new drug, BG12, currently in development has the potential to be a game changer, reducing attacks by around 50% whilst currently promising to pose a very low risk of negative side effects. The manufacturers have applied for a first line licence at a low price in a bid to capture the first line market.

These drugs can have a powerful effect in reducing attacks. But, quoting Spiderman, Dr Silber stressed that with great power comes great responsibility. We want to offer our patients treatments. We want to give them as much information as we can about their disease to help them make informed decisions, and we want to stop their diseases becoming worse, he said. But we also need to be aware that the judgements we make can have profound consequences.

If you would like further information on any of the topics covered here, please contact uk.marketing@hannover-re.com

**Sources:** Dr Eli Silber www.silberneurology.com
HR UK Medical Officer Dr Emile de Sousa

---

**Graph 2: Overview of MS therapies: A double edged sword**
LUCID 2013

The Grand Hotel Brighton was the venue for the third LUCID (Life Underwriters, Claims and Insurance Doctors) conference. LUCID 2013 was the culmination of almost two years’ hard work by the organising committee, collaboration between the four underwriting and claims clubs operating in the UK. These are The Assurance Medical and Underwriting Society (AMUS), Forum of Claims & Underwriting Scotland (FOCUS), the Health Claims Forum (HCF) and Select 74.

The interdisciplinary conference held from 29 September to 2 October 2013 provided more than 250 delegates, including underwriting, claims assessors, doctors and third party providers the opportunity to discuss and debate new developments relevant to those working within the life and living benefits industry.

Hannover Re UK Life Branch’s (HR UK) Caroline Froude has been a committee member of LUCID since its inception in 2007 and is immensely proud of the work the committee has done in providing three intensive days of lectures and breakout sessions. The mission set by Chairman Simon Grant for this conference, was “to provide a unique opportunity for interdisciplinary discussion and debate.” We certainly believe this was achieved.

A great deal of thought and preparation went into the programme. This was reflected by the impressive speaker list which included experts and luminaries from outside of our industry ensuring a conference that was educational, stimulating, value for money and fun.
Each day was split between plenary lectures in the main auditorium during the morning and breakout sessions in the afternoon.

The event began with an evening reception held amongst the exhibitor stands in the exhibition space on Sunday evening of which HR UK were one of the sponsors and exhibitors. Then, bright and early on Monday morning, the Chairman formally welcomed delegates, speakers, sponsors and exhibitors to LUCID 2013 before handing over to the first speaker, BBC presenter, commentator and reporter Russell Fuller.

On Monday afternoon delegates were delighted to welcome Tony O’Leary to the stage, to give the R D C Brackenridge Memorial Lecture. Tony looked back at Brack’s legacy and at how we can best carry forward the work he began. A pioneer of medical underwriting, Dr RDC Brackenridge laid the foundations for the evidence-based approach to risk assessment and his 1962 book, The Medical Aspects of Life Assurance now in its 5th edition, is seen as the definitive text on the subject.

Over the two and a half days that followed, delegates enjoyed presentations from 15 different speakers, covering a huge range of topics including: Liver Fibrosis, Impaired Annuities, a European view of insurance legislation, Stem Cell Science, the pros and cons of medical screening, Bar Coding Cancer, Multiple Sclerosis, and Adult Congenital Heart Disease - to name but a few.

The afternoon breakout sessions were run by various partners in LUCID, of whom one, of course, was HR UK. Jordi Posthumus, Vice President - Longevity Trading at Hannover Life Re Bermuda, ran a thought provoking session on Insurance and the Rise of Evolutionary Medicine, a summary of which appears in this edition of In Focus.

Other breakout sessions challenged delegates to re-think medical screening, the future of cancer diagnosis, does underwriting top athletes mean top risk and unforeseen claims?, honesty – for the best policies, are you fit4business, rebuilding the foundations; insights into developments in medical care in the UK and to think differently! – ‘we cannot solve our problems with the same thinking we used when we created them’.

On the last day the sponsored charity Help for Heroes gave a session by representative Hayley Humble, from Tedworth House in Wiltshire, one of four Recovery Centres run by the charity. Hayley spoke with passion about the fantastic work they do providing 360 degree holistic care to wounded soldiers on either a residential or day care basis. She was joined on stage by one of the “band of brothers” who was injured in Afghanistan and treated at Tedworth House. Throughout the conference nearly £2,000 was raised for this fabulous cause.

Over the course of LUCID 2013 delegates had learned a great deal about current protection topics of interest, and enjoyed many fascinating discussions with colleagues and peers. It wasn’t all hard work, of course. At the Conference Dinner, held on Tuesday at The Corn Exchange, live karaoke band Rockaoke invited the audience to “live the dream” and perform with a live rock band. An invitation many delegates could not resist to join the band and enjoy an evening of singing and dancing.

HR UK ran a ‘Guess the Calories’ competition throughout the conference, Graham Hull, Friends Life was the overall winner taking home a Kindle Fire HD and Amazon vouchers. The three runners up were Trisha Doyle, Caledonian Life, Jayne Maher, Wesleyan and Carley Simpson, Swiss Re Life & Health who each received Amazon vouchers and the Rosedale Diet Book.

Caroline Froude
Chief Underwriter and Head of Claims

Contact
uk.marketing@hannover-re.com

www.hannoverlifere.co.uk