Dementia – Not Just a Bad Memory

by Dr. Chris Ball, Gen Re, London

The one thing you cannot forget right now is Dementia. From the very real concerns over the care given in our institutions to the most abstruse research findings, dementia continues to make the headlines in the national press. This attention is certainly welcome after many years of relative neglect but the question is very much “Why now”? Perhaps the main answer to the question is that people are living longer. The International Longevity Centre reports that in 1950 male life expectancy at the age of 60 was 11 years, and over the subsequent half century this has doubled to 22 years. The Centre also predicts that this trend will continue and by 2050 it will have extended to 26 years.1

Another way of looking at this is life expectancy (LE) at birth. In the UK this is now 77.1 for men and 81.9 for women. Healthy life expectancy (HLE) is also growing; at birth this is 63.5 for men and 65.7 for women. Whilst HLE is slower growing than LE, the bar is set very low for being classified as unhealthy. These increases come about from a combination of improvements, mainly in lifestyle but also in medical care. The growth in the population over pensionable age has led to increasing concerns about how the state will support these individuals, or how they will support themselves, until their deaths. In this time of financial austerity the issue has become even more acute.

Dementia growing?

Ageing is the greatest risk factor for the development of dementia. Table 1 shows the prevalence of dementia for different age groups across Europe. Whilst the prevalence is relatively low between age 65 and 74, there is an exponential rise thereafter.

Table 1 – Total age specific prevalence rates Europe²

<table>
<thead>
<tr>
<th>Age range</th>
<th>Male prevalence %</th>
<th>Female prevalence %</th>
<th>Total prevalence %</th>
</tr>
</thead>
<tbody>
<tr>
<td>60-64</td>
<td>0.2</td>
<td>0.9</td>
<td>0.6</td>
</tr>
<tr>
<td>65-69</td>
<td>1.8</td>
<td>1.4</td>
<td>1.6</td>
</tr>
<tr>
<td>70-74</td>
<td>3.2</td>
<td>3.8</td>
<td>3.5</td>
</tr>
<tr>
<td>75-79</td>
<td>7.0</td>
<td>7.6</td>
<td>7.4</td>
</tr>
<tr>
<td>80-84</td>
<td>14.5</td>
<td>16.4</td>
<td>15.7</td>
</tr>
<tr>
<td>85-89</td>
<td>20.9</td>
<td>28.5</td>
<td>26.2</td>
</tr>
<tr>
<td>90-94</td>
<td>29.2</td>
<td>44.4</td>
<td>41.0</td>
</tr>
<tr>
<td>&gt;95</td>
<td>32.4</td>
<td>48.8</td>
<td>46.3</td>
</tr>
</tbody>
</table>

Source: ec.europa.eu

About This Newsletter

Risk Insights is a technical publication produced four times a year by Gen Re for life and health insurance executives worldwide. Articles focus on actuarial, underwriting, claims, medical and risk management issues. Products receiving emphasis include life, health, disability income, long term care and critical illness insurance.
The number of individuals surviving into these greater ages (where the prevalence is high) is relatively small, but growing. Currently for example, approximately 10,000 centenarians live in the UK, but this number is projected to rise to more than 250,000 by 2050.

In 2010 an estimated 35 million people worldwide had dementia and associated annual costs were US$604 billion. The expectation is that numbers will exceed 115 million by 2050. This assumes the incidence will remain constant; thus, the projected rise in the elderly population will bring a concomitant rise in the number with dementia. It was suggested in 2011 that the long-term care needs for older people present the “single largest global threat in the 21st century”.

Changes in population structure mean there will be relatively fewer young people to support the growing “burden” of elderly people – with or without dementia. This is commonly expressed as the “Dependency Ratio,” in which the proportion of those of pensionable age is contrasted with those of working age. This ratio is fuelled by a decreasing birth rate and the disintegration of traditional family models of care. Dependency ratios from Ireland (see Table 2), which is currently in a fairly healthy situation with a young workforce, serve to illustrate the point.

As the balance shifts, Ireland will be moving toward a situation that is becoming the norm in other parts of the world. In the EU the old-age dependency ratio based on current trends will almost double in 50 years from 24.1% to 47.2%.

Dementia slowing?
The apocalyptic vision of a Brobdingnagian society barely able to support people who no longer have any economic utility has not been embraced by everyone. After posing the question, “Is the prevalence of dementia changing?” one commentator noted robust clinical evidence of fewer cases of dementia than were predicted developing later in life. Manton et al. (2005) report a decline in prevalence of dementia in over-65s in the U.S. from 5.7% in 1982 to 2.7% in 1999. Langa et al. (2008) report a reduction between 1993 and 2002 in cognitive impairment in the over-70s of 3.5%. Mortality rates rose over time for individuals who developed moderate to severe disease, suggesting that although more recent cohorts are healthier and have a greater cognitive reserve, they decline faster once they do develop dementia.

The Rotterdam studies found a decrease in the age-adjusted incidence of dementia when comparing a cohort recruited in 1990 and a second recruited ten years later. The latter cohort showed decreased mortality rates, less brain atrophy and less cerebrovascular disease. Two studies using broadly similar methods have examined cohorts recruited 18 years apart. The first, conducted with over-65s between 1989 and 1994, predicted there would be 884,000 people with dementia in the UK in 2011. However, the second study, recruiting subjects between 2008 and 2011, suggested that there would be only 670,000 – a 24% decrease on the earlier estimate. Putting aside any methodological issues that may bias these findings, the results remain persuasive. Further weight is added by the study of cohorts aged over 90 in Denmark. The first cohort was born in 1905 and assessed at age 93. The second, born in 1915, were aged 95 at the time of assessment. Despite being older, the second group achieved significantly higher scores on the Mini-Mental State Examination (MMSE), a brief cognitive scoring instrument, with 23% versus 13% scoring between 28 and 30, i.e., within the normal range. The possibility that these reductions are a result of much improved cardiovascular health has some support, but with the rise in obesity and diabetes this trend could be reversed in the future.

The second challenge to the panic over dementia argues that the methods of calculating the old age dependency ratio are too simplistic. This argument claims that the calculation fails to take account of the realities of the economic situation and the fact that not everyone over age 65 is dependent; in fact this cohort is fitter, healthier and possesses greater financial muscle than ever before. Many continue to work either in paid employment or volunteer work, such as looking after

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Table 2 – Dependency ratios, Ireland

<table>
<thead>
<tr>
<th>Year</th>
<th>2006</th>
<th>2011</th>
<th>2016</th>
<th>2021</th>
<th>2026</th>
<th>2031</th>
<th>2036</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ratio</td>
<td>16.4%</td>
<td>17.6%</td>
<td>19.9%</td>
<td>22.4%</td>
<td>25.2%</td>
<td>28.5%</td>
<td>32.1%</td>
</tr>
</tbody>
</table>

grandchildren. Most financial costs associated with ageing occur in the last few months of life regardless of the age when this happens. By counting only those who are actually working, it would appear that more dependents of working age exist than elderly ones.

If one accepts this argument, then the dependency ratio in many countries is declining rather than rising, meaning the costs of old age are significantly over-estimated. Although age-specific disability rates have been falling, the authors also draw attention to the cohorts, which appear to have a worse risk profile, particularly obesity with its associated cardiovascular disease risk.

**Dementia confusion?**

The terminology surrounding dementia is often misunderstood both by the laity and in the press, where standards should be higher. Dementia is an umbrella term that describes a syndrome that consists of acquired (i.e.; you are not born with it), progressive (to distinguish from the static damage from head injury or stroke), chronic (to distinguish from the acute cognitive problems resulting from infection or other process), and impairment in a number of cognitive processes rather than memory alone. There is a very long list of possible causes for these problems but the most common is AD.

It is important to note that dementia is not merely a disorder of memory. In some dementias, profound impairment in other areas may not be accompanied by a similar decrement in memory (e.g., semantic dementia). Despite this, most clinical services that are established for the early diagnosis and management of dementia are called “Memory Services”, many of these services in the UK being established on the back of the government’s “Dementia” Strategy. The fear that the “D” word invokes means that people are still happier to have their “memories” assessed than undergo exploration for possible “dementia”. The Royal College of Psychiatrists still brings these services together under the umbrella Memory Services National Accreditation Programme.

For many, the term dementia and AD appear synonymous. This is not surprising given that AD is the most common form of the disease with vascular dementia the second commonest form. Many people have dementia as a result of a combination of both pathologies, so called “mixed dementia”.

Burns et al. (1995) summarised the symptoms of dementia using five “A”s:

- **Amnesia** – Problems with memory, initially with new learning but loss of older memories as the disease progresses.
- **Agnosia** – Inability to recognise objects or people although the specific sense is not defective. As a result, sufferers can become lost or not recognise familiar people.
- **Apraxia** – Difficulty in completing learned movements. This results in difficulty dressing and completing day-to-day tasks.
- **Aphasia** – Language problems, such as finding the right word, being able to express oneself or understand what is being said.
- **Associated Symptoms** – A range of problems, such as depression, hallucinations, wandering and other behavioural difficulties.

The different forms of dementia have different symptom profiles. In AD the brain changes that contribute to the development of these symptoms are the accumulation of an abnormal protein (beta-amyloid) in plaques outside of the brain cells and a second abnormal protein (tau) as tangles within the cells. These processes interfere with the cells’ ability to communicate and eventually cause cell death. It is likely that these changes are present in the brain many years before the onset of clinical symptoms. Finding acceptable biological tests that identify these changes is the focus of much current research effort. Other forms of dementia have different mechanisms of destroying the communications between cells and causing cell death.

**Dementia treatment**

Currently, two classes of drugs are licensed to treat AD in the UK: the Acetylcholine Esterase Inhibitors (ACIs) Donepezil, Galantamine and Rivastigmine, and an NMDA receptor antagonist (Memantine). The ACIs stop the breakdown of a chemical messenger named choline while Memantine blocks the messenger glutamate. Neither class of drug strikes at the fundamental pathological processes of AD and so cannot be regarded as curative in any sense.
In their 2010 review Casey et al. concluded that the ACIs show a small initial gain in cognitive function that drops back to baseline in six to nine months with some modest value in continuing the medication after this point. More recent work demonstrates the value of continuing the medication until death. These cohort benefits are small, but looking at individual responses only about half of people show this improvement whilst a small proportion show a dramatic response.

In the author’s clinical experience, the changes reported in the research arena do not always capture the benefits of the medication that are seen in day-to-day clinical practice. Four of the five studies addressing nursing-home placement showed a delay for those who are taking medication. The use of Memantine is less well characterized but shows some benefits in middle to late stage disease.

Too often, however, there is little to offer medically for the dementia sufferer who has something other than AD. For those with vascular dementia caused by problems with the blood supply to the brain, ensuring that the vascular risk is well-managed is vital but there is no specific treatment. Nor is there any treatment for many other forms of the syndrome, although dementia with Lewy bodies (DLB), the cause of around 10% of dementia, can respond well to ACIs.

To take a purely medical model for treatment is a very limited approach, as it is for any chronic progressive disease. The importance of emotional support, planning and help for carers cannot be underestimated. The majority of care for this group remains in the hands of family and friends, often at great personal cost. Interventions to support carers have demonstrated significant value for the person with dementia and the carer, and in the savings to the overall care bill.

**Dementia diagnosis**

It is important that the efficacy of treatments is demonstrated in clinical trials, but this becomes academic if those treatments cannot be delivered to those with the disease. However, studies from high income countries report that only between 20% and 50% of all cases of dementia are even recorded in the primary care records, the median being 39%.

Barriers to the early diagnosis from both the patient and carer perspective have been identified as:

- Lack of knowledge about the closeness of AD symptoms to ageing
- Gradual manifestation and inability to treat problems as acute and serious
- Fear and a denial of the disease and its implications
- Lack of trust in the health system
- Lack of support from family, friends and professionals

The time taken for the diagnosis of dementia to be made after symptoms are noticed varies significantly from country to country (see Table 3), and depends not only on the attitudes of the person with dementia and their family but also on the health structures available to access help.

<table>
<thead>
<tr>
<th>Country</th>
<th>Months from onset to diagnosis</th>
</tr>
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<tbody>
<tr>
<td>UK</td>
<td>32</td>
</tr>
<tr>
<td>France</td>
<td>24</td>
</tr>
<tr>
<td>Poland</td>
<td>23</td>
</tr>
<tr>
<td>Spain</td>
<td>18</td>
</tr>
<tr>
<td>Italy</td>
<td>14</td>
</tr>
<tr>
<td>Germany</td>
<td>10</td>
</tr>
<tr>
<td>Mean</td>
<td>20</td>
</tr>
</tbody>
</table>

Source: Adapted from Bradford A, et al. 19

To date, population screening for dementia has not been considered cost-effective in any country, but some recommend that opportunistic screening should take place. Increasingly, there has been a push for non-specialists to make the diagnosis and initiate treatment to improve the outcome for this group. The effectiveness of educational programs in primary care remains unanswered but the current drive of diagnosis-for-diagnosis-sake is not always beneficial for the patient, particularly late in the disease or in the presence of significant physical pathology.

The “bedside” cognitive screening tests available to clinicians are often sensitive to the diagnosis but not specific, leading to a high false positive rate. The quest to develop a quick, acceptable, cheap and reliable dementia biomarker continues. Press reports of breakthroughs are frequent but often overblown; biomarkers usually identify only AD
and are often reported in populations with high prevalence of this disease. A full evaluation of biomarkers as a screening or predictive tool in the general population is still to emerge.

There is no substitute for careful history-taking (including from an informant), coupled with physical examination, cognitive assessment and investigations such as neuroimaging. This is both a time-consuming and expensive process; however, taking time to make the diagnosis of a progressive and incurable disease is of the utmost importance.

**Dementia implications**

The stark truth is that since the introduction of Memantine, originally synthesised in 1968 but only approved for use in 2002, and Donepezil (1996), no new treatments have become available to treat AD or indeed any of the other forms of dementia.21 In 2013 research highlights in dementia showed a heavy bias towards AD. It is now thought likely the brain changes of AD begin decades before the onset of clinical symptoms. This means that identifying biomarkers in at-risk people would be a major first step to developing significant disease altering treatments.

Progress has been made in neuroimaging, genetics and blood tests; these are still a long way from clinical utility but significantly improve the researcher’s ability to measure the outcomes of their interventions. The results of clinical trials have been largely disappointing but the message is increasingly that interventions need to be given in the preclinical stage to be effective.

The importance of research into the condition cannot be understated. An intervention to delay the onset of dementia by five years has been estimated to halve the number of deaths, saving 30,000 lives a year. Significant strides have been made to bring together the disparate research entities across Europe with the hope that significant benefits will soon be translatable from the laboratory to routine clinical practice.22 This was a much-needed intervention since, in late 2012, the Alzheimer’s Society vented their frustration that “There are 150 times more clinical trials focusing on treating people in the late stages of cancer than Alzheimer’s disease”.23

Significant advances in dementia diagnosis and treatment could impact annuity writers if improved treatments increase life expectancy materially. Other life insurers are unlikely to see a notable change in experience because term assurance makes up the bulk of their portfolios, and these benefits typically cease by age 70. Screening, as it might be done today, is unlikely to be recommended before people reach their 60s, given the low dementia prevalence at younger ages. Should techniques become available to diagnose AD before symptoms are evident then this might change.

Critical Illness policies require both a definite diagnosis and severe levels of impairment for AD and other forms of dementia. Screening is unlikely to increase claim rates, as it accelerates only the diagnosis, while subsequent interventions may delay impairment. Whilst this suggests there may be no impact in the short term, increasing awareness of the illness – and the implications of funding care late in life – means that there are opportunities for the development of products that will not only provide a stable income in old age but also provide flexibility to meet care needs in the event of a diagnosis of dementia.

**About the Author**

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Endnotes
1  www.ilcuk.org.uk/.
3  “The Future Economic, Health and Social Care Costs of Dementia” International Longevity Centre (UK) and the Actuarial Profession joint debates (2011).
14  www.nice.org.uk/TA217.
20  Brunet, M. “New dementia diagnosis targets will lead to overdiagnosis”, BMJ, 2014; 348 (apr01 2): g2224 DOI: 10.1136/bmj.g2224
22  http://www.mrc.ac.uk/Newpublications/News/ MRC006232.
Breathing Easier – Advances in Asthma
by Dr. John A. O’Brien, Gen Re, Cape Town

In all fields of science and medicine, there is an explosion of new information. When contemplating what is new in asthma the temptation is to focus only on new developments – information regarding pathways of inflammation or potential products that may modify the asthma disease process. However, as recent years have seen few new treatments become available, it is more beneficial to focus on the progress in understanding asthma and how the treatment has changed with time. Despite the availability of effective treatments, the control of many individuals’ asthmatic condition remains sub-optimal, and this has a significant impact on their school and workplace attendance and performance.

What is asthma?

“Asthma is one of the most common chronic diseases, with an estimated 300 million individuals affected worldwide. Its prevalence is increasing, especially among children” according to the Global Initiative for Asthma (GINA), which was launched in 1993 in collaboration with the National Heart, Lung, and Blood Institute, National institutes of Health, and the World Health Organization. This major initiative is being undertaken to improve knowledge about and the standards of care of asthma and to produce guidelines for clinicians and information for patients.

Global initiative for asthma – definition of asthma, May 2014

Asthma is a heterogeneous disease, usually characterised by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation.


The typical young asthmatic presents with episodes of bronchospasm and complains of wheeze, tight chest, difficulty breathing and cough. There is no question that spasm of the smooth muscle in the airway is a major cause of these symptoms, but it is crucial to realize that this is secondary to airway inflammation.

How large is the problem?

GINA commissioned the Global Burden of Asthma report, published in 2004, which estimated that approximately 300 million people worldwide have asthma. In 2001 asthma was ranked the 25th leading cause of disability-adjusted life years (DALYs), representing the sum of years of potential life lost due to premature mortality and the years of productive life lost due to disability. It is now estimated that asthma accounts for 15 million lost DALYs per year – a level similar to diabetes, cirrhosis of the liver or schizophrenia.

Asthma accounts for approximately one in every 250 deaths worldwide. Therefore, with the development in the understanding of the pathogenesis of asthma and the role of the available medications came the realization that asthma appeared to be becoming more common and possibly more severe. Explanations for this have been sought, and the numerous theories include increased urbanization.

With the proportion of the world’s population that is urban projected to increase from 45% today to 59% in 2025, there is expected to be a marked increase in the number of asthmatics. It has been projected that by this date there may be an additional 100 million. Other causal factors include pollution, obesity and smoking although current theory suggests that different factors may be responsible in different social environments. For example, in Eastern European countries infection, diet and pollution have been identified as contributing factors while in the U.S. inner city population poverty, allergens and stress may all contribute.

In affluent societies potential triggers and aggravators include smoking, air pollution and obesity, family size and even children’s day-care facilities. It has long been known that obese asthmatics are more difficult to treat; conventionally this has been ascribed to increased thoracic and
abdominal fat causing pressure on the airways. There is now evidence that the adipose tissue itself is metabolically active and produces mediators that act on the airways. Obesity not only complicates asthma but can be causal.1

**Treatment of asthma**

Treatments in the first part of the 20th century focused on medications designed to relieve airway smooth muscle spasm. Some early suggestions for treatment look surprising today as they included smoking medicated “anti-asthma” cigarettes and inhaling the fumes of combustible powders with such ingredients as tobacco, potash and plants.2 The presence of inflammation in the airway of asthmatics has been recognised for many years. With the development of oral corticosteroids, many asthmatics were placed on cortisone. As a powerful anti-inflammatory this worked well to control asthma and emphasised the importance of airway inflammation in the pathogenesis or cause of asthma. However, the improved control came at a price, as systemic corticosteroid has a number of predictable side effects, including skin fragility, weight gain, osteoporosis, glucose intolerance and a number of others.

In the early 1970s inhaled corticosteroids first became available, offering significant advantages. The corticosteroid dosage was now measured in micrograms (µg) and could be delivered directly to the site of inflammation in the airways. These new products dramatically reduced the need for asthmatic patients to receive oral steroid therapy. Side effects from the inhaled steroids are minimal. Inhaled steroids were first introduced with a low dosage of 50 µg requiring users to take two puffs four times a day. Adherence to this type of regime was clearly difficult and the next advance was the development of higher dose preparations – inhalers of 100 µg and 250 µg with use frequency reduced to twice daily, which is quite acceptable to most patients.

Apart from the inhaled steroids, few medications were available. Patients continued to rely on short-acting beta₂-agonist bronchodilators and there was widespread use of the oral theophyllins. The latter medication is used far less frequently today due to the narrow therapeutic balance between its beneficial effects and the side-effects. It is held that

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**Bad Air Day – The Impact of Pollution on Asthma**

*by Ross Campbell, Gen Re, London*

We inhale approximately 20,000 times each day, averaging 50 litres of air each minute while running, 25 litres walking, and almost 10 litres just sitting down.1 Harmful substances in the air we breathe can damage the airways and cause short-term and long-term health impacts.

Air pollution is the release of natural or man-made particles and noxious gases into the air, much of which is a result of the combustion of fossil fuels. Air pollution poses a serious threat of lung disease, including lung cancer, and it may worsen existing asthma, bronchitis and emphysema. It can cause heart and circulatory problems and is linked to increased cancer of the urinary tract and bladder.

Ambient (outdoor) pollution is estimated to cause 3.7 million premature deaths worldwide per year.2 The World Health Organisation (WHO) estimates that 80% of ambient pollution-related premature death results from ischaemic heart disease and stroke, that chronic obstructive pulmonary disease and acute lower respiratory infections account for 14% and lung cancer is the cause of 6%.

Air quality is typically described using particulate matter (PM) concentrations. PM is a mix of solid and liquid particles of organic and inorganic substances including sulphate, nitrates, ammonia, sodium chloride, black carbon, mineral dust, sand, pollen and water that is suspended in the air. PM varies in origin, shape, size, composition, and from place to place and time to time.

Small particles are defined as 10 microns (PM₁₀) or 2.5 microns (PM₂.₅) in diameter.1 The smallest can lodge deep inside the lungs. Chronic exposure causes inflammation and a worsening of heart and lung diseases. It is linked to deaths from cancer, cardiovascular and respiratory disease.4 The health impact of small PM pollution is greater than any other pollutant even at very low concentrations – indeed no safe level exists. The WHO estimates that PM alone causes 800,000 (20%) of all pollution-related, premature deaths every year.

The other main pollutants of concern are nitrogen dioxide, ground level ozone, sulphur dioxide, carbon monoxide, hydrocarbons and lead. Nitrogen dioxide inflames the lining of the lungs, causing shortness of breath and cough, and it acts to reduce immunity to infection and bronchitis. The effect is worse on people with asthma. Nitrogen dioxide contributes to the formation of
particles and reacts with hydrocarbons in the presence of sunlight to create ground-level ozone. High levels of ozone can narrow the airways and inflame the lungs. For asthmatics, ozone pollution episodes can trigger attacks or generally worsen the condition. European studies have reported a rise in daily mortality of 0.3% per 10 µg/m³ increase in ozone exposure. Sulphur dioxide gases irritate the airways of the lungs increasing symptoms for people who have lung disease. Hospital admissions for cardiac disease and mortality increase on days with higher sulphur dioxide levels. Carbon monoxide gas impairs the uptake of oxygen by the blood and can negatively affect heart function.

The weather plays a part; harmful levels of pollution can build on calm days that are either very cold or very hot. Temperature inversion traps pollutants below a band of cooler air to form “winter smog”, exacerbating lung disease due to the combination of cold and poor air quality. A “summer smog”, when nitrogen dioxide forms a cocktail of ozone and PM, may create a pollution episode, putting asthmatics at particular risk. Estimations of the threat to health are based on the concentration of pollutants in the air, the time spent in the polluted environment and the amount of air breathed. The long-term health effects of air pollution are thought to be the most significant. These include slowly developing conditions such as cancer, COPD and heart disease. These stem from exposure to lower pollution levels than result in short-term health effects (e.g., asthma attacks) and to which people are unwittingly exposed over time. It is unclear if air pollution is a fundamental cause of asthma but people with severe disease are likely to be affected by it as they have lower reserves of lung function. The main effect is observed in the elderly with chronic lung disease, which includes asthma.

Exposure to high concentrations of small particulates (PM₁₀ and PM₂.₅) is linked with increased mortality or morbidity, both daily and over time. Reduced exposure to air pollution will increase life expectancy and total years lived in a population. In the UK, for example, removing man-made PM2.₅ would increase life expectancy from birth by six months, according to estimates. This net gain is similar to what would be realized if there was no more infectious or parasitic disease. To put it in further perspective, eradicating all cancer would increase life expectancy by only around three years, a phenomenon known as the taeuber paradox. Nevertheless, air pollution is a major environmental risk to health. By reducing air pollution levels, countries can reduce the burden of disease from stroke, heart disease, lung cancer, and both chronic and acute respiratory diseases including asthma.

Endnotes
2 http://www.who.int/mediacentre/factsheets/fs313/en/.
3 PM₂.₅ is defined as the mass per cubic meter of airborne particles passing through the inlet of a size selective sampler with a transmission efficiency of 50% at an aerodynamic diameter of 2.₅ µm. In practice, PM₂.₅ represents the mass concentration of all particles of less than 2.₅ µm aerodynamic diameter.
5 http://uk-air.defra.gov.uk/air-pollution/effects.
6 Ibid.
9 http://www.comeap.org.uk/.
10 The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom (2010), Committee on the Medical Effects of Air Pollutants, available at www.comeap.org.uk.

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they act as bronchodilators but perhaps do have mild anti-inflammatory effects as well.

The next major advance was the development of long-acting inhaled beta₂-agonist bronchodilators – medications that work to relax the muscles surrounding the airways – but the development of these drugs was dogged by safety concerns. In the late 1970s and early 1980s there were epidemics of asthma deaths and analysis pointed to a link with beta₂-agonist treatment. It was not clear initially if the product itself was toxic or whether an excess use of bronchodilators was itself a marker of asthma severity, meaning the deaths were the result of the poor assessment by clinicians and poor management by patients.

This controversy still exists although it is now generally accepted that both types of beta₂-agonists – short-acting and long-acting – are safe if used in conjunction with inhaled corticosteroids. To emphasise this point the U.S. Food and Drug Administration marks long-acting beta₂-agonists with a “black box” warning that indicates they should not be used alone for the treatment of asthma.

How successful is the treatment?

The frightening information regarding asthma mortality triggered an era of international asthma education with the intent of ensuring that patients and care givers had a better understanding of the disease and were appropriately informed and counselled so that they could react appropriately if there were any deterioration in patients’ conditions. The standard of treatment in the 1970s and 1980s was increased doses of inhaled steroid driven by symptom severity. Indeed, this proved effective and with robust endpoints, such as asthma deaths, correlation could be drawn between the number of canisters of inhaled steroids used in a year and the risk of asthma.

However, not all patients achieved optimal control using inhaled steroid alone, prompting the question whether it was better to continue to increase the dose of inhaled steroid or to add additional treatment, in particular the relatively new class of agents, long-acting beta₂-agonist inhalers. A number of studies were performed which overwhelmingly confirmed the fact that adding a long acting beta₂ agonist to an inhaled steroid was a more effective intervention than simply increasing the dose of inhaled steroid.³

Because this blend of medication was so effective, the large pharmaceutical companies began to produce combination inhalers designed for use just twice a day, a development that revolutionised asthma treatment allowing the vast majority of asthmatics to become well controlled.

Around this time a number of worldwide studies indicated that the general standard of asthma control was disappointingly poor despite the apparent availability of good medications. Indeed in the early 1990s, a pharmaceutical firm ran an art competition for young asthmatics and asked them to paint what their asthma represented to them. A number of horrifying images like the ones used to illustrate this article emerged.

The asthma guidelines produced by numerous national and international bodies stressed the assessment of severity as the starting point for the initiation of appropriate treatment. Difficulties in using these guidelines arose as clinicians sought to slot patients into severity grades when they were already receiving treatment. The guidelines did, however, serve the purpose of ensuring the severity of asthma was not underestimated.
Numerous studies compared the two combination inhalers and the various inhaled steroids on the market. These were mostly commercially driven and designed to show the superiority of one product over another. When the studies were re-analysed with a different intention, however, it was demonstrated that even in a closely monitored and supervised study situation a surprisingly small number of patients achieved good control. It was recognised that while the treatment of conditions such as hypertension and diabetes were guided by clear end points; the control of blood pressure and of glucose levels, the treatment of asthma was often not pursued to the point of achieving optimal control. In other words, as long as the patient was on treatment and not complaining, therapy was considered adequate.

The Gaining Optimal Asthma Control (GOAL) study was one of the landmark investigations looking for optimal asthma treatment. In this study subjects entered with varying severity and treatment regimens but had their treatment increased over the course of a year until they achieved optimal asthma control or reached maximal therapy. In order to qualify as “totally controlled” the subjects health had to reflect the following:

- No daytime symptoms
- No use of rescue beta2- agonist
- No night time symptoms, exacerbations or emergency room visits
- An objective measurement of peak flow at greater than 80% of predicted

To be “well-controlled” required minimal symptoms and no markers of severity, such as the emergency room visits. The medical profession has recognised over the last 10 to 15 years that for asthma to be adequately controlled the symptoms must be controlled and lung function optimised so that the asthma interferes as little as possible in the daily lives and activities of asthmatics, including school, sport and work.

The approach of modifying treatment progressively until optimal control is achieved prompted change to many international guidelines, including those of GINA. The emphasis is now on assessing control rather severity alone, so that the condition of asthmatics is classified as well-controlled, partially controlled or poorly controlled (Table 1). An asthmatic with severe disease that is well-controlled may now be contrasted with a mild asthmatic with poor control.

There are well-validated simple asthma control questionnaires that can be used to assess the level of control. These together with objective measurements of lung function can be used to guide and modify treatment. The 2006 European National Health and Wellness Survey showed that a large proportion of asthmatics had uncontrolled asthma. A follow-up in 2008 estimated asthma prevalence and control in five European countries. The prevalence of self-reported physician diagnosis of asthma was 6.1%. In treated asthmatics 18 years and older, 56.6% were found “not well controlled” when using the five-question, self-completed asthma control test (ACT). The study also looked at the impact of asthma control and found subjects with asthma that was “not well-controlled” suffered significant interference with their work, school and home activities compared to those who were “at least well-controlled” (Table 2).

### Table 1 – Levels of asthma control (adapted from GINA guidelines)

<table>
<thead>
<tr>
<th>Condition</th>
<th>Controlled</th>
<th>Partly controlled</th>
<th>Uncontrolled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daytime symptoms</td>
<td>Twice or less per week</td>
<td>More than twice</td>
<td>3 or more features of partly controlled asthma</td>
</tr>
<tr>
<td>Activity limitation</td>
<td>None</td>
<td>Any</td>
<td></td>
</tr>
<tr>
<td>Night time symptoms</td>
<td>None</td>
<td>Any</td>
<td></td>
</tr>
<tr>
<td>Rescue treatment</td>
<td>Twice or less</td>
<td>More than twice</td>
<td></td>
</tr>
<tr>
<td>Lung function PEF or FEV1</td>
<td>Normal</td>
<td>&lt;80% predicted</td>
<td></td>
</tr>
</tbody>
</table>


### Table 2 – Impact of asthma on work school and home activities in past four weeks

<table>
<thead>
<tr>
<th>Time</th>
<th>Not well controlled</th>
<th>At least well controlled</th>
</tr>
</thead>
<tbody>
<tr>
<td>All of the time</td>
<td>3.4</td>
<td>0.1</td>
</tr>
<tr>
<td>Most of the time</td>
<td>10.1</td>
<td>0.0</td>
</tr>
<tr>
<td>Some of the time</td>
<td>27.3</td>
<td>1.4</td>
</tr>
<tr>
<td>A little of the time</td>
<td>33.7</td>
<td>18.7</td>
</tr>
<tr>
<td>None of the time</td>
<td>25.4</td>
<td>79.8</td>
</tr>
</tbody>
</table>
It is established that, even in countries with well-developed health systems, asthma control is suboptimal in many cases. This is despite the availability of medications that promise control of the disease with minimal or no side effects. So why should we be doing so badly?

It is the nature of asthma to cause symptoms so it is natural that patients will determine their treatment by the presence of symptoms rather than commit to regular preventative treatment. In conditions that are not symptom-driven, such as hypertension, patients will take the medication regularly in order to reduce the likelihood of poor long-term outcomes. Many asthmatics need to be convinced that poor control of asthma has the potential to lead to fixed airway obstruction and adverse outcomes, including acute deteriorations and hospitalisation and even death.

Commonly, there is non-adherence to medication regimes, a tendency for people to “deny” their disease and to resist inhaler therapy. Some favour taking tablets, feeling that using an inhaler reinforces the presence of their disease – a constant reminder to themselves as well as others. Many such problems, including poor inhaler technique, can be overcome with appropriate education and support that encourage patients to use objective outcome measures, including lung function, control questionnaires and action plans, if their symptoms escalate.

It is difficult to control an inflammatory airway disease in people who continue to smoke. Emotional factors and stress can also lead to poor control. Doctors must be alert to other contributing and aggravating factors, such as upper airways allergy, sinusitis and gastrointestinal reflux.

Although most combination inhalers are prescribed for use twice a day, it is not uncommon for one dose to be omitted. Once-a-day combination inhalers will soon be commercially available, which should prove beneficial in this respect.

**What new treatments are available?**

Improved understanding of different inflammatory pathways in asthma has prompted investigation of the potential for treating it using biological agents.

The sole available product of this type is an anti-IgE antibody drug Omaluzimab (Xolair). IgE is the antibody pivotal to the allergic reaction and its activation releases many inflammatory mediators. In theory, by blocking IgE the inflammatory pathway is aborted. Omaluzimab works by binding to IgE when it is released and inactivating it. Antibody to IgE is produced in a murine model before being humanized and given by subcutaneous injection every two or four weeks. The product has shown most success in reducing exacerbations in severe asthmatics who have elevated IgE, but its cost means widespread use has been limited.

With long-standing severe asthma, hyperplasia and hypertrophy of the airway smooth muscle can take place. A bronchoscopic technique known as bronchial thermoplasty has been introduced. A bronroscope is inserted into the airway and a current is applied via an endobronchial catheter. This causes regression of bronchial smooth muscle and perhaps modifies the asthma process by other mechanisms including denervation. The results of this technique have now been followed for five years or more. The technique appears to show promise in severe asthmatics by reducing exacerbation rates but has little effect on improving lung function. Cost once again is a significant limiting factor and patient selection is important as experience with the technique continues.

Asthma is already common and is set to become more so. Despite the availability of effective medications, the asthmatic condition of a surprisingly large number of individuals remains poorly controlled. This has significant impact on their performance and indeed asthma is potentially fatal. In the assessment of an asthmatic, both severity and adequacy of control are important. Control can be assessed by asthma control questionnaires and lung function. The medications used to treat asthma are generally safe with minimal long-term side effects or complications. The one exception is oral corticosteroid which is seldom required as maintenance treatment today and would be reserved for only the most severe asthmatics.
Endnotes

About the Author
Dr. John O’Brien is the CMA for Gen Re in South Africa. He is a pulmonologist in practice in Cape Town. He is a past president of the South African Thoracic Society and is on the editorial committees for the South African Thoracic Society guidelines for asthma and COPD. He has been a clinical investigator in more than 70 clinical trials in asthma and COPD. Tel. +27 21 412 7700 or john.obrien@genre.com.
Eating Our Own Cooking – Reinsurers As Advisers

by Bernhard Wolters, Gen Re, Cologne

The core business of reinsurers lies in evaluating and assuming risk (in short: risk transfer). Moreover, they design new products for cedants or actively participate in their product development. Last but not least, reinsurers provide a comprehensive transfer of knowledge in the form of seminars, training, publications and systems.

The concentric bands in Figure 1 highlight the nucleus of reinsurance that risk transfer forms. Around it, all other activities fall into place and assume their auxiliary natures. Product development, for instance, is commonly not offered on a standalone (fee) basis but free of charge in return for the prospect of reinsuring the new product after its market launch. In the same vein, knowledge transfer in its various forms usually does not carry a (significant) price tag for existing cedants.

Figure 1 – Reinsurers’ business model

The figure and its interpretation can be viewed as representative of how most reinsurers would describe their business model. By and large, the majority of cedants has subscribed to this perception. Therefore, whenever product development is their major concern, they are likely to consider risk transfer as a necessary evil, or even a deal-breaker, rather than an added value.

The intention of this article is to unmask this view as a misconception that may have enticed many direct insurers to turn to the services of consultants while they could have obtained much better support from their reinsurers. It will be shown that, rather than a necessary evil, the reinsurers’ participation in the insurance risk of (co-)developed products turns out to be a key element in significantly enhancing the direct insurers’ chances of success.

We will also explore the nature of advice in a business-to-business environment and explain why advice can be biased to the detriment of its recipients. Finally, we will explain how incentives must be structured in order to streamline the interests of adviser and advisee. The conclusion contains a summary of the elements that constitute the reinsurer’s competitive advantage in its combined role as a consultant and a risk-taker over “risk-free” forms of advice.

Nature of advice and its possible bias

Advice is always triggered by a problem. If the economic actor facing the problem is aware of it and recognises a need for advice, he will actively seek it. If not, advice may also come unsolicited whenever the adviser sees a problem facing the advisee of which the latter is not aware. In its most abstract form, advice can arguably be considered as an expansion of the scope of action a decision maker has at his disposal in order to solve a problem. For advice to be successful, the additional scope of action must be relevant for the solution of the problem. The need for advice arises because the advisee lacks the necessary information to (better) solve a particular problem.

We assume that in their interaction both adviser and advisee attempt to maximize their respective profits. Without further incentive adjustments, the profit targets can be antagonistic because, on the one hand, the revenue generated by the adviser constitutes an expense for the advisee. On the other hand, any cost reduction on the part of the adviser tends to reduce the quality of advice and the resulting profit accruing to the advisee. The inevitable information asymmetry between the adviser and the advisee will likely induce the former to maximize his profit at the expense of the latter unless both parties’ incentives are brought in line. This incentive problem is tackled by Principal-Agent Theory, from which our further analysis will borrow.
Theoretical framework – Principal-Agent Theory

The starting point of Principal-Agent Theory is the lack of information preventing an economic actor (the Principal P) to solve a problem. Another economic actor (the Agent A) who possesses the necessary information (e.g., skills, knowledge) performs the required task in lieu of the principal.\(^8\) According to one expert, “The central concern [of principal-agent theory (the author)] is how P can best motivate A to perform as P would prefer, taking into account the difficulties in monitoring A’s activities.”\(^9\)

The example in Figure 2 provides a useful framework to explore in more depth the relationship between P and A.

Figure 2 – Principle-agent relationship

<table>
<thead>
<tr>
<th>e = 0</th>
<th>X</th>
<th>100 (good)</th>
<th>P_g = 0.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>e = 40</td>
<td>X</td>
<td>170 (good)</td>
<td>P_g = 0.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50 (bad)</td>
<td>P_b = 0.5</td>
</tr>
<tr>
<td></td>
<td>100 (bad)</td>
<td>P_b = 0.5</td>
<td></td>
</tr>
</tbody>
</table>

The payoffs X in Figure 2 accruing to P are subject to a random process with equal probabilities P_g and P_b. Moreover, they depend on A’s effort e, which, in our example, is either equal to nil or to 40.

In our advice setting, the advisee will serve the role of the principal P while the adviser will be the agent A. Other applications include, for example, employer (principal) and employee (agent) or regulator (principal) and regulated firm (agent).\(^10\) Principal-Agent Theory thus provides a general framework for the analysis of economic interaction in the face of information asymmetry. In what follows, we will first look at its classic treatment of the major incentive problems, i.e., the elimination of moral hazard and the distribution of risk.\(^11\) The insights gained from the classic treatment will subsequently be used to discuss the specific case of product development in insurance.

The classic case

In the classic case, P can observe neither A’s effort e nor the outcome of the random process OR (e.g., good or bad weather in the case of a harvest). A, however, always knows e and OR. Figure 3 below illustrates the timing of the interaction between P and A. First P offers a contract to A, which the latter can either accept or reject (A1). If he accepts, A will then observe the outcome of nature’s (N) random process OR in order to choose the level of his effort e (A2).

Figure 3 – Timing of interaction between principal and agent

If the contract, that P offers A, remunerated A for his effort e, this would enable the latter to cheat (moral hazard). He could simply claim that OR = “bad” and e = 40 while in fact OR = “good” and e = 0 (see Figure 2). To avoid that, P could ask A to pay a fee F of 90 in exchange for the right to collect the payoff resulting from the effort e and the outcome of the random process. If accepted, this contract would induce A to make an effort e equal to 40 in order to obtain an expected profit Y of 5. However, he would have to carry the entire risk because he effectively buys P’s operation for a fee F. To see this, consider equation (1):

\( Y = E(X(40)) - F - e \)

where \( E(X(40)) \) is the expected value of X given e = 40

Solving for e = 40 and F = 90 gives:

\( Y = 0.5 \times 170 + 0.5 \times 100 - 90 - 40 \iff Y = 5 \)

With this contract, P is better off compared to the situation in which e = 0, for he collects a certain profit of 90 instead of an expected profit of 75 (0.5 \( \times 100 + 0.5 \times 50 \)). A makes an expected profit of 5 instead of nil. Whether or not A accepts P’s offer depends on his risk attitude. If he is risk-neutral or risk-seeking, he will happily pay a fee of 90 for the
right to collect the random payoffs at e = 40. If, however, he is risk-averse, he may consider the possible profit of 40 (170 - 90 - 40) in case of X = 170 as insufficient to compensate for the equally probable loss of 30 (100 - 90 - 40) in case of X = 100.12

The specific setting – product development

The treatment of advice in product development differs from the classic case in two important ways.

Firstly, in the classic case the advisee (P) knows X(e), i.e., the adviser’s productivity, but cannot observe e. We have seen above that in this setting the risk of moral hazard on the part of the adviser is eliminated by a contractual arrangement in which the adviser pays an upfront (franchise) fee to the advisee. In product development, however, it is more realistic to assume that the advisee can in fact observe the adviser’s input e but does not know X(e). Thus, the insurance company obtaining advice has a pretty good idea of how much time and effort the consultant has invested (e.g., reports and meetings). However, it is not clear whether the output X the insurance company observes corresponds to the output according to the consultant’s true productivity X(e). In other words, the advisee simply does not know whether the adviser’s input e is efficient or whether he has charged too many redundant consulting hours.

Secondly, and unlike the classic case, the specific setting of product development assumes that in the case of the adviser (A) purchasing the operation from the advisee (P), the successful outcome still depends on P’s input.

Figure 4 illustrates the timing of the interaction between P and A in the new setting. First P offers a contract to A, which the latter can either accept or reject (A1). If he accepts, A will then choose the level of his effort e (A2). The final outcome depends on nature’s (N) random process OR.

Under these circumstances, a fee per hour would induce an (inefficient) oversupply of e because A can take advantage of the fact that P is unable to judge the necessary level of e for the fulfillment of the task at hand. As in the classic treatment of the agency problem discussed above, the insurance company could alternatively ask for a (franchise) fee in return for ceding the expected profits from the newly developed insurance product to the adviser. While eliminating the adviser’s moral hazard, this may create a moral hazard in the advisee. To see this, consider that the advisee, after having received the fee, will no longer have any incentive to work because any incremental profit generated by his effort will fully accrue to the adviser according to the terms of the contract. This will not be problematic if the advisee’s effort does not make any difference, such as in the classic treatment discussed above. If, however, the advisee’s input matters, as it is reasonable to assume in the case of product development,13 the adviser will be fully exposed to the former’s moral hazard.

We have thus identified two contractual arrangements that are unlikely to materialize because the risks of moral hazard and volatility have to be fully borne either by the advisee or the adviser:

a) The adviser gets a payment equal to his effort e. This allows him to cheat the advisee since the latter does not know how much output he can reasonably expect for a certain amount of input e (the adviser’s productivity). Moreover, the advisee carries the entire volatility risk because he only receives the residual profit after deduction of the fixed fee e from the random output X.

The advisee thus carries the entire volatility risk and the risk of the adviser’s moral hazard.
b) The advisee gets a payment $F$. This way the adviser carries the entire volatility risk because he only receives the residual profit after subtracting from the random output $X$ his effort $e$ and the payment $F$. To make matters worse, the advisee, after having received the payment $F$, will no longer have an incentive to help the adviser achieve his target output. So if the output (partly) depends on the advisee’s effort, the adviser may fall short of his profit target.

The adviser carries the entire volatility risk and the risk of the advisee’s moral hazard.

These problems can be circumvented by the following contract:

c) The adviser provides $e = 40$ free of charge and, in return, profits and losses are shared equally between the two parties.

Using the example from Figure 2 gross profits for $e = 40$ are distributed as follows:

- Gross Profit (good) = $170 - 40 = 130$
- Gross Profit (bad) = $100 - 40 = 60$

To arrive at the advisee’s net profit we have to deduct the payoffs for $e = 0$, i.e., the profit the advisee would have obtained if he had chosen not to involve the adviser.

- Net Profit (good) = $130 - 100 = 30$
- Net Profit (bad) = $60 - 50 = 10$

The expected value of the net profit is equal to $20 \cdot (0.5 \cdot 30 + 0.5 \cdot 10)$.

If results are shared equally, each party will on average expect a net profit of 10.

In this profit-sharing arrangement, the problems highlighted for the contracts under a) and b) disappear. Both parties benefit from working diligently and efficiently towards a maximum profit. Neither has an incentive to cheat and moral hazard is therefore no longer an issue. Moreover, both parties assume equal shares of the volatility risk.

**Conclusion**

We have analysed the incentive problems surrounding advice within the theoretical framework of Principal-Agent Theory. In general terms, Principal-Agent Theory deals with interactions between economic actors whose knowledge and information are unequally distributed. This is also the starting point in consulting – the interaction between adviser and advisee. We could satisfy ourselves with the fact that the interests of both adviser and advisee are best served when both parties participate in the (volatility) risk of the endeavour, which in our example is the development of a new insurance product. For the sake of simplicity, we have talked so far of volatility risk. In practice, however, the far greater risks in developing and launching a new insurance product are possible pricing errors and unforeseen changes in the pricing parameters (risk of error and risk of change). Unlike volatility risk, these two risks can, to a considerable extent, be contained by timely and diligent monitoring.

Figure 5 below summarizes our major findings. If the adviser participates in the risks pertaining to his consulting, there will be no moral hazard on his part. This describes the situation of the reinsurer as consultant (the upper left quadrant in Figure 5). If, on the other hand, the adviser carries no risk at all, his actions will be prone to moral hazard. This characterizes the situation of the consultant (the lower right quadrant in Figure 5). Moral hazard and risk can both only be absent in an ideal world in which all economic actors possess perfect information and perfect foresight (the lower left quadrant in Figure 5). In such a world, the need for advice would not arise in the first place. The upper right quadrant in Figure 5, i.e., a situation in which the consultant is subject to moral hazard and risk alike, is difficult to imagine in view of our analysis above.

**Figure 5 – Risk and moral hazard in advice**
• Lack of redundancy since the consulting activity itself is not remunerated\(^{14}\)

• Relentless pursuit of diligence during the consulting process since the reinsurer’s success directly depends on the quality of their work

• Strict monitoring of the business in order to contain the risk of error and the risk of change since, like the advisee, the consulting reinsurer is equally exposed to these risks

Risk sharing, rather than a nuisance, is an essential element of good advice. Therefore, reinsurers are in a better position than anybody else to provide their clients with high-quality (product development) service. After all, reinsurers eat their own cooking. As Warren Buffett explains, “We want to make money only when our partners do and in exactly the same proportion.”\(^{15}\)

### Endnotes


2 With the possible exception of systems, for example expert medical underwriting systems.

3 Source of graphs the author unless otherwise stated.

4 Advice in a business-to-non-business environment was treated by the author in an article published in Risk Insights, 2011 – No. 4.

5 The advisee is the party seeking advice.

6 The terms consultant and adviser are used interchangeably.

7 One might add a second reason, i.e., the evasion of accountability. In this case the adviser assumes the role of the scapegoat when unpopular decisions have to be made.

8 The principal could equally involve the agent for want of time to perform a certain task. But also in this case the agency problem arises because the principal cannot monitor the agent’s activities, i.e., he lacks necessary information.


10 Ibid., p. 46.

11 Ibid., p. 46.

12 Risk aversion implies that when you are asked to pay a fee for the right to play a game of chance, you will always pay less than the expected value of this game. If you are risk-seeking, you will always pay more than the expected value.

13 The success of the new product heavily depends on the insurance company’s sales effort and sales skills.

14 The total lack of fees may also reduce the effect of a psychological phenomenon called “sunk cost,” i.e., the reluctance to stop a project when a significant investment has already been made.


### About the Author

Bernhard Wolters is Vice President within the Life/Health department of Gen Re and has for many years been responsible for the Dutch and Scandinavian markets. He can be reached at Tel. +49 221 9738 848 or wolters@genre.com.
Bariatric Surgery
Matthew Ramjan and Lyn Franks talk about the excess of body weight that is associated with an increased risk of morbidity and mortality from cardiovascular diseases.

Our Publications

Mental Health Claims – It’s Time To Think Differently
Viviane Murphy explores the options for managing disability claims, especially those resulting from psychological distress. This issue includes a practical guide to developing a partnership and support model for the management of subjective claims.

Client Seminars

Gen Re, Madrid ran a life seminar in Lisbon on 11 March; “Encontro Análise de Risco” that was attended by 33 delegates from 14 companies. Presentations included; Angel Luis González and Javier Ruiz del Moral, Senior Account Executives, Gen Re, Madrid “Basic Concepts of Life Reinsurance Treaties”, Gloria Palma, Underwriter Life, Gen Re, Madrid “Lifestyle Risk Assessment “, Ana Páez, Underwriter Life, Gen Re, Madrid “The Relevance of the Health Questionnaire” and Dr. Oñoro, CMO, “Respiratory Diseases and the Life Underwriting Process”.

Gen Re, Cologne Gen Re Business School in cooperation with the University of Cologne held the 7th congress “Trends und Risiken in der Lebens- und Krankenversicherung” (Trends and Risks in Life and Health Insurance) on 29 April. 49 participants from 29 insurance companies attended. Gen Re speakers included Andres Webersinke, Regional Director and Head of Life/Health Research & Development; Ulrich Pasdika, Regional Unit Manager Life/Health Germany; Holger Schmarowski, Head of Underwriting International, Life/Health Client Services and Dr. Bernhard Balg, Consultant Medical Officer. Presentations included; “Alternatives to Disability Products”, “Aviation Risks in Underwriting”, “Problems with Prescription and Intake of Medication”, “Incidental Findings in Imaging Techniques” and “Risk Behaviour of “Generation Y”.


Gen Re, Mexico ran a seminar for life and non-life insurers in Cancun 7 to 9 May. Over 40 participants from 10 different Latin American countries attended. Dr. Winfried Heinen, member of General Re’s Board of Executive Directors, presented “The Impact of Global Economic Environment on the Insurance Sector”. Carmelo Galante, Regional Manager, Life/Health, Gen Re Latin America co-presented “The Potential Impact of Emerging Risks”, Alfredo Fetter-Nathansky, Head of Region, Gen Re Latin America and Mediterranean, co-presented “Dynamics of the International Reinsurance Market and Future Prospects”.

Gen Re, China held the 6th Summit for Chief Operations Officers and Senior Underwriting and Claims Managers in Yinchuan from 21 to 23 May, attended by 50 participants from 32 companies. Topics included; Dread Disease in China, Medical Insurance Claims, Simplified Underwriting and Medical Experience in Asia.

Gen Re, Mexico hosted a one-day seminar on 4 June for Latin American Life and Health insurers. Alberto Zazo, Underwriter Life, Gen Re, Madrid and Sarah Ramirez, Account Executive, Gen Re, Mexico presented.
Andres Webersinke, Regional Director and Head of LifeHealth Research & Development, presented “(In)consistency analysis of underwriting decisions” at the Third Conference of the European Life and Health Underwriters’ Association (ELHUA) in Prague on 10 April.

Andres Webersinke, Regional Director and Head of LifeHealth Research & Development, presented, together with Prof. Dr. Fred Wagner, Chairman of the Institute for Insurance Sciences at the University of Leipzig, results of an empirical study on Long-Term Care insurance addressing views and optimisation measures from a sales perspective in Leipzig on 25 March. The study was conducted jointly by Gen Re and the Institute.

Mark Your Calendar

Gen Re, UK: ReGenerate seminar – 16 and 17 June 2014
Gen Re, China: Medical Seminars – 16 and 17 June 2014
Gen Re, Madrid: Life and Property/Casualty Spanish Market Seminar – 27 June 2014
Gen Re, Sydney: Roundtable on Genetics – 17 and 24 July 2014
Archibald dinner – 20 August 2014
Gen Re, Mexico: XIV Curso de Vida y Salud – 14 to 19 September 2014
IV Curso de Suscripción Vida y Salud – 14 to 19 September 2014
Gen Re, Cologne: German Disability Insurance Seminar – 30 June 2104
12th International Seminar on Risk Management – 22 and 23 September 2014
7th Seminar of International Product Trends – 25 and 26 September 2014

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