Many children under the age of seven benefit from ventilation or continuous positive airway pressure (CPAP) but treatment has been hampered until now by the lack of availability of specific masks to suit this age group. In many pediatric units, masks have been made to measure for children requiring CPAP or noninvasive ventilation (NIV). ResMed’s new Pixi™ mask, designed specifically for young children aged two to seven, will make the task of pediatricians much easier. It is already making life easier for families with children who need CPAP or NIV due to conditions such as cystic fibrosis, achondroplasia, progressive neuromuscular diseases and obstructive sleep apnea (OSA).

It is estimated that 12% of all children snore regularly and that 3–4% of children have a sleep apnea. Two recent studies have made the detection and treatment of SDB in children more urgent. In May 2011, a team from the Murdoch Children’s Research Institute, Melbourne, Australia showed that neurocognitive deficits are common in children with sleep disordered breathing (SDB) regardless of disease severity. In July 2011, a team from the Monash Institute of Medical Research, Australia, published an article showing that SDB in children, regardless of its severity, was associated with increases in blood pressure during sleep and wake compared with non-snoring control children.

Dr Karen Waters at the Children’s Hospital, Westmead, in Sydney’s western suburbs treats children with OSA, narcolepsy and a range of other sleep disorders. Most children who present with OSA can be treated by an adenotonsillectomy, but there is a small number who still need CPAP therapy instead of or after surgery. Her unit is currently monitoring 165 children on CPAP.

The importance of sleep for older children can’t be overestimated either. As Dr Mary Carskadon, Professor of Psychiatry & Human Behavior, Brown University, Rhode Island, USA says of the adolescents she works with, ‘Sleepy people lack attention, lack motivation, and are easily distracted.’ This is true for children, teenagers and adults yet symptoms can differ between the ages. Children with sleep disorders might present with behavioral problems or bedwetting; adolescents might be depressed or have low motivation; adults generally snore, but may also have nightmares or sexual dysfunction. At any age, OSA can increase a person’s risk of high blood pressure, diabetes and heart problems.

The final article in this edition of ResMedica assesses Climate Control, the new approach to humidification from ResMed. Dr Claus Ziegenbein describes the problems with traditional humidifiers and reviews results from the use of Climate Control.

This edition of ResMedica marks the tenth anniversary of our publication. The first edition, in 2002, featured a lengthy interview with CPAP pioneer Dr Michel Berthon-Jones; subsequent editions have focused on topics such as Cheyne-Stokes respiration, congestive heart failure, diabetes, obesity, fatigue, complex sleep apnea, NIV, hypertension, PAP compliance, the economics of OSA and the invention of CPAP. All back issues are available on our website.

Our objective in 2002 was to be a valuable source of information on sleep disordered breathing, and we continue to aim for that goal. I hope you enjoy this anniversary edition of ResMedica and I look forward to your comments.

We are grateful to all of our interviewees for generously sharing their time and knowledge with us.

Sleep well,
Alison Hansford, Global Editor.

Developing a pediatric mask: 
An interview with Alicia Wells

Alicia Wells was the Team Leader of the design team and the systems engineer for the Pixi™ mask. She started working at ResMed six years ago with qualifications in Mechanical Engineering and Commerce. She is now a Section Leader and Systems Engineer.

Are pediatric masks just smaller versions of adult masks?

Usually the masks that children use are a scaled-down version or the smallest size of an adult mask. This is because there haven’t been mask options for pediatric patients and so the clinician has just tried to make the available masks work for their patients. That’s why the Pixi mask is such a good addition to our mask portfolio – it has been specifically designed for kids aged two to seven. Children this age have a very different face shape to adults; they aren’t just smaller versions of an adult. If you look at an average two-year-old, they have a little button nose with no defined nose bridge and their forehead is further out compared to their face. As we grow, our faces literally grow outwards into our heads! This is important when we consider designs for children because if you put an adult mask with a forehead support on a child, the mask has to be overtightened to get a seal around the nose, and this ends up compressing their soft and growing facial bone structures.

Do children have different needs in a mask?

Yes! It’s not just their anatomy that is different, but the use and fitting scenario is different as well. Can you imagine coaxing an average two-year-old to wear a mask each time they sleep? The Pixi mask and all its peripheral support material have been designed to make this as easy as possible. All the mask adjustment and fitting features are designed so the parent can face the child while fitting and adjusting the mask. There is no forehead support or anything covering the child’s eyes, so that the parent and child can see each other and, as one trial participant said, give each other bed-time kisses!

In addition to this, the marketing team have done a great job with developing a lot of support material for the kids and their parents, with the Pixi Character, a parent guide, coloring sheets and in some regions, a Pixi Story book.

How did you decide on that particular design?

We started by scanning ResMed employees’ children’s heads and feeding this information into computer-aided design (CAD) software. The mask was then developed from that data. It was trialled on children at the Children’s Hospital, Westmead in Sydney. The design was refined based on the feedback from this trial and from subsequent trials.

The number one aim was to make the cushion as soft and as compliant as possible. This was to minimize the chance that it would compress the child’s facial bones and to allow it to fit both a two-year-old and a seven-year-old. In everything we did the safety of the child was considered, with any small parts being permanently tethered to the mask. From a design process point of view, it was designed to have no new process, materials or suppliers. This minimized risk, design time and the size of the design team working on the product.

What trials have been done on the Pixi?

The original trial was completed at Westmead Hospital, Sydney, Australia. We then refined the design and had two fitting trials on ResMed employees’ children, where they trialled the mask for as long as they could manage while we assessed ease of fit, seal and comfort. Once the design was further refined it was trialled again at the Mater Children’s Hospital, Brisbane, Australia and three hospitals in the USA.

“All the mask adjustment and fitting features are designed so the parent can face the child while fitting and adjusting the mask.”

Anything else you want to add about the design process?

Many people from all parts of the business have put in extra effort because it is such a new market and patient population. Normally we would extensively test the fit of the mask on ourselves in the design team. However, with this design that wasn’t possible. Lots of the usual bench tests in V&V (Verification & Validation) had to be re-thought to make them relevant to this patient population. Even trialling the mask was difficult, and the clinical team had to work hard to get ethics permission and then find children who were willing to trial the mask. It was a big team effort across the ResMed business and a significant investment for some sick children who don’t have many options for comfortable, effective therapy.
Jo and Greg’s son Isaac is now five and a half years old. He was born with achondroplasia, a disorder of bone growth that causes a common type of dwarfism. He has been using CPAP since he was 6 months old.

Isaac was tiny when he was born – only five pounds (2.3 kgs) – and the pediatrician told Jo and Greg that there may be ‘an issue’. The issue took some time to resolve as the first two sets of tests were inconclusive, but finally Isaac’s worried parents received the news that their son had achondroplasia. It was confronting news, but they have supported each other through the struggles it entailed.

One of the consequences of Isaac’s condition is that he has very small airways. CPAP was recommended to improve his breathing at night and the flow of oxygen to his brain, and his parents took on this new challenge with patience and perseverance.

“From the beginning Jo and I did this together,” Greg tells us. “We had never had any experience with CPAP ourselves so we didn’t know what to expect. We worked with the Mater Hospital [in Brisbane, Queensland] to establish what was required, but then on that first night at home we were on our own.” It took them an hour and a half to get the mask on Isaac and for him to fall asleep. The next night, working together again, it was a little easier. It took the best part of six months for the three of them to be comfortable with the process.

“Greg was so patient,” Jo adds. “He always said, ‘We can do it.’ But it was difficult at first, getting Isaac used to the airflow from the CPAP machine. He was so tiny, and at that stage we only had a petite adult mask for him. The mesh headgear was enormous on his head, and we would have to tuck it and pin it to hold it on him. It was hard work, breastfeeding him at night then getting the mask back on and putting him to sleep.”

Jo and Greg made a conscious decision that Isaac would use his CPAP device every night. “We don’t miss a night for anything,” Greg says. “It’s important to keep the routine going – once you break the routine you’ll have trouble.” With earlier, larger CPAP devices this kept them at home, but now they have a smaller, portable device and can be more flexible, even enjoying the freedom of camping.

All three of them are experts with CPAP now. Jo and Greg can tell if there is something wrong just by the noises the machine makes, such as water in the tubing or leaks from the mask. Isaac is also accomplished in using the equipment, is able to put the mask on and off himself, and tells his parents if there are leaks so they can adjust it.

The benefits are enormous. Isaac is not just developing well – he’s more lively than the other children around him. “He’s so energetic,” Jo exclaims. “He wakes up refreshed. Other kids might be sleepy or grumpy but he runs on six cylinders every day.”

Isaac has been participating in a trial on the new pediatric mask, the ResMed Pixi™ mask, and they’re finding it is a big improvement on the previous masks they were using. “We’ve got through a lot of masks since we started,” Jo explains. “All that baby drool meant they didn’t last long! We had to make all sorts of adjustments in the past, but we’re finding that the Pixi fits him perfectly. One of the good things about it is that he can lie in bed and see the book that we’re reading to him. The big masks used to get in the way, but now he can see the whole book. I also notice that the strapping is better. One completely unexpected benefit is that it makes his hair more manageable because it’s not being tangled by the straps. But the main thing is that he can see us, and we can see him unobstructed. It’s lovely for us to be able to go in when he’s sleeping and see his face.”

“Once the parents accept the treatment, it’s easier for the child to accept it as well.”

When we asked Jo and Greg about the effect that Isaac’s use of CPAP has had on them, they both immediately say one thing: they haven’t been able to go out at night. “Everyone was very supportive of us,” Jo says, “but they wouldn’t babysit. They didn’t know how to put the mask on him. The mask and the machine made them feel too responsible. They felt if something went wrong it would be life-threatening.”

But even that hurdle is clearing now, as they have trained Jo’s mother in putting the mask on, and Isaac himself can always pull it on and position it – he just needs help to clip it up. In a few weeks Isaac is going for his first night away with his grandmother, and it’s hard to say who is more excited by the prospect.
CPAP FOR BABIES: AN INTERVIEW WITH JO AND GREG
CONTINUED >

Jo and Greg have a wealth of experience with CPAP and mask-fitting. Their advice for parents of children who need CPAP is simple: Be persistent. Don’t give up. It does get easier. “Children are very resilient,” Jo says. “It’s hard to get used to at first, but if it’s part of their routine they don’t know any different.” “And ask as many questions as you can,” Greg adds. “Find out what back-up is available and use it. Looking back, it would have been good if we had had more training on fitting those first masks. It’s hard enough fitting a mask to your own face, let alone fitting it to a baby’s face. At first you don’t know how tight it needs to be – you want to avoid leaks but you don’t want it to hurt them. Babies can’t tell you!”

And their final tip: acceptance. “Once the parents accept the treatment, it’s easier for the child to accept it as well,” Greg says.

No-one knows how long Isaac will need to use his CPAP for. His facial structure may change, his airways may one day be large enough to cope on their own. But Jo and Greg aren’t worried about that. Isaac is happy with his routine, he’s bursting with energy after a refreshing sleep every night, and his parents are about to get back to enjoying some adult company.

Isaac and his parents were part of a trial on the Pixi mask conducted by the Mater Children’s Hospital, Brisbane, Australia.¹ The report on the trial concluded that: ‘This study has shown that the novel paediatric mask system, known as Pixi, is suitable for the treatment of SDB in children aged 2–7yrs. Children’s parents showed a preference for the usability (comfort, seal, stability, ease of adjusting and overall preference) of the Pixi mask, when comparing to their usual mask. Data taken from PSGs demonstrated that the Pixi mask is efficacious in treating SDB.’

The Pixi mask has also been trialled in a multicenter study in the US.² The study was conducted through Stanford University, Children’s Hospital Colorado, and Gaylord Sleep Medicine, Wallingford. The Pixi (Nemo in the US) mask was evaluated on 14 children for a minimum of 21 nights, once they were acclimatised to the new mask. The study found that, compared to their current mask, there was significantly less leak with the Pixi, and the patients in the study reported significantly more restful sleep and less trouble getting to sleep and staying asleep. No adverse events were reported.

The US trial findings were presented to SLEEP 2012 in Boston, Massachusetts June 9–13, the 26th Annual Meeting of the Associated Professional Sleep Societies, LLC.


Summary of trial findings

The ResMed team (left to right) Joshua Gudiksen, Alicia Wells, Amal Amarasinghe and Kate Molony receives the Design Award for the Pixi mask at the 2012 Australian International Design Awards. Photo: Good Design Australia.
Treating children with SDB: An interview with Dr Karen Waters

Dr Karen Waters is Professor of Paediatrics and Child Health at the Children's Hospital, Westmead, in Sydney's western suburbs. The Children's Hospital is New South Wales' largest dedicated hospital for pediatric care, with 7674 admissions, resulting in 25,435 bed days, in the quarter July–September 2011. Dr Waters heads the SIDS and Sleep Apnea Research Group, which is associated with the Hospital's Sleep Disorders Unit and the David Read Laboratory at the University of Sydney.

Please tell us about your clinical work at the Children's Hospital.

Most of the patients that we see have sleep-disordered breathing (SDB) in one form or another. Probably 60% have obstructive sleep apnea (OSA). Then we have a proportion who come through because of parasomnias, difficult night time behaviors, or sleepiness problems. We get children or infants who need monitoring because they have had a worrying event, and their sleep study shows that they're at risk of having more of those.

We also offer a support service for treating breathing problems during sleep. That includes our program to support continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP) and our home ventilation service. Our home ventilation service manages the majority of children who are ventilated in NSW. We generally start our involvement with those children while they are inpatients but we keep monitoring them as outpatients, so they'll have sleep studies and adjustment of their ventilation and we look after any reviews that they need. We have a case manager at the hospital who manages a lot of issues to do with funding and support. At any one time, we supervise about 16 children on ventilation. Most often that means that they have a tracheostomy and are ventilated so they need carers in the home because of their level of health support.

We have home oxygen, with 15 or 16 kids on oxygen at any one time, particularly neonates, or the newborns who are still on oxygen after they've come out of the nursery. The biggest number of children we support are on CPAP: we have around 165 kids on CPAP who we continue to monitor. We also manage 90 kids on night-time bilevel support. We can't use self-titrating devices on kids yet because the flow detectors are set in the adult range.

Please tell us about your group’s research work.

The group's laboratory research focuses on risk factors for SIDS – nicotine exposure and intermittent hypoxia. Our clinical research focuses on the causes, complications, investigation and treatment of sleep apnea and sleep disorders in children. The group is also studying how the presence of OSA interacts with obesity to influence cardiovascular function in children. Most recently, we are involved with a multicentre Paediatric Sleep Centre collaboration across Australia. That group established a protocol to undertake a definitive study of whether adenotonsillectomy (AT) improves the IQ in children with OSA.

What sorts of patients do you see?

Apart from OSA, our patients might come to us because of concerns about hypoventilation either due to underlying chronic diseases or progressive muscular-type diseases. Their most vulnerable time is in sleep. For example, kids with progressive muscular weakness might start to develop respiratory problems in sleep before they actually get daytime problems. But if we can catch it early on then it reduces the number of hospitalisations and improves their quality of life. By giving them effective ventilation at night, it doesn't change the underlying illness, in terms of their muscular disease, but it certainly improves their respiratory state and ability to function during the day. What tends to happen if we don’t treat them is that they’ll get episodes of respiratory failure and have to come into hospital, often into intensive care, because of those episodes.

We also see a variety of less common pediatric sleep disorders, such as narcolepsy. As pediatricians we’ll see narcolepsy in its early stages, because it often seems to develop in the teenage years. It’s a disorder of the control systems for sleep and wake. One of the presenting symptoms is that they’re incredibly sleepy. They can fall asleep just about anywhere and have sleep attacks. It’s a lifelong disorder that requires medical therapy.

Now that there is more community awareness of sleep disorders we’re seeing them earlier. It’s really important to identify OSA, especially in kids who have other problems. For example Down Syndrome has a high risk for sleep apnea, and it’s quite important to treat their sleep apnea and maximise their quality of life by treatment. Kids who have other syndromes, rather than being exempt from OSA are often more at risk for it. Parents won’t always realise there’s a problem because their child has had noisy or difficult breathing since they were a tiny baby. Sometimes they’ll have been told it’s part of the disorder, or they’ll just think it’s how their child ‘is’. So it’s really important to ask, ‘Does your child snore?’ ‘Do they have trouble breathing when they’re asleep?’ When there are lots and lots of problems parents may just accept it as part of what’s going on with the child and not think of it as another problem.

What are the main problems that children have with sleep?

The biggest proportion of breathing problems involves children with OSA. If you look at the epidemiology it is estimated that about 12% of all children in the community would have regular...
snoring and between 3–4% have OSA. But in children the age of greatest risk is between two and six years of age, and it closely responds to the time when their tonsils and adenoids are biggest. In a pediatric hospital like ours we see children who have other complications, or other problems that increase their risk for OSA.

How do you treat OSA in children?
The first line of treatment is AT but if we get kids with very severe OSA, they can have 60–80 events per hour and the oxygen desaturation can drop to 50%. In that situation we would put them on CPAP as soon as possible and try to get some recovery of sleep and get them settled before we proceed to surgery. Also, if the surgery doesn’t cure their OSA (more likely if it is very bad in the first place), if they’re already used to CPAP they’re more receptive if they continue to need it after surgery. Kids with OSA have a higher risk of complications. They no longer have their tonsils and adenoids (TAs) but they may have ongoing obstruction after the operation, like swelling of the airway. It’s quite likely they’re at risk because they have a relatively small airway and that’s why they had a problem with their TAs. And if in association with their breathing problem they’ve ended up with some breathing control problems, so they’re not as sensitive to CO₂ plus you’ve given them an anesthetic, then they don’t wake up as easily as they should when they have some breathing problems like obstruction or desaturation. There’s quite a bit of evidence now that there are more problems for them in the first day, the first post-operative hours, than for kids who have their tonsils out for another reason.

How often is AT performed for children?
Estimates are that about 2% of children need to have their tonsils out, but we think that is not enough if you consider the number of children who have sleep apnea. About 80–90% of kids have their tonsils out because of symptoms of snoring or apnea. That surgery doesn’t reduce the number of throat infections, but it improves OSA. There have been studies where they’ve compared one group of kids who went to surgery and one group who didn’t, and it made no difference to the number of infections that the kids had in subsequent years.

How does OSA change as children age?
Children with OSA do tend to improve to the point where there are no further obstructions after their TAs are out. But when you look at the proportion of teenagers with OSA it still seems to be around that 4–5%. At that stage it’s evolving into more of an adult disease because they less frequently have big tonsils. Certainly in current populations, they’re more frequently overweight. Younger kids tend not to be overweight – their TAs tend to be their only problem.

What are the symptoms of OSA in kids?
The three big symptoms of OSA that parents see, or should be concerned about in their children, are:
1. regular snoring
2. working hard to breathe when they’re asleep
3. having episodes of apnea or not being able to get breath through.

They are the big three but there are lots of other symptoms, such as mouth breathing, sweating, restless sleep, daytime behavior problems, and bed-wetting. In the daytime, many kids seem to get more hyperactive – one of the questionnaires for OSA describes it as ‘going as if they’ve got a motor’. They tend to get more frequent upper respiratory infections, and more often get illnesses that will end up with them having to visit doctors and take antibiotics. So the effects of OSA are quite far-reaching.

What concerns are there about the long-term affects of OSA?
The first of the three big areas of concern at the moment would be global-diminution of cognitive development – diminished learning and concentration. When you do tests of IQ you can see that even kids who snore perform more poorly compared to kids who don’t snore, even if they don’t have apneas. The second issue that’s had quite a bit of attention paid to it is the cardiovascular system – blood pressure and the functions of the heart, where OSA has something of a dose-related effect on blood pressure. And then the last group is the metabolic dysfunctions. If you look at it in adults, it’s called the metabolic syndrome. So you might not get the whole spectrum of problems in kids but you’ll see higher insulin levels, abnormal lipid profiles and so on. That’s a particular concern in older kids and teenagers who are overweight where we’re already seeing those problems.

How do you treat children with OSA?
First we do a sleep study to see how bad the disease is. Once we’ve identified bad OSA an AT will be recommended and we’ll try to get that as soon as possible. If they’re very bad, we try to stabilise them on CPAP first. For kids with more mild problems, options might be nasal sprays, especially if they have allergies, just to try to reduce the amount of oro-nasal obstruction. If they have persisting sleep apnea, despite having their TAs out, we’ll look at treatment with CPAP. One surgical procedure that we use, especially with babies who have very small jaws as a cause for their OSA, is a mandibular distraction to lengthen the bone and make the jaw bigger, and that affects the size of the airway. That certainly helps get some babies off ventilators and avoid a tracheotomy.
How do you help children adapt to using a mask and flow generator?

The biggest thing that makes a difference is getting them to make some sort of behavioral adaptation before they start CPAP. We fit the mask and get them to work on just being able to wear the mask at home before we even think about getting them used to the CPAP pressure. Usually with their parents’ help, they’re able to adapt to having the mask on their face. We try to have it open so they are able to get used to wearing it as they are going to sleep. Once we’ve done all of that we start introducing them to CPAP pressure. We start with low pressures and gradually increase to the kind of pressure that they need. But we don’t do that over the course of one night – we do it over the course of several days, so that one night we might have the minimum pressure then the next night increase it by one or two centimetres.

Is there any difference between boys and girls and rates or symptoms of OSA?

A lot of the papers will say there’s no difference in the proportions, and no difference in the symptoms, but we tend to see more boys coming through the sleep unit – about 60% compared to 40% girls – and I’ve seen other countries where that’s been a consistent report as well, but it’s not clear why this occurs.

What comorbidities are associated with OSA in children?

Obesity is probably the biggest one. Often children have comorbid sleep problems and OSA can make several other diseases worsen because it causes sleep deprivation. Parasomnias may be worse. Sometimes we’ll see kids with seizures – they’ll get worse until you start treating the sleep apnea. We see kids with cerebral palsy who have sleep apnea because of the general motor dysfunction, but the upper-airway in particular. Kids like that, who are already vulnerable, get more episodes of aspiration, or have more chest infections or more hospital admissions. If it’s causing daytime dysfunction then I see it as a comorbid disorder.

When you treat OSA do you see a reversal of the ill-effects such as learning difficulties, heart function and so on?

We have seen improvement in a number of the problems associated with OSA. There’s a big need for better quality intervention studies – we need to find out how much everything improves with treatment. It’s a big issue to design and undertake those studies, because they tend to be expensive and hard to fund.

Do children with OSA continue to have it as adults?

There are no long-term studies to show yes or no. As we get cohort studies going through that information will slowly help. It’s definitely a disease that runs in families so it seems quite likely that there is an inherited element to it.

The economic cost of sleep disorders in Australia: an update

In late 2011 Deloitte Access Economics published a report called Re-awakening Australia. It was commissioned by the Sleep Health Foundation to update a 2004 estimate of the cost of sleep disorders.

The report focused on the three most well-recognised sleep disorders: obstructive sleep apnea (OSA); restless legs syndrome (RLS); and primary insomnia. It found that in 2010 there were an estimated 1.5 million Australians (8.9% of the population) with these sleep disorders.

The total cost associated with sleep disorders in Australia was estimated at $36.4 billion, of which $5.1 billion was financial costs and $31.4 billion was nonfinancial costs.

The total cost of OSA was estimated at $21.2 billion. Costs were calculated to include the cost of treating OSA and its associated conditions, citing ‘evidence of a causal relationship between sleep disorders and other illnesses and injuries’, particularly for OSA where stroke, depression and motor vehicle accidents form the three largest comorbidities.

The report also assessed indirect financial costs associated with sleep disorders and conditions attributable to them and found that OSA accounted for 62% of the total cost ($2.6 billion). Indirect costs are based on factors such as lost productivity due to premature workforce separation and mortality, and absenteeism; and informal care and other costs of motor vehicle and workplace accidents.

The third issue that the report considered was the human cost of sleep disorders, and estimated the total cost of lost wellbeing at $31.4 billion. It acknowledged that a person living with a sleep disorder will probably experience a lower quality of life through increased morbidity, and may die prematurely, for instance from a motor vehicle accident.

As in 2004, the report found that CPAP is a highly cost effective treatment for OSA, making it a ‘dominant’ intervention for OSA from a societal perspective – saving healthy life and dollars.

The report makes recommendations for priority interventions to address the current fragmented and under-resourced sleep health landscape and concludes that ‘the future is positive if opportunities for action are pursued since such a large proportion of sleep-related impacts are preventable or treatable’.

Dr Mary Carskadon is Professor of Psychiatry & Human Behavior, Brown University, Rhode Island, USA and Director of Chronobiology and Sleep Research at Bradley Hospital. Dr Carskadon received a B.A. in psychology from Gettysburg College (1969) and doctorate with distinction in neuro- and biobehavioral sciences from Stanford University (1979). Dr Carskadon has received many honors, including an honorary doctor of sciences degree from Gettysburg College, Lifetime Achievement Award of the National Sleep Foundation, Outstanding Educator and Distinguished Scientist Awards of the Sleep Research Society. She is an elected Fellow of the Association for Psychological Science and Fellow of the American Association for the Advancement of Science.

Could you please briefly describe your work as the Director of Chronobiology and Sleep Research at Bradley Hospital, Brown University?

I run a ‘cottage industry’, almost literally. We have a small laboratory located on the grounds of Butler Hospital in Providence, Rhode Island. We work with Bradley Hospital, but Butler is just where the space was available. It suits us because it’s close to the Brown University campus, so it’s more convenient for students. I write grant applications and papers for journals. I do research, mentor other researchers, and teach undergraduate students.

We’re currently undertaking two major research projects, both funded by the National Institute of Mental Health (NIMH). We use laboratory research and field research. This means we cover a wide range of research techniques, from the molecular to the behavioural, using methods and measures from PSG to internet research to DNA assessment. I like to stay fresh in my work and am always looking at ways of advancing thinking about the areas I work in.

A major focus of your research is the sleep patterns of young people, particularly adolescents. Could you tell us what is meant by the term ‘adolescence’?

Everyone has their own idea of what is meant by ‘adolescence’. When I looked into it I found that there are many definitions, such as ‘the time between childhood and responsible adulthood’ – this could last a very long time! I prefer to define it as ‘the second decade’. This fits in with the World Health Organisation (WHO) definitions of ages 10-14 as early adolescence and ages 15-19 as late adolescence. There are all sorts of social and cultural factors that influence our perceptions of adolescence.

It’s like the term ‘elderly’ – it used to be that someone who was 65 was ‘elderly’ but that’s no longer the case. At the recent World Sleep conference in Kyoto the keynote speaker was a biologist who has been studying apes. He had a slide showing the life stages in a whole range of primates – humans are the only ones with defined childhood and adolescence.

In my science we look at biological factors and behavioural factors. Biologically, adolescence is linked to the maturation of the reproductive system. It’s also a time when the brain is still wiring up in substantial ways that continue until the early 20s. There’s also a new term – emerging adulthood. We’re looking at first-year university students who fit into that category.

Are there intrinsic biological differences between an adolescent’s brain and younger children? And adults?

Major changes take place to the brain during adolescence – metabolic and structural. We’re still learning exactly what is happening, but broadly, in late childhood and early adolescence we’re programmed for synaptic pruning. The cortical neurones start losing their synapses, with a decline of up to 40% during the adolescent years. This is reflected in sleep electroencephalographic (EEG) data. If you compare a 10 year old, a 15 year old and a 20 year old you’ll see differences in the amplitude of the signal, especially in slow-wave sleep.

If you compare a 10 year old, a 15 year old and a 20 year old you’ll see differences in the amplitude of the signal, especially in slow-wave sleep.

There are other changes as well. Longitudinal neural imaging studies have shown changes in the tracks laid down in the white matter in the brain during adolescence. Once again the sleep EEG reflects these changes, showing the concordance between different brain regions becoming stronger than it was in childhood.

The changes in adolescence are very rapid, beginning between 11-12 and 13-14, with further changes between 15-16 and 17-18-19. These changes continue into early adulthood. There is a further decline in the synapses with aging, but whether it’s part of the same process is not known.
What is sleep like in adolescence?

One of my favourite quotes is from a book by Mary Gordon, where she describes adolescent sleep as being ‘long, dark and sullen’. I love that, because when adolescents are given the opportunity to have those sleeps it’s very apt. They’re ‘sullen’ because of the difficulty they have in waking up – most must wake up well before they would like to. They stay awake later but they would prefer to stay asleep well into the morning.

Adolescents need about the same amount of sleep as in the pre-teen years but their timing shifts. We don’t know why. Their circadian rhythms change. They report that they feel better later in the evening. We call this ‘phase preference’. It seems to be related to pubertal development. When we measure dim light melatonin onset (DMLO) in adolescents we find it’s occurring later in the evening than for younger children.

Sleep is regulated by circadian rhythms and a homeostatic drive (‘sleep pressure’). This sleep pressure builds across the day and then dissolves as you sleep. When we model the build-up of sleep pressure, we can see it’s slower in late adolescence. We infer that the brain is changing to allow them to stay awake longer, even though they still need the same amount of sleep.

Then there are all the attractions of the fun things that adolescents like to do in the evenings. This builds onto this biological tendency and also provides further arousal, compounding the issue. But they still have to get up in the morning and go to school or work!

How does melatonin affect sleep? Is this different for teenagers?

We use the measure of dim light melatonin onset (DMLO) phase as a marker of the biological circadian clock. When the clock mechanism in your brain determines that night-time is coming on the brain produces melatonin; in the morning it shuts off production. Melatonin is associated with night-time activity – for humans, with sleep. DMLO moves later in the evening during adolescent development. If you measure it in 11-12 year olds on a fixed 2200 to 0800 [10pm to 8am] sleep schedule it occurs at about 21:30, but in 13-14 year olds it’s at about 2300.

The enzyme that produces melatonin is affected by bright light and you can influence melatonin production by turning lights on or off. Melatonin secretion takes place for a longer time in the evening during adolescent development. If you measure it 11-12 year olds on a fixed 2200 to 0800 [10pm to 8am] sleep schedule it occurs at about 21:30, but in 13-14 year olds it’s at about 2300.

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Could you explain your investigation into morning light exposure on young adults?

In this study we were looking at two ways of potentially affecting the sleep patterns of young adults. The people we studied were real ‘night owls’, but they still had to get up and do stuff in the morning. We moved their bedtimes to an earlier time and gave them a fixed schedule allowing for 7.5 hours of sleep per night. Our monitoring was able to check that this was occurring. One of the other interventions was to sit in front of a bright light after waking in the morning, but what we found was that the simpler intervention of moving their bedtime was more effective in changing them to an earlier sleep schedule. Giving them that ‘dark’ window was the biggest factor in readjusting their schedule of melatonin onset – bigger than morning light. This was an unexpected result!

How many adolescents are affected by poor sleep patterns?

The majority. Every time I look at data from big samples of adolescents, I see that more than half of them are affected by poor sleep. The most concerning data I’ve seen recently came from South Korea where a study showed that students in the 11th and 12th grades were only getting five hours of sleep per night.

You have attributed cognitive impairment in adolescents to sleep deprivation. What sort of cognitive impairment is taking place?

Cognitive impairment is taking place on a number of levels. One of the big tasks of adolescence is for memory and learning to occur. Acquisition of information is affected by excessive sleepiness. Sleepy people lack attention, lack motivation, and are easily distracted. If there’s a lack of attention the information is not getting in. Once the information is in the brain it needs to be consolidated and enhanced in the neuronal network. In the last decade we’ve learnt that if you sleep well your learning consolidation is better. When we look at retrieving and using information the same factors that affect acquisition come into play – too sleepy, not motivated, easily distracted. The speed of intellectual activity also slows.

Is this cognitive impairment temporary, or are there long-term effects on the brain from sleep deprivation in adolescence and childhood?

This is something we don’t know. It hasn’t been studied, but it’s an important question.

How much is sleep disordered breathing (SDB) contributing to excessive daytime sleepiness in teenagers?

We don’t have very good epidemiologic data about excessive daytime sleepiness in adolescents, but it’s an area of concern. As we’re seeing bigger and bigger kids, who are more susceptible to SDB, we need to know.

Poor sleep in teenagers appears to be a mix of societal and biological factors. What solutions do you propose to help teenagers get better sleep?

Here in the USA, we’ve been looking at how early adolescents have to go to school, and making an effort to delay the school starting times. Where that has been implemented, we’ve seen better attendance rates, lower rates of tardiness, lower rates of car crashes among the students. They have a better approach altogether, and are getting better grades. But it’s a very challenging change to make. Schools here are managed town by town. They’re very locally controlled so wide-scale change is hard to implement.

That’s a societal approach – there’s also the issue of education that’s been missing for decades. There’s a real gap in teaching...
about sleep and circadian rhythms. There are the common sense things that individuals need to do – like not having caffeine after 4pm, not using or even having electronic equipment in the bedroom, not sleeping with your phone nearby. There are also environmental factors like getting more light in the morning and having less light at night.

It appears that sleep isn’t a priority with families. There used to be social rhythms in place that helped with the circadian rhythms – like having supper and breakfast together. Where families aren’t doing that it’s harder for good sleep patterns to develop.

Do teenagers with poor sleep patterns carry those patterns into adulthood or does something change as they get older?

Hard data on this is difficult to come by, but Dr Till Roenneberg used chronotypes as a measure in thousands of people to come up with a biological marker for the ‘end of adolescence’. He observed that sleep changed at around the age of 20. Between the ages of 10 and 20 people sleep later and later; around the age of 20 they start to sleep a little earlier again. We don’t know whether that change is influenced by a biological drive or whether it’s because of different kinds of social input, responsibilities, cognitive functions. It may be that the changes of adolescence relax as we reach adulthood.

Whether the bad sleep habits of adolescence will change or whether they’ll carry over – we don’t know. There’s a risk of it occurring. The hard data is sparse, but popular literature shows that we’re increasingly wedded to the electronic devices that disrupt our sleep.

You have been publishing papers on your research into sleep since 1974. During that time you have worked with some of the pioneers of sleep research, such as William C Dement. Could you tell us something of the changes in sleep research that you have seen over that time?

When I started this was a tiny field. There were no sleep clinics. I would go to national sleep conferences and there would be 150 people. I’ve just been to a conference in Minneapolis and there were 7000 people there.

We were a small but dedicated cadre of scientists working to ‘discover the night’. Everywhere you looked there was new knowledge, so the early years were very exciting. There was big excitement as the clinical services grew – the first was in 1970. At that time there were no sleep journals. The first edition of ‘Sleep’ wasn’t until 1978.

There was the discovery and definition of sleep disorders, then of sleep disordered breathing syndrome. There was huge interest in the science and medicine of sleep, and people were attracted to this new field.

Sadly, since 2008 and the GFC, there’s been a loss of societal will to do science, and that’s making things difficult. We would like to keep building on what we’ve learnt. The depth and breadth of the science has grown exponentially.

This is a field that keeps growing. There are new methods of analysis and measurement. At the World Sleep conference in Kyoto, Japan in October a different way of thinking about sleep and its impact was emerging. For me, the excitement just keeps rising around sleep science.

1 ‘Intrinsic Circadian Period: Development, Delayed Phase, and Genetic Associations’

“...This is a field that keeps growing. There are new methods of analysis and measurement.”

and ‘Prospective Study of Depressed Mood, Short Sleep and Serotonergic Genes’ (Does Short Sleep Lead to Depressed Mood by Altering Serotonin Receptor Function?)

2 J Yamagiwa, Dean, Graduate School and Faculty of Science, Kyoto University, Japan, and President, International Primatological Society. Opening lecture: Evolution of life history strategy in human and non-human primates.


Dr Ziegenbein is Senior Manager Marketing of the Homecare Company of ResMed in Germany.

The discussion about humidification is as old as positive airway pressure (PAP) therapy itself. The therapy pressures that are required for medical efficacy lead to elevated flow rates and air volumes passing through the upper airways. This often overtaxes the natural mechanisms in the patient’s body that humidify and heat the air. These unusual conditions affect the mucosa and frequently lead to symptoms such as dryness in the throat and nasopharyngeal area or rhinorrhea. These symptoms can be alleviated using a heated humidifier.1,2,3

The principle of humidification is quite simple. However, the effective and practical implementation of adequate humidification can be rather complicated due to the underlying physical conditions. The ability of air to transport water is limited and greatly dependent on temperature. Only warm air can transport a sufficient amount of moisture. A process that usually works well during the summer will prove itself limited during the transition and winter months. Decreasing temperatures in the bedroom can quickly lead to the much-dreaded rainout in the tube and mask. It is not uncommon for patients to be woken from their sleep by gurgling sounds and water dropping from their noses.

Tubing wrap

Tubing jackets or ‘tried and tested’ methods such as placing the tube under the bed covers to minimize the drop in temperature along the tubing are some of the remedies that are frequently used. Alternatively, the performance of the humidifier can be adjusted to the ambient conditions manually (in the case of some devices automatically) to prevent rainout. This adjustment will generally make it easier for the patient to sleep through the night; however, there is a risk of the symptoms of dryness recurring. A reduction in the humidifying performance leads to a reduction in the absolute water volume supplied and may result in a recurrence of respiratory tract irritation.

How can physics be used to maintain sleep comfort while increasing compliance and therapeutic efficiency?

The answer is quite simple, but the actual implementation is complicated. Once we actively create suitable conditions for effective humidification, we automatically become independent from outside factors.

Absolute humidity, relative humidity, and rainout

Controlling absolute humidity is a decisive factor when creating the ‘proper conditions’. The absolute humidity alone determines whether or not a patient receives the required levels of humidity to meet his/her individual needs and to address his/her symptoms. Maintaining absolute humidity is an efficient way of eliminating the undesirable side effects of PAP therapy.

Relative humidity, on the other hand, indicates the current percentage of utilization of the physically defined water absorption capacity and is greatly dependent on temperature. Maintaining relative humidity does not guarantee that the supplied water volume, ie the absolute humidity of the gas mixture, will be maintained at fluctuating temperatures.

Previous efforts to control the air humidification during PAP therapy focused on the prevention of rainout, ie keeping the relative humidity below 100% – in other words, below the dew point.

The Climate Control algorithm

Climate Control by ResMed takes a different approach. The patented Climate Control algorithm keeps the absolute humidity constant, even under fluctuating ambient conditions.

Climate Control takes care of the patient throughout the night, the level of humidification preset by the patient himself/herself. The Climate Control system is controlled simply by using the S9™ setting dial to set the preferred air temperature on the large color display. Climate Control automatically provides 80% relative humidity based on the preset temperature. In other words, the patient needs to set his/her preferred air temperature. Daily therapy application by thousands of satisfied patients confirm that Climate Control is setting new standards.4

Five sensors transmit the necessary data regarding ambient conditions and the current therapeutic situation to the S9 system, allowing the system to react immediately with pinpoint accuracy. The ClimateLine™ heated tube, included with the H5i™, offers a unique temperature sensor positioned near the mask that compares the current air temperature at the mask with the air temperature preset by the patient in order to control the heater.

For the patient, this means constant conditions throughout the night in terms of temperature and humidity levels. Rainout is effectively prevented and the urgently required respiratory humidification is provided without compromise since the supplied humidity volume remains constant throughout the night, even during fluctuating ambient conditions.
CLIMATE CONTROL: REVOLUTIONARY OR GIMMICK? HUMIDIFICATION DURING PAP THERAPY
CONTINUED >

WITHOUT CLIMATE CONTROL

Fig. 1: Relative and absolute humidity at fluctuating temperatures

<table>
<thead>
<tr>
<th>Time</th>
<th>Temperature</th>
<th>AH (mg/l)</th>
<th>RH (%)</th>
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<td>6 AM</td>
<td>15°C</td>
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WITH CLIMATE CONTROL

Fig. 3: Relative and absolute humidity at fluctuating temperatures with Climate Control

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<tr>
<th>Time</th>
<th>Temperature</th>
<th>AH (mg/l)</th>
<th>RH (%)</th>
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<td>2 AM</td>
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<tr>
<td>6 AM</td>
<td>24°C</td>
<td>17</td>
<td>80</td>
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Fig. 2: Humidification without Climate Control

Fig. 4: Humidification with Climate Control
Fig. 5: The five sensors of the Climate Control system

Conclusion
S9 with H5i and ClimateLine is the first system to provide genuine humidification with constant temperature and humidity levels throughout the night, completely independent of the ambient conditions in the bedroom. With regard to humidification and its possible side effects, Climate Control is a huge step forward in terms of achieving compliance and therapeutic efficiency.

4 ResMed Homecare Company, Germany
OBJECTIVES: This revised clinical practice guideline, intended for use by primary care clinicians, provides recommendations for the diagnosis and management of the obstructive sleep apnea syndrome (OSAS) in children and adolescents. This practice guideline focuses on uncomplicated childhood OSAS, that is, OSAS associated with adenotonsillar hypertrophy and/or obesity in an otherwise healthy child who is being treated in the primary care setting. METHODS: Of 3166 articles from 1999–2010, 350 provided relevant data. Most articles were level II–IV. The resulting evidence report was used to formulate recommendations. RESULTS AND CONCLUSIONS: The following recommendations are made. (1) All children/adolescents should be screened for snoring. (2) Polysomnography should be performed in children/adolescents with snoring and symptoms/signs of OSAS; if polysomnography is not available, then alternative diagnostic tests or referral to a specialist for more extensive evaluation may be considered. (3) Adenotonsillectomy is recommended as the first-line treatment of patients with adenotonsillar hypertrophy. (4) High-risk patients should be monitored as inpatients postoperatively. (5) Patients should be reevaluated postoperatively to determine whether further treatment is required. Objective testing should be performed in patients who are high risk or have persistent symptoms/signs of OSAS after therapy. (6) Continuous positive airway pressure is recommended as treatment if adenotonsillectomy is not performed or if OSAS persists postoperatively. (7) Weight loss is recommended in addition to other therapy in patients who are overweight or obese. (8) Intranasal corticosteroids are an option for children with mild OSAS in whom adenotonsillectomy is contraindicated or for mild postoperative OSAS.


OBJECTIVES: Examine statistical effects of sleep-disordered breathing (SDB) symptom trajectories from 6 months to 7 years on subsequent behavior. METHODS: Parents in the Avon Longitudinal Study of Parents and Children reported on children’s snoring, mouth breathing, and witnessed apnea at >=2 surveys at 6, 18, 30, 42, 57, and 69 months, and completed the Strengths and Difficulties Questionnaire at 4 (n = 9140) and 7 (n = 8098) years. Cluster analysis produced 5 “Early” (6-42 months) and “Later” (6-69 months) symptom trajectories (“clusters”). Adverse behavioral outcomes were defined by top 10th percentiles on Strengths and Difficulties Questionnaire total and subscales, at 4 and 7 years, in multivariable logistic regression models. RESULTS: The SDB clusters predicted (approximate)20% to 100% increased odds of problematic behavior, controlling for 15 potential confounders. Early trajectories predicted problematic behavior at 7 years equally well as at 4 years. In Later trajectories, the “Worst Case” cluster, with peak symptoms at 30 months that abated thereafter, nonetheless at 7 years predicted hyperactivity (1.85 [1.30-2.63]), and conduct (1.60 [1.18-2.16]) and peer difficulties (1.37 [1.04-1.80]), whereas a “Later Symptom” cluster predicted emotional difficulties (1.65 [1.21-2.07]) and hyperactivity (1.88 [1.42-2.49]). The 2 clusters with peak symptoms before 18 months that resolve thereafter still predicted 40% to 50% increased odds of behavior problems at 7 years. CONCLUSIONS: In this large, population-based, longitudinal study, early-life SDB symptoms had strong, persistent statistical effects on subsequent behavior in childhood. Findings suggest that SDB symptoms may require attention as early as the first year of life.


PURPOSE: We aimed to investigate the prevalence of primary snoring (PS) and its association with neurocognitive impairments. METHODS: Data from a community-based study in 1,114 primary school children were used to identify children who never snored (N = 410) or habitually snored (N = 114). In order to separate children with PS from those with upper airway resistance syndrome (UARS) or obstructive sleep apnoea (OSA), home polysomnography was conducted in all habitually snoring children. Neurocognitive impairments and poor school performance were compared between children who never snored, PS, and UARS/OSA. RESULTS: Polysomnography was successfully conducted in 92 habitual snorers. Of these, 69 and 23 had PS and UARS/OSA, respectively. Prevalence [95% confidence interval (95% CI)] of PS was 6.1% (4.5-7.7). Compared to children who had never snored, children with PS had more hyperactive (39% vs. 20%) and inattentive behaviour (33% vs. 11%), as well as poor school performance in mathematics (29% vs. 16%), science (23% vs. 12%), and spelling (33% vs. 20%; all P values <0.05). PS was a significant risk factor (odds ratio; 95% CI) for hyperactive behaviour (2.8; 1.6-4.8), inattentive behaviour (4.4; 2.4-8.1), as well as daytime sleepiness (10.7; 4.0-28.4). PS was also an independent risk factor for poor school performance in mathematics (2.6; 1.2-5.8), science (3.3; 1.2-8.8), and spelling (2.5; 1.1-5.5). Odds ratios throughout were similar to the UARS/OSA group. CONCLUSIONS: Children
with non-hypoxic, non-apnoeic PS may exhibit significant neurocognitive impairments. Consequences may be similar to those associated with UARS or OSA. If confirmed, PS is not “benign” and may require treatment.


**BACKGROUND:** This study aims to evaluate left ventricular (LV) structure and function and inflammation in a paediatric population with sleep-disordered breathing (SDB) and in control subjects. **METHODS:** Forty-nine children with SDB and 21 healthy, age-matched subjects were enrolled. The diagnosis of obstructive sleep apnoea syndrome (OSAS) was confirmed by the laboratory polysomnography, showing an obstructive apnoea/hypopnoea index of more than one per hour, according to the criteria of the American Academy of Sleep Medicine and modified for paediatric population. Fasting blood samples for the biochemical evaluation (including high-sensitivity C-reactive protein (hsCRP) were drawn in the morning, after the polysomnographic examination in all patients with SDB and in the control group. All children underwent a two-dimensional colour Doppler cardiac examination with LV mass assessment and systolic and diastolic function evaluation. **RESULTS:** Higher hsCRP levels were observed in subjects with OSAS than in children with primary snoring and in controls (0.8 +/- 0.7 vs 0.3 +/- 0.1 ng/dl, p = 0.001, and 0.4 +/- 0.2 ng/dl, p = 0.01, respectively). The LV diastolic dysfunction was significantly more frequent in patients with severe OSAS and higher hsCRP levels than in control group. **CONCLUSIONS:** This study shows that OSAS in children is associated with higher LV mass, early LV diastolic dysfunction and a pro-inflammatory state (high CRP levels). These findings might help to explain the higher incidence of cardiovascular morbidity in patients with OSAS.


**RATIONALE:** Academic success involves the ability to use cognitive skills in a school environment. Poor academic performance has been linked to disrupted sleep associated with sleep-disordered breathing (SDB). In parallel, poor sleep is associated with increased risk for obesity, and weight management problems have been linked to executive dysfunction, suggesting that interactions may be operational between SDB and obesity to adversely affect neurocognitive outcomes. **OBJECTIVES:** To test whether mediator relationships exist between body weight, SDB, and cognition. **METHODS:** Structural equation modeling was conducted on data from 351 children in a community-based cohort assessed with the core subtests of the Differential Abilities Scales after an overnight polysomnogram. Body mass index, apnoea-hypopnoea index, and cognitive abilities were modeled as latent constructs. **MEASUREMENTS AND MAIN RESULTS:** In a sample of predominantly white children 6 to 10 years of age, SDB amplified the adverse cognitive and weight outcomes by 0.55- to 0.46-fold, respectively. Weight amplified the risk by 0.39- to 0.40-fold for SDB and cognitive outcomes, respectively. Poor ability to perform complex mental processing functions increased the risk of adverse weight and SDB outcomes by 2.9- and 79-fold, respectively. **CONCLUSIONS:** Cognitive functioning in children is adversely affected by frequent health-related problems, such as obesity and SDB. Furthermore, poorer integrative mental processing may place a child at a bigger risk for adverse health outcomes.


**REVIEW SUMMARY:** Review discusses sleep disorders encountered by general pediatricians and pediatric subspecialists in obese pediatric patients and describes evidence linking these disorders to obesity.


**REVIEW SUMMARY:** Noninvasive respiratory support safe and effective in children with acute respiratory failure.

**Monthly Literature Review**

The Monthly Literature Review is created by the ResMed Science Center from abstracts of articles as they appear each month in Medline and highlights the ones of most interest. To receive this review monthly, please email the ResMedScienceCenter@resmed.com.au
## 2013 Calendar of Events

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<tr>
<td>16–17 March 2013</td>
<td>Bangkok, Thailand</td>
<td>Inaugural Asia Pacific Paediatric Sleep Conference</td>
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<tr>
<td>11–13 April 2013</td>
<td>Berlin, Germany</td>
<td>Sleep and Breathing 2013</td>
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<tr>
<td>7–22 May 2013</td>
<td>Philadelphia, USA</td>
<td>ATS 2013 American Thoracic Society</td>
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<td>1–5 June 2013</td>
<td>Baltimore, USA</td>
<td>SLEEP 2013</td>
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<td>7–11 September 2013</td>
<td>Barcelona, Spain</td>
<td>ERS 2013 European Respiratory Society</td>
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<td>28 September – 2 October 2013</td>
<td>Valencia, Spain</td>
<td>WASM 2013 World Association of Sleep Medicine</td>
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<td>Yokohama, Japan</td>
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**AN IMPORTANT NOTE TO YOU, THE READER**

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