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Editorial

Professor Robin Green (Editor)

Professor and Head Department of Paediatrics and Child Health, University of Pretoria

Dear Colleagues

2012 is turning out to be another busy year for all of us. Already this year we have hosted the University of Pretoria, Department of Paediatrics, UPdate at the CSIR, the PMG Congress sponsored by Pfizer in Clarens and both World Allergy Week and World Asthma Day.



Reports on both the Congresses listed above will be published in the next issue

of Paediatric Focus. Every year the World Allergy Organisation hosts World Allergy Week in April and the Global Initiative for Asthma hosts World Asthma Day on the 1st of May. This year both these events were celebrated in South Africa with a number of activities. There were radio and TV interviews conducted by Executive members of the Allergy Society of South Africa and National Asthma Education Programme. These activities were co-ordinated by Lynne Zurnamer of Oz Advertising and PR.

Since both diseases begin in childhood we, as Paediatric doctors, need to be especially mindful of the fact that both allergy and asthma are the commonest chronic diseases in South Africa. They are frequently missed as conditions and children suffer unnecessary symptoms and impaired quality of life as a result. Even when appropriately diagnosed, therapy often fails to deliver on the promise of 'return to normal life'. After all, why would we label a child as having asthma, for example, if not to end his suffering and improve his life to normal? Only normalising of symptoms will do.

I trust you will join me this year, and in perpetuity, of taking good care of your patients with allergic diseases and asthma. Let's get South African kids well again.

Best wishes

Robin J Green

2012 Congresses				
Congress	Location	Date	Contact/URL	
11th International Congress on Paediatric Pulmonology 2012 (CIPP 2012)	Centara Grand and Bangkok Convention Centre, Bangkok	30 June - 2 July 2012	www.cipp-meeting.org/welcome_ welcome_address.htm	
Allergy Society of South Africa Congress 2012 (ALLSA)	13-15 July 2012	The Vineyard Hotel, Newlands, Cape Town	www.allergysa.org 011 447 3876 Clauds.suemc@tiscali.co.za	
21st World Congress of Asthma 2012 (WCA 2012)	Québec City Convention Center, Quebec City, Quebec, Canada	18-21 August 2012	www.wca-2012.com/	
SA Paediatric Association Congress "Bana Pele" 2012	Protea Hotel, The Ranch, Polokwane, Limpopo	22-26 August 2012	www.paediatrician.co.za, 011 676 3464 sapa2012@eventdynamics.co.za	
GP Paediatric Update 2012	Upper East Side Hotel, Woodstock, Cape Town	5-6 October 2012	www.scah.uct.ac.za E-mail: jan.suemc@tiscali.co.za	
American Academy of Pediatrics Conference 2012 (AAP 2012)	Ernest N Morial Convention Center New Orleans	20-23 October 2012	www.aapexperience.org/	
6th Asian Congress of Paediatric Infectious Diseases 2012 (ACPID 2012)	Bandaranaike Memorial International Conference Hall Colombo 7	24-27 October 2012	www.acpid2012.org/	
VACFA - Vaccines for Africa International African Vaccinology Conference	Lagoon Beach Hotel, Cape Town	9-12 November 2012	http://iavc2012.vacfa.com// Bianca.Allison@uct.ac.za,	
Excellence in Paediatrics 2012 (EIP 2012)	Hotel Auditorium Madrid, Madrid, Spain	28 November - 1 December 2012	www.2012.excellence-in- paediatrics.org/node/2023	

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Influenza vaccine in children

Prof. Barry D Schoub MD DSc FRCPath

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nfluenza vaccine is commonly associated with the elderly and the chronically ill. It is only in more recent times that the value of influenza vaccines to the paediatric population has been appreciated and is increasingly being advocated.

The annual South African influenza guidelines, published every year in the February issue of the South African Medical Journal, currently limits its paediatric recommendations to children with underlying illnesses such as pulmonary, cardiac, metabolic and immunosuppressive conditions (including HIV), children on long-term aspirin therapy (because of the risk of Reye's syndrome) and also children who are family contacts of highrisk cases.¹ In the USA, however, the Advisory Committee on Immunisation Practices (ACIP), as far back as 2002, encouraged annual influenza vaccination for all infants between six and 24 months

This was later extended to all infants and children from six months to 18 years and more recently, in 2010, it was further expanded in fact, to all individuals, children and adults, over six months of age.² Paediatric immunisation occupies a prominent role in ACIP recommendations, so much so that even when vaccine is in short supply, it is recommended that children between six and 59 months should be prioritised.² Why has there been this increasing attention, of late, to immunising children against influenza?

Essentially the rationale for paediatric influenza vaccination falls into three groups: -

1. Preventing influenza in children

- a. High-risk children: As is the case for high-risk adults, children with chronic underlying disease are more vulnerable to the severe complications of influenza. This category has long been part of official recommendations, as mentioned above.
- b. Healthy children: Although mortality from influenza in otherwise healthy children is very rare, the overall illness burden from influenza and influenzarelated diseases is considerably greater in children than in adults. Numerous studies have demonstrated excess hospitalisation, doctor visits,

and prescriptions for antibiotics in **3. Immunising children to** otherwise healthy children, especially in infants under two years of age.2-5 So, for example, in the USA from 10 to 19% of medical office visits and 6 to 29% of emergency room visits were due to laboratory confirmed influenza in children less than five years of age during the 2002/3 and 2003/4 seasons.2

Not surprisingly it has been demonstrated in the USA6 and in Finland⁷ that immunising infants and children is both cost-beneficial as well as cost-saving. In addition the benefits of immunising children in order to reduce school absenteeism is significant. Thus it was shown in a USA study that for every 20% increase in the influenza vaccination rate for elementary school aged children there was a 4% decrease in school absenteeism.8

2. Protecting immediate contacts

Local and international influenza guidelines have long recommended that contacts, including healthcare workers as well as family members living under the same roof as high-risk individuals, should be vaccinated annually.^{1,2} Vaccination of childhood contacts is of particular importance given that children constitute the main reservoir of influenza in the community and the most important source of transmission of the virus. Several studies have demonstrated the protective effect of immunising children in order to protect the elderly and other vulnerable family contacts.9-11

This is of particular importance given the relatively poor immunogenicity and effectiveness of influenza vaccination in the elderly and those with underlying medical illnesses. It has been shown that even if only 20% of school aged children are vaccinated, this could have a marked effect in preventing death in the elderly from influenza - in fact even more so than increasing vaccination rates in the elderly, because of limitations in the effectiveness of vaccination in the elderly population and other vulnerable groups.¹²⁻¹⁶

protect the general population through herd immunity

The value of immunising children to convey herd immunity to the population was originally demonstrated in a landmark study in 1968/69 by Arnold Monto and colleagues in a small town called Tecumseh in Michigan, USA.¹⁷ It was shown in that study that by vaccinating 85% of schoolchildren in the town illness rates were reduced threefold in all ages compared to neighbouring towns, even when almost no adults were vaccinated.

The proof of concept for the value for population immunity was demonstrated in Japan which introduced its nationwide school immunisation programme in 1962 and which was subsequently made mandatory in 1977.18 Coverage of 50 to 85% of schoolchildren was achieved in the 70s and 80s which resulted in a reduction in deaths from influenza and pneumonia in the elderly, who rarely received vaccine, by over 10,000 per year. More recently when the programme was stopped, there was a resultant significant rise in mortality in the elderly. Programmes for universal immunisation of children in order to effect herd immunity in the population were subsequently introduced into several populations in the USA¹⁹ and Canada,²⁰ with similar benefits.

The effectiveness of vaccinating healthy children to protect the community, both the healthy as well as the vulnerable who are not able to fully mount an effective vaccine response, is now well established.²¹ The pivotal role of children in the epidemiology of influenza is further strengthened by observations of the effectiveness of school closures during the 2009 H1N1 pandemic.22

Immunising pregnant women to protect young infants

Until relatively recently influenza vaccination was contraindicated in pregnancy, particularly in the first trimester, because of theoretical concerns for potential damage to the foetus. However numerous observations of inadvertently administered vaccine in pregnancy have failed to establish any risk

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from the vaccine either to the mother or to the foetus. Pregnancy itself, however, has been identified to be a major risk factor for complications of influenza especially the later stages of pregnancy.²³ The vaccine is now strongly advocated for all pregnant women irrespective of the stage of pregnancy.^{1,2}

A further benefit of vaccination during pregnancy has recently been demonstrated by showing the protective effect of passively transferred maternal antibodies to the infant.²⁴⁻²⁶

This is particularly important as infants less than six months of age are especially vulnerable to a higher illness burden while, unfortunately, vaccine is contraindicated in this age group. In a recent study in Bangladesh 61 to 93% of infants born to mothers who had received influenza vaccine during pregnancy had protective antibody titres to the two circulating influenza A virus strains.²⁶

Paediatric vaccination schedules:

The schedule for paediatric vaccination is seen in the accompanying table: -

Children 9-12 years	1 adult dose	
Children 3-9years	2 x adult doses*	
who have never, or		
have been incom-		
pletely, vaccinated		
Children 6mo	2 x paed doses **	
3years:	•	
Infants< 6mo.:	Not recommended	

*Two doses separated a month apart are given to young children because of their lack of prior exposure to the virus. The first dose functions as a priming dose followed by the second dose, given a month later, which acts as a booster.

**A paediatric dose is half the adult dose. Influenza vaccines should be given preferably by intramuscular injection rather than subcutaneously.

As with adults the timing of vaccination is ideally placed close to the onset of winter in order for the peak antibody response to coincide with influenza season, but not too late to miss the boat. Thus the optimal timing is March or April.

Barriers to paediatric immunisation

A recent publication lists some 838 reasons associated with under-vaccination of children-45% of them related to immunisation systems, 26% to family characteristics including religious, philosophical and cultural objections to immunisation, 22% to parental attitudes and knowledge and 7% to limits in knowledge and deficiencies in communication.²⁷ Over and above the general obstacles to immunisation, introducing an annual vaccination by injection to at-risk children, let alone healthy children for herd immunity for the population, will require a huge educational drive. Barriers of fear, misinformation and mistrust, not infrequently fuelled by incorrect media messaging,²⁸ will require innovative and imaginative strategising.

Official guidelines for South Africa still only recommend immunising at-risk children as well as direct contacts.¹ It may require more userfriendly routes for immunising children other than the annual injections if we are to extend the spectrum to the general healthy paediatric population. Intensive research for better noninjectable vaccines including improved live attenuated vaccines or vaccines providing more durable immunity may eventually achieve this goal.^{29,30}

References

1. Schoub BD. Recommendations pertaining to the use of viral vaccines: Influenza. S Afr Med J 2012;102:73.

- Fiore AE, Uyeki TM, Broder K, Finelli L, Euler GL, Singleton J. A., Iskander JK, Wortley PM, Shay DK, Bresee JS, Cox NJ. Prevention and control of influenza with vaccines: recommendations of the Advisory Committee on Immunization Practices (ACIP) 2010. MMWR Recomm Rep 2010;59:1-62.
- Heikkenen T, Silvennoinen H, Peltola V, et al. Burden of influenza in children in the community. J Infect Dis 2004;190:1369-73.
- Principi N, Esposito S. Are we ready for universal influenza vaccination in paediatrics? Lancet Infect Dis 2004;4:75-83.
- Terebuh P, Uyeki T, Fukuda K. Impact of influenza on young children and the shaping of United States influenza vaccine policy. Pediatr Infect Dis J 2003:22, S231-5.
- Meltzer MI, Neuzil KM, Griffin MR, Fukuda K. An economic analysis of annual influenza vaccination of children. Vaccine 2005;23:1004-14.
- Salo H., Kilpi T., Sintonen H., Linna M., Peltola V., Heikkenen T. Cost-effectiveness of influenza vaccination of healthy children. Vaccine 2006;24:4934-41.
- King JC, Jr., Beckett D, Snyder J, Cummings GE, King BS, Magder LS. Direct and indirect impact of influenza vaccination of young children on school absenteeism. Vaccine 2012;30:289-93.
- Cohen J. Vaccine policy. Immunizing kids against flu may prevent deaths among the elderly. Science 2004:306, 1123.

- Dushoff J, Plotkin JB, Viboud C., Simonsen L, Miller M, Loeb M, Earn DJ. Vaccinating to protect a vulnerable subpopulation. PLoS Med 2007;4:e174.
- King JC, Jr., Lchenstein R, Cummings GE, Magder LS. Impact of influenza vaccination of schoolchildren on medical outcomes among all residents of Maryland. Vaccine 2010; 28:7737-42.
- 12. Jordan RE, Hawker JI. Influenza in elderly people in care homes. Bmj 2006:333:1229-30.
- Jordan RE, Hawker JI. Influenza vaccine in the over 65s. Bmj 2008;337:a2545.
- Kunisaki KM, Ianoff EN. Influenza in immunosuppressed populations: a review of infection frequency, morbidity, mortality, and vaccine responses. Lancet Infect Dis 2009;9:493-504.
- Simonsen L, Taylor RJ, Viboud C, Miller MA, Jackson LA. Mortality benefits of influenza vaccination in elderly people: an ongoing controversy. Lancet Infect Dis 2007;7:658-66.
- Van Vuuren A, Rheeder P, Hak E. Effectiveness of influenza vaccination in the elderly in South Africa. Epidemiol Infect 2009;137:994-1002.
- Monto AS, Davenport FM, Napier JA, Francis T. Jr. Effect of vaccination of a school-age population upon the course of an A2-Hong Kong influenza epidemic. Bull World Health Organ 1969;41:537-42.
- Reichert TA. The Japanese program of vaccination of schoolchildren against influenza: implications for control of the disease. Semin Pediatr Infect Dis 2002;13:104-11.
- 19. Glezen WP. Herd protection against influenza. J Clin Virol 2006;37:237-43.
- Loeb M., Russell ML, Moss L, et al. Effect of influenza vaccination of children on infection rates in Hutterite communities: a randomized trial. Jama 2010;303;943-50.
- Jordan R, Connock M, Albon E., Fry-Smith A, Olowokure B, Hawker J, Burls A. Universal vaccination of children against influenza: are there indirect benefits to the community? A systematic review of the evidence. Vaccine 2006;24:1047-62.
- Earn DJ, He D, Loeb MB, Fonseca K, Lee BE, Dushoff J. Effects of school closure on incidence of pandemic influenza in Alberta, Canada. Ann Intern Med 2012;156:173-81.
- Dlugacz Y, Fleischer A, Carney MT, et al. H1N1 vaccination by pregnant women during the 2009-10 H1N1 influenza pandemic. Am J Obstet Gynecol 2011.
- Blanchard-Rohner G, Siegrist CA. Vaccination during pregnancy to protect infants against influenza: why and why not? Vaccine 2011;29:7542-50.
- Eick AA, Uyeki TM, Klimov A, Hall H, Reid R, Santosham M, O'Brien KL. Maternal influenza vaccination and effect on influenza virus infection in young infants. Arch Pediatr Adolesc Med 2011;165:104-11.
- Steinhoff MC, Omer SB, Roy E, Arifeen SE, Raqib R, Altaye M, Breiman RF, M, BBSK. Influenza immunization in pregnancy - antibody responses in mothers and infants. N Engl J Med 2010;362:1644-6.
- Bhat-Schelbert K, LIN, CJ, Matambanadzo A, Hannibal K, Nowalk MP, Zimmerman RK. Barriers to and facilitators of child influenza vaccine - Perspectives from parents, teens, marketing and healthcare professionals. Vaccine 2012;30:2448-52.
- Rainey JJ, Watkins M, Ryman TK, Sandhu P, Bo A, Banerjee K. Reasons related to non-vaccination and under-vaccination of children in low and middle income countries: findings from a systematic review of the published literature, 1999-2009. Vaccine 2011;29:8215-21.
- 29. Lambert LC, Fauci AS. Influenza vaccines for the future. N Engl J Med 2010;363:2036-44.
- Osterhaus A, Fouchier R, Rimmelzwaan G. Towards universal influenza vaccines? Philos Trans R Soc Lond B Biol Sci 2011;366:2766-73.

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Feeding preterm infants on cue

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eeding difficulties are expected in preterm infants due to their immature feeding system, including the muscles of the face, mouth and oesophagus. Irrespective of birth weight the preterm infant seems to be at risk for feeding challenges ranging from mild to severe. The most common being gastrooesophageal reflux, constipation due to gastric immaturity and poor feeding endurance with poor weight gain. The aim of this article is to provide an understanding of preterm infant maturation and the relation thereof, to feeding success and challenges.

In the NICU

Preterm infants should be started on oral feeds as soon as possible after birth to ensure development of the lining of the gut and consequently protection against infection. Guidelines on parenteral feeding are available in the document 'Clinical guidelines: nutrition of the premature and low birth weight infant'. Human breastmilk is the feeding of choice, since the benefits of human milk feeds for the preterm infant include host defence, gastrointestinal development, and special nutrition according to preterm infant needs, improving neurodevelopmental outcome and physical and psychological health in the mother. In addition, it has economic and environmental benefits.

Clinical problems may however be experienced by preterm infants with regard to breastfeeding such as insufficient colostrum available to start priming of the gastro-intestinal tract and difficulty in establishing and maintaining a mother's full milk supply. Transitioning to breastfeeding, while maintaining a full milk supply, may further be very challenging.

It is important to ensure early feeding success in the preterm infant, since this is related to long-term breastfeeding success and continuation. Mothers can be supported by a professional lactation consultant, starting skin-to-skin care immediately after birth (even when ventilated), allowing the infant to suckle on the expressed breast before she is ready to fully coordinate suck, swallow and breathing and by encouraging early nutritive breastfeeding.

It is crucial that healthcare professionals understand the physiology of breastfeeding, since expressing of breast milk needs to start within four hours after birth and continue during the first week to ensure the availability of prolactin receptors later in the feeding process and consequently the ability to increase breast milk production. Galactogogues such as meoclopramide, domperidone, fenugreek, prolacting and



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Non-nutritive sucking increases absorption of feeds due to an increase in gastrin secretion, decreased somatostatin secretion and enhanced functioning of the gastro-intestinal tract.

herbal medications are further very useful in increasing breast milk production.

Feeding should be started by means non-nutritive sucking providing of opportunities for the preterm infant. This is the sucking preceding nutritive sucking and is characterised by shorter sucking bursts. Non-nutritive sucking is beneficial to the newborn infant in that it contributes to physiological stability, including higher levels of oxygenation and a decrease in heart rate. It protects against aspiration, since sucking inhibits swallowing and improves glucose utilisation due to an increase in insulin secretion. Non-nutritive sucking increases absorption of feeds due to an increase in gastrin secretion, decreased somatostatin secretion and enhanced functioning of the gastro-intestinal track. Non-nutritive sucking further contributes to faster transition from tube to oral feeds due to an acceleration in maturation, greater weight gain resulting in earlier discharge home and is beneficial in soothing and self-consolation. It increases self-regulatory state modulation with increased levels of alertness and increase time sleeping and finally improving muscle tone and coordination.

To ensure successful oral feeding it is important to provide the preterm infant with non-nutritive sucking opportunities, in order to prevent the infant from losing the sucking reflex. A pacifier should have certain characteristics. In infants that still demonstrate uncoordinated suck-swallow and breathing rhythms the pacifier should be shaped similar to the infant's thumb to promote tongue cupping. It should have a nipple long enough to reach and stimulate the soft palate with a bolus on the end in order to stimulate the limbic system. The mouth shield should be large and soft to stimulate the nerve endings around the mouth and a'handle' on the shield will support the hands in the midline as well as the hands-to-mouth position that supports self-soothing and midline positioning. Once the infant matures enough to coordinate sucking (around 32 weeks) the nipple of the pacifier can be thicker and closer to the size of the mother's nipple, since this is what the infant would be sucking on at this stage.

When to start breastfeeding

Nutritive breastfeeding should be started when the infant shows readiness, where weight and gestational age do not play a role. Feeding maturity is determined by neurological maturity which can be accelerated by starting with human milk feeds immediately after birth and allowing skin-to-skin care even for the ventilated infant. These two interventions contribute to the myelination process of the nervous system resulting in neurological maturity. Some indicators for feeding readiness include sucking well on a finger, fist, pacifier or expressed breast as well as the infant being able to handling her own secretions. The infant should be medically stable but may still be receiving oxygen supplementation. Table 1 indicates readiness to try oral feeding in the preterm infant.

Switching from tube to oral feeding

It is important to understand and apply the neonate's maturation process. Once the infant shows signs of feeding readiness the

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Table 1: Readiness to try oral feeding			
Breathing	 Comfortable, stable breathing (no rib retraction or grunting) Breathing rate of less than 70/min at rest Less than 40% oxygen 		
Heart	Stable, between 120 and 160 beats per minute		
Stomach	Sufficient bowel soundsTolerates tube feeds every 2-3 hours		
Neurological	 Gestational age of more than 30 weeks Coordinates sucking, swallowing and breathing Sucking on the expressed breast or pacifier, mouthing, suck on hands Maintains own body temperature outside incubator (or with KMC) Overall condition is stable Rooting and sucking reflexes sufficient 		
Nutritional	• Gains weight at 15g/kg/day		
State and behaviour	 Maintains quiet alert state; ability to relax; bright, healthy look Shows cues for engagement: mouths "oohs" makes eye contact, moves hands to mouth while mouthing Shows hunger cues - mouthing, rooting, wake for feeds Focuses on food source 		

following regime could be followed. Select the feeding time when the infant is the most awake and try an oral feed. If the feed is successful two oral feeds; one oral, two tube feeds, one oral, two tube feeds should follow the following day. Once the infant can manage this then alternate tube and oral feeds can follow and finally all feeds should be oral feeds.

Each feed can be rated to determine how successful it was. A good feed is noted when the infant latches well, has a good position for feeding, sucks continuously with or without stimulation and swallows frequently. With a good feed no 'top-up' is needed, since the infant takes more than half of the volume of a feed during the first third of the feed. A fair feed is noted when the infant latches and starts sucking, but loses grip and 'fights' on the breast. For fair feeds it is advisable to give half the feed via nasogastric tube. A poor feed is noted when the infant does not latch or suckle at all and therefore the whole volume of the feed is given via nasogastric tube.

Guidelines for preterm infant feeding after discharge

It is important to teach mothers to read their infant's cues with regards to feeding, instead of relying on monitors, such as scales to indicated feeding success. The most important guideline is that mom should hear her baby swallow. The 'premmie' should gain weight on any kind of feed, whether it is formula or breast feeding. Infants may receive supplement feeds if necessary, provided that an experienced lactation consultant has assisted to ensure breast feeding establishment.



Semi-demand feeding is more suitable for preterm infants.These infants reach full oral feeding five days earlier than infants on scheduled feeds.

Hunger cues may be unreliable, therefore the feeding schedule that was used in the NICU should be continued or babies should be woken every three hours during the day until a weight of 2,5kg is achieved. When feeding on demand the 'premmie' should feed at least eight times in 24 hours, which does not necessarily have to be every three hours. If the baby feeds every two to twoand-a-half hours during the day and every four hours at night, this is acceptable, since both mom and baby will get more rest. However, the 'premmie' should not sleep for more than four hours between feeds until a weight of 2,5kg is achieved. Thereafter, sleeping for up to five hours at night to allow both mom and baby their much-needed rest is acceptable. Although scheduled feeding will ensure sufficient caloric intake, demand and semi-demand have been proven to be more effective in ensuring feeding success. Demand or ad lib feedings means that the infant is fed when hungry.

This infant will take fewer feeds per day and will be discharged home 6.2 days sooner than infants on scheduled feeds. They also exhibit more hunger cues and may consume fewer calories in 24 hours. However, there is no difference in weight gain compared to infants on scheduled feeds, due to the longer sleep periods.

Semi-demand feeding is more suitable for preterm infants. In this case the infant is assessed every three hours for behavioural signs of hunger. If the infant is sleeping, reassess 30 minutes later and if the infant is still sleepy give a tube feed. If the infant wakes up and demonstrates hunger signs before the three hours are completed, the feeding can be provided earlier. These infants reach full oral feeding five days earlier than infants on scheduled feeds.

A preterm baby should gain between 142 and 170g per week, but the change of environment from hospital to home has a large impact on energy use. This may influence weight gain in the first week. Weight gain may not occur in the first week at home, but after adaptation, more weight gain than is required may occur in the following weeks. Rather track weight gain over a bi-weekly period.

Supplements

The breastfed baby, weighing less than 1,5kg at birth, should receive a breast milk fortifier until 2kg weight is achieved. The reason for supplementing breast milk is because the mother produces mature milk by the time her premmie is discharged from the hospital and the preterm baby may still need additional proteins for a few months to address



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nutritional needs. Fortification does however have some possible drawbacks including partial compromise of immune function of fresh or frozen human milk, delayed emptying from the stomach, harder stools, hyperosmolality and excessive growth.

The preterm baby on demand breastfeeding should start with unfortified breast-feeding at least one week before discharge to help with feeding transition from hospital to home. Breast-fed infants should also receive a prescribed multivitamin supplement and iron on discharge until 1 year of age, but formula contains preadded supplements, so additional supplementing is unnecessary. When changing from breast-feeding to formula, it is important to remember that supplements, such as iron should be discontinued, since formula milk is already supplemented. Parents should consult with the doctor or dietician when changing from breastfeeding to formula.

Introducing solids

It is suggested that complementary food for 'premmies' should not be introduced before 16 weeks chronological age (post-delivery); that is not prior to when the baby reaches 40 weeks gestational age and weighs at least 5kg. Use the baby's weight at 40 weeks gestation as reference for the introduction of solids.

If solids are started too early, they will decrease the breast milk production due to



The preterm baby is ready for solids when:

- doubled 40-week gestational weight,
- is able to keep head upright without support,
- can touch, hold and taste objects,
- grabs the spoon,
- moves tongue back and forth and does not push it out,
- forms a tight seal around the nipple to lessen dribbling at the sides of the mouth.

less stimulation to the breasts. This could also lead to allergies such as asthma and allergic rhinitis. There might also be a risk for the development of diabetes mellitus, once again if predisposed. Starting solids before 4-6 months may trigger a food allergy in the infant and before 3 months the mouth muscles and digestive systems are too immature to handle solids. Until 6 months (corrected age) milk is the main source of nutrition. Solids are given to supplement the milk and teach the infant new tastes and textures. In the first few weeks, it does not matter how much is eaten, as long as this new skill is learnt, since predominant nutrition will still be derived from milk.

Once weaning has started, proceed according to the usual guidelines recommended for full-term infants.

Additional reading

- Edmond K, Bahl R. Optimal feeding of low-birth-weight infants: Technical review. World Health Organization. 2006. India.
- 2. Farley T. Oral Feeding success in the preterm infant NICUniversity.org NANN Lecture Details. Available from http://www.nicuniversity.org/
- 3. Lubbe W. Prematurity Adjusting your dream. Little Steps. 2008. Pretoria.
- Metropole Pediatric Interest Group: Western Cape. Clinical guideline: Nutrition of the premature and low birth weight infant. 2007. Tygerberg Hospital.
- Pinelli J, Symington A. Non-nutritive sucking for promoting physiologic stability and nutrition in preterm infants. Cochrane Library 2003; 3.
- Tosh K, McGuire W. Ad libitum or demand/semi-demand feeding versus scheduled interval feeding for preterm infants. Cochrane Library 2006; 3.

In the next issue of Paediatric Focus: Reportback: The Paediatric Management Group Conference 2012, Clarens





Photos by Dr Humphrey Lewis

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What do we know about serotype 6A and 19A, and how is this of importance for South Africa?

Dr Claire von Mollendorf

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here are 91 distinct serotypes of Streptococcus pneumoniae,^{1,2} but only 20 of these serotypes, including 14, 1, 6A, 6B, 3, 7F, 23F, 18C, 19F and 9V, account for more than 80% of invasive pneumococcal disease (IPD) globally. The distribution of these serotypes differ to some degree by region and age group.² Changes in serotypes have also occurred in countries who have introduced pneumococcal conjugate vaccines into their immunisation programmes. Antibiotic resistance in S. pneumoniae is related to the so called 'paediatric serotypes' (6A, 6B, 9V, 14, 15A, 19F, 19A, and 23F) which commonly colonise young infants. The prevalence of these resistant serotypes are higher in HIV infected children³ and adults.4;5 HIV has also increased the burden of pneumococcal disease in South Africa over the last 20 years.6

Surveillance in South Africa

South Africa has a long-standing national active laboratory-based surveillance programme coordinated by the Group for Enteric, Respiratory and Meningeal disease Surveillance in South Africa (GERMS-SA) since 2003. GERMS-SA is a collaborative effort between the National Institute for Communicable Diseases (NICD), participating South African universities, clinical microbiology laboratories and external funding agencies. The case definition for IPD included in the surveillance programme is S. pneumoniae identified from normally sterile-site specimens diagnosed at designated centres through laboratory testing, namely culture positive or latex agglutination test positive with an additional confirmatory test.7

Pneumococcal National Surveillance was originally started in South Africa in 1979 and was later extended in 1999 to include Haemophilus influenzae and Neisseria meningitidis surveillance. In 2003 enhanced surveillance sites, with dedicated surveillance officers, were established and these sites increased incrementally from 10 sentinel sites in 2003 to 15 in 2006 and 25 sites from 2008 onwards. In 2010 more than 200 clinical microbiology laboratories participated in the GERMS-SA surveillance programme covering a population of around 50 million.8

South African data

In 1977, pneumococci fully resistant to penicillin were first described in South Africa⁹ and in 1978 multiresistant strains were reported.^{10;11} In an analysis of 4766 pneumococcal isolates from 1979 to 1986, 92% of penicillin-resistant strains and all the multi-resistant strains (n=98) were from serogroups 6 (serotypes 6A and 6B) and 19 (predominantly 19A) or serotype 14.12;13 PROTEKT surveillance study data (2001-2004) from South Africa described penicillinnon-susceptibility rates of 74% for respiratory pneumococcal isolates.¹⁴



Baby Sophia, photo by Dr Eitzaz Sadia

Prior to the introduction of **PCV7** into the South African **Expanded Programme for Immunisation (EPI) the** seven serotypes in this vaccine were responsible for more than 60% of IPD and 80% of penicillinresistant isolates in children < 5 years of age.

Prior to the introduction of the 7-valent pneumococcal conjugate vaccine (PCV7) into the South African Expanded Programme for Immunisation (EPI) in April 2009, the seven serotypes (4, 6B, 9V, 14, 18C, 19F, and 23F) in this vaccine were responsible for more than 60% of IPD (70% with 6A) and 80% of

penicillin-resistant isolates in children less than 5 years of age.^{15;16} Other frequent serotypes included 6A (4th), 19A (6th) and 1 (7th). It was assumed that PCV7 would cover serotypes 6A and 19A through the cross-reactivity of antibody responses between structurally similar serotypes.¹⁷ In practice, however, cross-reactivity has only been demonstrated between serotypes 6B and 6A and not between 19F and 19A.18-20

In 2010 overall disease rates in children less than 5 years decreased significantly (n=647 in 2010 compared to n=1010 in 2009, p<0.001) with all seven serotypes in PCV7 showing a decrease in numbers. In 2010 only 50% (323/647) of IPD in children less than 5 years was caused by PCV7 serotypes and 13% (81/647) by serotype 6A, while 58% (377/647) of IPD would have potentially been prevented by the 10-valent pneumococcal conjugate vaccine (PCV10) and 82% (530/647) by the 13-valent vaccine (PCV13). Serotype 19A remained important and ranked 4th overall in terms of absolute numbers. Penicillin non-susceptible isolates remained stable at approximately 40% with ceftriaxone non-susceptibility of 8% in all IPD cases. Most of the ceftriaxone-resistant isolates were serotypes contained in PCV7.8

International data

Prior to PCV7 introduction into immunisation programmes, the most common resistant serotypes globally were vaccine serotypes, 6B, 9V, 14, 19F, 23F, and the non-vaccine serotypes, 6A and 19A.²¹ In the US in 1998 the 'paediatric serotypes' accounted for 91% of all penicillin-nonsusceptible pneumococci.22 After PCV7 was licensed for use in young children in the US in 2000, the incidence of IPD in children ≤ 5 years and adults ≥ 65 years of age decreased,23;24 while non-PCV7 serotypes like 19A increased from 20.4% in 2004 to 43.7% by 2008.24 This change in nonvaccine serotypes was also mirrored in other countries post-PCV7 introduction and is the most constant finding amongst countries that have introduced PCV7 into their national immunisation programme.²¹ Reasons for the increase in 19A are multiple and include:

- the ability of 19A to equally result in colonisation invasive disease or otitis media
- the fact that 19A was a common cause of invasive disease and carriage pre-vaccine

ediatric DCUS

Prevenar 13

introduction

• the high non-susceptibility of 19A to penicillin and other antibiotics.²⁵

It is important to note that an increase in the incidence of serotype 19A was noted in some countries pre-PCV7 introduction. The South Korean surveillance programme showed an 18% increase in 19A in children less than 5 years before PCV7 introduction in November 2003.²⁶ In Taiwan serotype 19A also increased while only 15.9% of children had received PCV7 vaccination.²⁷ The increase in the incidence of IPD caused by serotype 19A was also associated with an increase in antibiotic resistance in this serotype.²⁸

Serotype replacement

Serotype replacement in invasive disease is defined as an increase in the incidence of IPD caused by non-vaccine-type (NVT) serotypes after vaccine introduction. Most of the data on replacement is from observational studies and is expected to be more marked in wellvaccinated populations.²⁹ Studies often report a lag between vaccine introduction and an increase in IPD caused by NVTs.^{30;31} The lag is ascribed to the time taken for full vaccine coverage to be reached in the population and for vaccine serotypes to be eliminated.^{25;32} Most studies report a net decline in disease of 40-60% in children post-PCV7 introduction even after accounting for replacement, although many of these studies have short post-vaccine follow-up periods.33

Impact of higher valency vaccines

Most of the current published data reflects the changes in pneumococcal serotypes post-PCV7 introduction. PCV7 has been superceded by PCV10 (licensed in 2009) and PCV13 (licensed in 2010). The additional serotypes in PCV10 are 1, 5 and 7F, while PCV13 contains additional serotypes 1, 3, 5, 6A, 7F and 19A.

The Health Protection Agency (HPA) in England and Wales has surveillance which reports cumulative weekly numbers of invasive pneumococcal disease according to serotypes in PCV7, serotypes not in PCV7, serotypes not in PCV13; and serotypes in PCV13 but not in PCV7. In 2010 HPA data showed an overall reduction in disease in children less than 5 years despite significant serotype replacement post-PCV7 introduction, with no initial herdimmunity effect. Following the introduction of PCV13 into the immunisation programme in April 2010, an evaluation of the first 15 months of use showed protection against serotypes 7F and 19A and a 50% reduction in IPD cases in children less than 2 years due

to one of the additional serotypes covered by PCV13. $^{34;35}\!$

In a pneumococcal carriage study of children with acute otitis media in France, PCV13 was shown to have an impact on overall carriage, as well as on serotypes 19A, 7F, and 6C. There was no significant increase of nonvaccine PCV13 serotypes.³⁶

Conclusion

Several factors have influenced pneumococcal epidemiology in the last decade. These include the natural fluctuations of pneumococcal serotypes, the extensive use of antibiotics and the introduction of PCV7 into immunisation programmes.^{37,38}

In South Africa we have ongoing active IPD surveillance which is vital to monitor changes in serotypes and pneumococcal resistance with the use of conjugate vaccines.

In South Africa, serotypes 6A and 19A were important serotypes pre-PCV7 introduction in 2009 and have remained important in the immediate period post-PCV7 introduction. Reductions in vaccine serotypes have been noted in South Africa since 2009, but it is still too early to assess serotype replacement effects. In addition PCV13 replaced PCV7 in the South African EPI programme in June 2011 and this vaccine contains serotypes 6A and 19A which will also influence serotype changes. In South Africa we have ongoing active IPD surveillance which is vital to monitor changes in serotypes and pneumococcal resistance with the use of conjugate vaccines.37;39;40 It is also important for us to scrutinise data from other countries with longer-term use of conjugate vaccines to try and anticipate potential changes.

References:

- (Park IH, Pritchard DG, Cartee R, et al. Discovery of a new capsular serotype (6C) within serogroup 6 of Streptococcus pneumoniae. J Clin Microbiol 2007;45:1225-1233.
- Lynch JP 3rd, Zhanel GG. Streptococcus pneumoniae: epidemiology, risk factors, and strategies for prevention. Semin Respir Crit Care Med 2009; 30:189-209.
- Madhi SA, Petersen K, Madhi A, Wasas A, Klugman KP. Impact of human immunodeficiency virus type 1 on the disease spectrum of Streptococcus pneumoniae in South African children. Pediatr Infect Dis J 2000; 19(12):1141-1147.
- 4. Crewe-Brown HH, Karstaedt AS, Saunders GL, Khoosal M, Jones N, Wasas A, et al. Streptococcus pneumoniae

blood culture isolates from patients with and without Human Immunodeficiency Virus infection: Alterations in penicillin susceptibilities and in serogroups or serotypes. Clinic Infecti Dis 1997; 25:1165-1172.

- Klugman KP, Welsh KJ. Nasopharyngeal carriage of pneumococcal pediatric serotypes: A risk for acute and recurrent otitis media in children and for invasive disease in susceptible adults. Jour Infecti Dis 2005; 191:1790-1792.
- Koornhof HJ, Madhi SA, Feldman C, von Gottberg A, Klugman KP. A century of South African battles against the pneumococcus -'the captain of death'. South Afr J Epidemiol Infect 2009; 24(4):7-19.
- Y. National Institute for Communicable Diseases (NICD) of the National Health Laboratory Service (NHLS). Communicable Diseases Surveillance Bulletin. May 2006
- NICD. Group for Enteric, Respiratory and Meningeal disease Surveillance in South Africa. GERMS-SA Annual Report 2010.
- Appelbaum PC, Bhamjee A, Scragg JN, Hallett AF, Bowen AJ, Cooper RC. Streptococcus pneumoniae resistant to penicillin and chloramphenicol. Lancet 1977; 2:995-997.
- Jacobs MR, Koornhof HJ, Robins-Browne RM, Stevenson CM, Vermaak ZA, Freiman I et al. Emergence of multiply resistant pneumococci. N Eng J Med 1978; 299:735-740.
- 11. Klugman KP. Pneumococcal resistance to antibiotics. Clin Microbiol Review 1990; 3(2):171-196.
- Koornhof HJ, Wasas A, Klugman KP. Antimicrobial resistance in Streptococcus pneumoniae: A South African Perspective. Clin Infecti Dis 1992: 15:84-94.
- Klugman KP, Koornhof HJ. Drug resistance patterns and serogroups or serotypes of Pneumococcal isolates from cerebrospinal fluid or blood, 1979-1986. J Infecti Dis 1988; 158 (5):956-964.
- Linares J, Ardanuy C, Pallares R, Fenoll A. Changes in antimicrobial resistance, serotypes and genotypes in Streptococcus pneumoniae over a 30-year period. Clin Microbiol Infect 2010; 16:402-410.
- 15. NICD. Group for Enteric, Respiratory and Meningeal disease Surveillance in South Africa. GERMS-SA Annual Report 2008.
- 16. Silberbauer EI, Ismail N, von Gottberg A, Hoosen AA. Serotype and antimicrobial profile distribution of invasive pneumococcal isolates in the pre-vaccine introduction era in Pretoria, South Africa, 2005 through 2009. Diagnostic Microbiology and Infectious Disease 2011; 71:309-311.
- Jakobsen H, Sigurdsson VD, Sigurdardottir S, Schulz D, Jonsdottir I. Pneumococcal serotype 19F conjugate vaccine induces cross-protective immunity to serotype 19A in a murine pneumococcal pneumonia model. Infect Immun 2003; 71 (5):2956-2959.
- Pelton SI, Huot H, Finkelstein JA, Bishop CJ, Hsu KK, Kellenberg J et al. Emergence of 19A as virulent and multidrug resistant pneumococcus in Massachusetts following universal immunization of infants with pneumococcal conjugate vaccine. Pediatr Infect Dis J 2007; 26 (6):468-472.
- Whitney CG, Pilishvili T, Farley MM, Schaffner W, Craig AS, Lynfield R et al. Effectiveness of seven-valent pneumococcal conjugate vaccine against invasive pneumococcal disease: a matched case-control study. Lancet 2006; 368 (9546):1495-1502.
- Yu X, Gray B, Chang S, Ward JI, Edwards KM, Nahm MH. Immunity to Cross-Reactive Serotypes Induced by Pneumococcal Conjugate Vaccines in Infants. J Infect Dis 1999; 180:1569-1576.
- Song JH, Dagan R, Klugman KP, Fritzell B. The relationship between pneumococcal serotypes and antibiotic resistance. Vaccine 2012; 30:2728-2737.
- Whitney CG, Farley MM, Hadler J, et.al. Increasing prevalence of multidrug-resistant Streptococcus pneumoniae in the United States. NEJM 2000; 343:1917-1924.

References 23-40 on request.

Prevenar (13)



Reportback:

8th International Symposium on Pneumococci and Pneumococcal Diseases (ISPPD), Iguaçu Falls, 11-15 March 2012

 he 8th ISPPD was held in the town of Iguaçu Falls on the border between
 Brazil and Argentina. The town was named after the famous waterfalls that
 have recently been voted as one of
 the seven new natural wonders of the world.
 Most people will know the falls from the movie
 "The Mission" starring Robert De Niro, in the lead role, with the haunting score by Ennio
 Morricone. As visitors we were able to experience the falls first hand in an inflatable boat. The experience left us soaked but really awestruck with the greatness of the falls

Another fairly close attraction, elected as one of the seven modern wonders of the world, is the Itaipu Dam. The hydroelectric power plant situated here is the largest in the world. Built on the Paraná River near the border between Brazil and Paraguay, the plant generates a power of 12,600 megawatts. This represents 25% of the electricity consumed by Brazil and 95% of the energy consumed by Paraguay.

The town of Iguaçu Falls is unassuming and appears to have gotten stuck in the eighties. It is currently almost exclusively dependant on tourism to survive. The people of the region, including the neighbouring Argentinian town of Puerto Iguazú, are poor. Communication was problematic with only the exceptional cab driver being able to understand rudimentary English. This, however, did not detract from the enjoyable experience we had.

The conference was held in the Rafain Palace Hotel and Convention Center, with delegates being housed in different locations around town. South Africa was well represented with most of the delegates coming from the National Institute for Communicable Diseases.

Nine main themes

Nine main themes were addressed at the conference:

- Global Action Plan for Pneumonia Prevention and Control (GAPP): Tactics and tools for a global campaign against pneumonia
- Progresses and innovations in aetiological diagnosis of *S. pneumoniae* infections and nasopharyngeal colonisation
- Advanced epidemiology of pneumococcal infections and antimicrobial resistance
- Seven lessons from PCV-7. A tale of 4 countries
- Effectiveness of PCV introduction in middle and low-income countries
- S. pneumoniae biology and evolution

- Who gets pneumococcal disease and why?
- Immunology of pneumococcal infections and vaccines
- Novel pneumococcal vaccines and their regulatory pathways

Advocacy and prevention

During the first part of the conference advocacy and plans for pneumocococcal disease prevention were discussed. In 2006, the Sabin Vaccine Institute created the Pneumococcal Awareness Council of Experts (PACE), a group of 21 of the world's leading experts on infectious diseases and vaccines, to raise awareness about the disease and to advocate for its prevention. Orin Levine and Ciro de Quadros informed us of the lessons that they have learned during their five years of advocacy. Methods used to accomplish this goal included hosting high profile events; placing commentaries in global news outlets; convening meetings with government officials and publishing studies on pneumococcal disease and its impact. Since PACE's inception, 67 countries have introduced pneumococcal vaccine into their national immunisation programmes, a number of them in part due to PACE advocacy efforts. Much work still remains to be done. They stressed that reliable, evidence based data is critical to decision-making, supporting the notion that independent voices make for the most credible spokespersons.

Diagnosis

The conference then continued with a look at progress in the diagnosis of pneumococcal disease. Available data suggest that we underestimate the burden of pneumococcal pneumonia. Several speakers discussed newer diagnostic methods such as serology, urinary antigen testing and molecular methods on sterile and respiratory samples. Real-time polymerase chain reaction (rtPCR) provides guantification of bacterial DNA loads and correlates with quantitative culture results. Colonisation densities seem to distinguish pneumonia from asymptomatic carriage. A different hypothesis assumes that the bacterial-to-leukocyte ratio is higher in pneumonia than asymptomatic colonisation. Other investigations that hold promise for the future are multi-detection assays including multiplex rtPCR, mass spectrometry and microarrays.

On the question of why people get pneumococcal disease several presentations concentrated on the role of nasopharyngeal colonisation. The Dutch group from Utrecht demontstrated that both bacteria and respiratory viruses are

Professor Theuns Avenant Department of Paediatrics Kalafong Hospital, Pretoria

abundantly present in the nasopharynx of otherwise healthy young children. Furthermore, they demonstrated distinct associations between bacteria, viruses and environmental factors. Debby Bogaert discussed the fact that this cocolonisation, however, is also a prerequisite for consecutive infections. Her group hypothesised that disturbances in this equilibrium due to, for instance, new acquisitions of bacteria and viruses, antimicrobial therapy, vaccination and environmental factors may play a major role in susceptibility to consecutive infections. A better understanding of the dynamics of the nasopharyngeal microbiota and its interplay with host and environment may give us more insight into pathogenesis of respiratory diseases.

Although some knowledge exists on the interrelationship of *S. pneumoniae* with *H. influenzae* and *S. aureus*, data presented by Shiri and colleagues underscore the synergy between *S. pneumoniae* and *H. influenzae* and the antagonistic effect between *S. aureus* and *S. pneumoniae* colonisation. They warn that monitoring the impact of pneumococcal immunisation on the ecology of colonisation is warranted.

Cynthia Whitney of the Centers for Disease Control and Prevention's group presented the first results of the impact of PCV 13 in the United States of America. Rates of invasive pneumococcal disease in children who have received PVC 13 were 7.2/100 000 for the first quarter of 2011, much decreased from 27/100 000 for comparable periods in the years 2006 to 2008.

Some of the first results of the COMPAS trial, exploring the efficacy of PHiD-CV in Argentina, Colombia and Panama, were also presented. After overcoming multiple research challenges, they demonstrated a 26% efficacy against WHOdefined community-acquired pneumonia and a 7% reduction on suspected clinical pneumonia.

Eagerly awaited data on the effect of the introduction of PCV 7 into the South African immunisation schedule were presented by Ann von Gottberg and Cheryl Cohen from the NICD. Ann looked at trends in pneumococcal disease from 2005 to 2011 and concluded that the incidence of invasive pneumococcal disease decreased from 24/100 000 population to 9/100 000 in children under 2 years of age. The effect on HIV infected children were however mainly due to the improvements made in HIV management rather than vaccination.



Cheryl presented the preliminary results of the large case control study investigating the effectiveness of PCV 7 in South Africa. It revealed that PCV 7 is protecting HIV-uninfected children in South Africa. Unfortunately we may have to rethink our vaccination strategy in HIV infected children, where the vaccine does not seem to be effective.

Replacement of serotypes

An ever present concern is replacement of serotypes in pneumococcal disease. Daniel Feikin presented data showing that serotype replacement is a reality but that most of the replacing serotypes after the introduction of PCV 7 were contained in the PCV 13 vaccine. There is however still a large net benefit of using PCV 7 vaccine by reducing overall invasive pneumococcal disease in multiple countries.

To counteract the effect of potential serotype replacement all eyes are currently fixed on the development of a protein-based pneumococcal vaccine.

Early studies of investigational vaccine formulations containing pneumococcal proteins, with or without pneumococcal polysaccharide conjugates, showed that these vaccines were well tolerated by adults and toddlers. Proteins were immunogenic and did not seem to alter the immune responses elicited by conjugates when given as a combined formulation in toddlers. This promises well for the eventual development of a successful vaccine.

Social events included the opening ceremony, "Sputum" soccer cup match and "Fiesta Latino americana" banquet dinner.

At the opening ceremony the Itaipú Choir performed a suite of worldwide popular Latin American songs, followed by a special presentation of music from Chilean band Bordemar and images of the Deep South. The friendly game of soccer that takes place regularly at ISPPD meetings was held between conference participants divided into a Latin American and Rest of the World team. This proved to be a most enjoyable afternoon for the team members as well as supporters.

The banquet dinner held at the Rafain Palace was followed by a show that took us on a journey through the cultures of Paraguay, Argentina, Chile, Peru, Mexico, Cuba, Bolivia, Uruguay and Brazil. The group of over 50 people, including musicians and dancers entertained us late into the night.

The ISPPD Board selected Professor Keith Klugman as the 6th Robert Austrian Lecturer for ISPPD-8. Previous lectures were given by the "Who's Who" in the pneumococcal world including Mathu Santosham, (2006), Alexander Tomasz (2008) and Ron Dagan (2010).







Photo acknowledgement: www2.kenes.com/ISPPD/Symposium/Pages/PhotoGallery.aspx

Professor Klugman still spends 25% of his time as co-director of his MRC Research Unit in Johannesburg. He gave a brief but absorbing summary of developments in the pneumococcal field.

Robert Austrian Research Awards in Pneumococcal Vaccinology

Robert Austrian Research Awards in Pneumococcal Vaccinology are given to deserving young scientists of each continent to support their various research projects into the prevention of pneumococcal disease. This year the \$25,000 USD award for Africa went to Mignon du Plessis of the NICD in Johannesburg for her project "Molecular epidemiology of





Streptococcus pneumoniae serotype 1 in adults and children, pre- and post-PCV13 introduction". She is expected to present results of her projects at ISPPD-9 in 2014.

We are all looking forward to this symposium to be held in Hyderabad, India. It promises to answer some of the burning questions currently being researched.

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Ref 1. Dagan R, Klugman K.P. Impact of conjugate pneumococcal vaccines on antibiotic resistance. Lancet Infect Dis. 2008;8: 785-95