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References:
1. ULTIBRO BREEZHALER. Local Prescription Information November 2016.

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## Contents

### President Message
- New Year Message from the President
  Dr Raymond SK LO
  2

### Editorial
- Editorial
  Dr Jane CK CHAN
  4

### Guest Editorial
- Air Pollution is a Health Issue
  Ms Christine LOH, OBE, JP
  5

### Medical Bulletin
- Air Pollution and PM$_{2.5}$ Health Exposure in Hong Kong
  Dr Alexis KH LAU & Dr Ying LI
  7
- Air Pollution and Child Health
  Dr Alfred TAM
  10
- Air pollution: Its impact on adult patients with respiratory conditions
  Dr Jane CK CHAN & Dr Fanny WS KO
  12
- MCHK CME Programme Self-assessment Questions
  15
- Air Pollution and Cardiovascular Disease
  Dr Archie Ying-sui LO
  17
- Is Air Pollution Carcinogenic ??
  Dr George Tak-jor AU
  21
- The Mission and Vision of the Air Quality Health Index
  Prof Tze-wai WONG & Ms Andromeda HS WONG
  24
- Improving Air Quality of Hong Kong
  Mr Sik-wing PANG
  27

### Medical Bulletin
- To be a smart citizen in health protection and combating air pollution
  Ms Sum-yin KWONG
  29
- How to fight air pollution: The London experience
  Prof Frank J. KELLY
  31

### Life Style
- 孟澈雅製：現代竹木文玩
  何孟澈醫生
  34

### Dermatological Quiz
- Dermatological Quiz
  Dr Lai-yin CHONG
  30

### Medical Diary of January
  36

### Calendar of Events
  37

### Federation News
  38

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**The Cover Shot**

**The aesthetic kapok**

The three red kapok trees in Guangdong, Dongguan, Humen Town are over three hundred years old since the Qianlong period of the Qing dynasty.

I have been taking pictures of them on several occasions in 2005-2006. The results were unsatisfactory due to strong light conditions.

I visited them again in 2007. It was raining heavily the night before. When I arrived at the site, the sky was overcast. The mood was entirely different. The tree barks were soaked with rain-water and became dark & shiny. The red kapok flowers & the dark tree barks were so contrasting yet in good harmony.

Using the double exposure technique, the final image turned out to be a beautiful Chinese ink & colour painting.

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**Dr Leo KK WONG**
FRCP(Lond., Edin., Glasg.), Hon.FRPS, Hon.PSA, MFIAP

**The aesthetic kapok**

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New Year Message from the President

Dr Raymond SK LO

President
The Federation of Medical Societies of Hong Kong

On behalf of the Federation of Medical Societies of Hong Kong, may I first of all wish you a happy, healthy and fruitful new year. The year 2015 is especially meaningful for the Federation, as it marks our 50th anniversary. The Federation was established in the year 1965, and we owe much to our predecessors, present colleagues, members as well as partners from different fields, for the unfailing support and advice to help build our Federation to the current status and strength.

The year 2014 had not been easy for fellow citizens of Hong Kong, with events that had affected every strata of our society. While the Federation will remain apolitical, we will not shy away from situations that call for our efforts to uphold the health and well-being of our professionals and the public. Last October, we had issued a press release reminding the public of the dangers and safety measures of mass gathering. We organised a public talk on Depression and Suicide Prevention in the mental health month of November. We also organised in December a symposium on Family Cohesion-Caring for the Younger Generation for health and educational professionals, in response to the needs for our future generations. We look forward to further collaboration with members and partners, in offering more educational events in our anniversary year.

As the umbrella organisation of medical, dental, nursing and allied health societies of Hong Kong, the Federation endeavours to accomplish our missions. To date, our membership has increased to 135 professional societies, with two new associate members, namely the Hong Kong Clinical Psychologists Association and the Hong Kong Disaster Medicine Association. We hope to continue our best support in promoting the networking and fraternity of our members. The Beijing and Shenzhen study visits held last year were a good success, and our recreational outings were popular. More activities will be arranged this year.

On the advocacy role, the Federation will rely on your concerted efforts. Last year we surveyed the public on the needs of our elderly, in conjunction with our annual scientific meeting on Care for Our Older Population. We hope to follow on with further consultations and feedback. On the charity side, our Foundation will soldier on with various ongoing projects. For our bereaved children support programme, theatre sports and drum circle event were well received. We welcome further suggestions and initiatives. As for our publications, the Medical Diary this year will cover various global health concerns, with this rich January issue setting an excellent start.

The year 2015 will be a memorable milestone of the Federation. A series of celebratory activities are already being planned, notably the 50th Anniversary Gala Dinner at the Hotel Intercontinental on Sunday, March 15th. Continuing growth of the Federation will rely on your advice and participation. Thank you in anticipation of your support, and the Federation will strive to serve the finest for our health professions.
Let's join together to reverse Hong Kong's air pollution!

Number of days with API exceeding 100

Source: Environmental Protection Department, Hong Kong

Use private cars less often
Encourage greening of our city
Encourage cycling where and when it is safe

Yes, we can!
The year 2015 has arrived and soon we will also be welcoming the Year of the Sheep. This January issue of the Medical Diary will arrive at your doorsteps together with warm greetings from our Federation President Dr Raymond Lo and his cabinet, as well as from me as issue editor. May the New Year bring you happiness, good health, prosperity, and peace as always.

I would ask for your indulgence in tolerating/accepting a few “deviations from the norm” in this issue’s layout, deviations that our Chief Editor Dr CO Mok has kindly condoned. Firstly, I have carved out a section entitled “Guest editorial” specifically for Ms Christine Loh, Under Secretary for the Environment, so that the medical community can get a glimpse of the wisdom of our policy leader. Secondly, the pages for this issue have far exceeded the usual quota, and such would have been inexcusable till we take a good look at this issue and decide that our issue can proudly stand as a compendium on air pollution for the medical as well as lay community. Last but not least, the Life Style section has been written in Chinese by 何志堅醫生, who has graced our issue with its talent in and passion for Chinese literature, art and scholar’s objects. My deep gratitude goes to all authors who have poured their hearts to support this issue.

Despite this being an auspicious time embracing our new year, I would highlight a recently released epic science fiction film, “Interstellar”, which depicts the end of the Earth. The movie tells how humans struggled to look for habitable planets in outer space while humanity is being driven to near extinction by resource exhaustion and bad air, sending a chilly reminder to us all that the Earth can one day become uninhabitable if we keep on abusing it. In the real world, ground-breaking commitment was secured, in the November 2014 APEC summit in Beijing, from China, the world’s biggest polluter reportedly, to set greenhouse gas emission targets for the first time unilaterally. This Issue is also blessed with cross-disciplinary collaboration of our medical community with our Government, the academia, and the non-governmental organisations. This Issue is an attempt to capture and to expand the spirit of cross-disciplinary collaboration of our medical community with our Government, the academia, and the non-governmental organisations. This Issue is an attempt to capture and to expand the spirit of cross-disciplinary collaboration of our medical community with our Government, the academia, and the non-governmental organisations. This Issue is an attempt to capture and to expand the spirit of cross-disciplinary collaboration of our medical community with our Government, the academia, and the non-governmental organisations. This Issue is an attempt to capture and to expand the spirit of cross-disciplinary collaboration of our medical community with our Government, the academia, and the non-governmental organisations. This Issue is also blessed with cross-disciplinary collaboration of our medical community with our Government, the academia, and the non-governmental organisations.

Grateful acknowledgement goes to the Hong Kong Institute of Allergy, the organiser of the recent Hong Kong Allergy Convention, as well as the Hong Kong Thoracic Society, the Hong Kong Society of Paediatric Respiratory (HKSPR), and the Hong Kong Association of Allergy (HKAA); the 4 societies joined hands during the 2014 Allergy Convention in organising a press conference on air pollution, during which the recent U.K. findings by Prof Alfred Tam of HKSPR, Dr Marco Ho of HKAA, as well as Ms Christine Loh. Air scientists from both the Government and academia were in attendance. Dr Marco Ho of HKAA, as well as Ms Christine Loh. Air scientists from both the Government and academia were in attendance.

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Air Pollution is a Health Issue

Ms Christine LOH, OBE, JP
Under Secretary for the Environment

The people are impatient. They want much improved air quality. Over the years, they have demanded that the Government take aggressive action. Health professionals have remained silent on the whole with a few notable exceptions. The time has come for you to help change the public mindset that air pollution is not just an environmental issue but a major public health issue.

The people complained they have suffered serious air pollution for long enough. The historical facts are clear enough. Air quality improved steadily deterioration through the 1990s reaching a high point in 2003. Since then, there has in fact been a gradual turnaround although the overall levels of pollutants are still very high when seen against the World Health Organization’s Air Quality Guidelines (WHOAQG).

Our A Clean Air Plan for Hong Kong, published in March 2013, makes it clear that even if Hong Kong emits no air pollutants, our air quality will still not meet the WHOAQG. This is because Hong Kong shares the same air-shed with the Pearl River Delta, which has a population of nearly 60 million people and is a major industrial area with high emissions.

Hong Kong and Guangdong have a unique relationship in air quality management. Since 2000, the two sides agreed to set emissions reduction targets, and we are now at the second round of targets from 2012 to 2020 with a review after 2015. There has been a gradual improvement in regional air quality in the past decade because of the efforts of both Hong Kong and Guangdong in pollution reduction. The challenge is to do more and do it faster.

Fortunately, since 2013 the national and Guangdong provincial governments are redoubling their efforts to fight air pollution with Guangdong putting forward a wide-ranging plan to meet the target of reducing PM2.5 by 15% by 2017. Hong Kong and Guangdong is collaborating on air science, reducing shipping emissions and working with the manufacturing sector to adopt cleaner production technologies.

Despite high regional air emissions, this does not absolve Hong Kong from doing its utmost to reduce its own pollution because local emissions have the greatest health impact on local residents. This is especially true for emissions from vehicles in busy urban areas, where the health impact of the pollutants is compounded by the phenomenon of ‘street canyons’ where buildings trap pollutants. This is the reason why we have focused much of our recent firepower at reducing vehicular emissions through three schemes. These relate to mandatory replacement of 82,000 pre-Euro IV diesel commercial vehicles by 2020, replacing the catalytic converters for over 20,000 LPG-powered taxis and public light buses within six months from October 2013 to March 2014, and fitting selective catalytic reduction devices on about 1,400 older franchised buses, which will be completed by early 2017. Each of them is designed to reduce specific pollutants. Seen as a whole, Hong Kong’s effort to clean-up its commercial fleets of vehicles represent one of the largest, if not the largest, in the world at a cost of about HK$12 billion.

Other aggressive measures include mandating ocean-going vessels to switch to using a cleaner fuel while at berth in Hong Kong (within 2015), mandating a cleaner marine fuel to be sold in Hong Kong (done in 2014); and to reduce the use of coal in local electricity generation by 2020 by using more natural gas. Our mandatory efforts in dealing with marine emissions are the most aggressive among port cities outside North America and Europe.

While the above represent a package of highly effective end-of-pipe emissions reduction efforts, we have also been relooking at how we manage pollution by managing not only reducing pollution but also population exposure. However, this involves separating people from pollution sources, such as redirecting traffic in urban centres, creating pedestrians-only areas or reducing the number of vehicles allowed into busy urban areas, such as low emission zones. These efforts require the collaboration not only within the government but also with the transport services operators and district interests since it requires significant behaviour change and acceptance of what may be regarded as ‘inconvenience’.

The medical and health sector can help. Health professionals are in the best position to articulate the benefits from exposure reduction. At any one time there is always a line of health professionals who advise their patients that even small efforts to reduce exposure can have benefits, especially with vulnerable groups. Our new Air Quality Health Index (AQHI) is designed for this purpose although we need to promote it more effectively together with health professionals.

For example, people can reduce their daily exposure to pollution by walking on a road with lower emission. This is why we are beginning to see city authorities, such as London, work with air quality scientists and health experts to devise real time, on-line communication tools that give residents information about the city’s roadside pollution levels. In other words, people can make daily choices to reduce their exposure.

There are other things still that Hong Kong can consider, such as strengthening emissions regulation for vehicular testing and maintenance; and using financial tools to change behaviour like pricing the use of roads for vehicles. Health professionals can be part of the discussion on how making Hong Kong healthier and at the same time improve the environment are exactly what Hong Kong needs to be livable.

With air pollution being a major public health risk in Hong Kong, health professionals must press home the point that pollution reduction is a public health issue and not just about environmental protection. Only this will give air pollution the attention it deserves in our community. The evidence is there – we need health professionals to make it clear the very high risk to public health arising from air pollution and there is a case to be made to give it much higher attention still. This is not the time to be self-satisfied even though Hong Kong has major end-of-pipe emission reduction programmes.
Cesarean Delivery vs Vaginal Delivery - Are There Any Differences?

Gastrointestinal symptoms are more prevalent in Cesarean-born infants up to 1 year of age.

Rate of gastrointestinal symptoms in hospitalized infants at 1 month and 1 year of age:

- Vaginal delivery (n=2541)
- Cesarean delivery (n=2479)

1 month after birth

- Vaginal delivery: 23%
- Cesarean delivery: 28% (p < 0.0001)

1 year after birth

- Vaginal delivery: 19%
- Cesarean delivery: 24% (p < 0.0001)

A meta-analysis confirms that Cesarean delivery is a specific risk factor for allergies up to 23% more risk.

ESPAGHAN recognizes the efficacy of B. lactis for the prevention of gastrointestinal infections. 46% risk reduction.

Increased allergy risk after Cesarean delivery

- Allergic rhinitis: OR = 1.23 (95% CI)
- Atopic dermatitis: OR = 1.16 (95% CI)
- Hospitalization for allergy: OR = 1.11 (95% CI)

ESPAGHAN: Effect of B. lactis (alone or in combination) on risk of non-specific gastrointestinal infections - Meta-analysis

- Odds ratio (OR) = 0.54 (95% CI)

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- Routine formula for over 25 years experience

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For healthy infants who are not exclusively breastfed and who have a family history of allergy, feeding a 100% Whey Protein Partially Hydrolyzed infant formula from birth up to 4 months of age instead of a formula containing infant cow’s milk proteins may reduce the risk of developing atopic dermatitis throughout the 1st year of life. FDA has concluded that the relationship between 100% Whey Protein Partially Hydrolyzed infant formulas and the reduced risk of chronic dermatitis is unproven, because there is little scientific evidence for the relationship. Partially hydrolyzed formulas should not be fed to infants who are allergic to milk or to infants with existing milk allergy symptoms. If you suspect your baby is allergic to milk, or if your baby is on a special formula for the treatment of allergy, your baby's care and feeding choices should be under a doctor's supervision.

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Air Pollution and PM$_{2.5}$ Health Exposure in Hong Kong

Dr Alexis KH LAU and Dr Ying LI

Division of Environment, The Hong Kong University of Science and Technology

Regional and Local Air Pollution over Hong Kong

According to the World Health Organization (WHO), air pollution contributed to over 7 million premature deaths worldwide in 2012. Hong Kong, despite being one of the most well-developed and modern cities in the world, still has a serious air quality problem. Our air problem has two parts. The first part is the more apparent regional air pollution problem, commonly seen as haze or smog covering the whole city; it is caused mainly by transport of pollutants from the mainland, especially the Pearl River Delta (PRD) region during the winter season. This regional smog is mainly composed of high concentrations of particulate matter (PM) and/or ozone (O$_3$). Secondly, because of the dominance of diesel engines in our commercial fleet (including both vehicles and marine vessels) and the enhanced trapping of pollutants by our high-rise buildings, we also have a severe transport and city-planning related urban air pollution problem. This local urban air pollution problem is mostly associated with high levels of PM and nitrogen oxides (NOx). Hence, both regional and local emission sources contribute significantly to our overall air pollution problem.

Air Quality Improvement in the Past Decade

During the past decade, the Government has worked hard to reduce emissions from local sources including power plants, on-road vehicles, and marine vessels. This includes mandating the installation of fuel gas desulphurisation and denitrification systems at power plants, tightening power plant emission caps, adopting tighter vehicles fuel standards, requiring Euro-V emission standards for newly registered vehicles, continuing to promote the replacement of ageing commercial diesel vehicles, as well as new regulations for local vessels to use low-sulphur fuels vessels and for ocean-going vessels to switch to low-sulphur fuel at berth. Hence, compared with 1999, the 2013 roadside respirable suspended particulates (RSP or PM$_{10}$) level has reduced by 37% and the corresponding nitrogen dioxide (NO$_2$) level has reduced by 29%.$^{2,3}$

In addition, Hong Kong has also worked closely with Guangdong to improve the air quality over the south China region. These efforts include the setting up of the 2010 HK/PRD joint emission reduction targets for RSP, SO$_2$, NO$_X$, and VOC (volatile organic compound, precursors of photochemical oxidants like ozone), the PRD Regional Air Quality Management Plan, and the regional air quality monitoring network in 2005. These efforts have led to gradual improvement of regional air quality. For example, using 1996 as the reference, Figure 1 shows the percentage change of regionally averaged air pollutant concentrations of SO$_2$, NO$_2$, PM$_{10}$, and PM$_{2.5}$ from the regional air quality monitoring network. They all have a clear decreasing trend over the PRD from 2006 to 2011.$^4$ More recently released data from the same network show that, compared with 2006, the average annual concentrations of SO$_2$, NO$_2$, and PM$_{10}$ have decreased by 62%, 17%, and 24%, respectively.

Figure 1 Air Quality trend over the PRD from 2006-2011.

Challenges Ahead

Regarding challenges, Figure 1 shows that ozone, a key criteria pollutant and a major constituent of photochemical smog, is on a steady rising trend. Ozone is a secondary pollutant, meaning that it is not directly released at emission sources, but instead it is formed in the atmosphere from other pollutants (ozone precursors). Studies have suggested that the steady rise of ozone over Hong Kong and the PRD is super-regional in nature (i.e. a large portion of the precursors for the ozone measured in Hong Kong and the PRD are emitted outside this region)$^5,6$. This implies that ozone improvement requires emission control of ozone precursors beyond the PRD, which requires concerted national efforts to better quantify the NOX and VOCs emissions to help develop targeted and more effective ozone control strategies.$^6$

Furthermore, we cannot be satisfied with the current PM or NOX situation just because of the decreasing trend of these pollutants shown in Figure 1. The actual annual average concentrations for these pollutants are still significantly higher than the corresponding Air Quality Guideline (AQG) concentration levels recommended by the WHO. For example, the Environmental Protection Department (EPD) reported that the highest annual mean concentration of respirable suspended particulates (RSP or PM$_{10}$) at Hong Kong’s air quality monitoring station was 64 µg/m$^3$, this is more than three times the corresponding WHO AQG levels of 20 µg/m$^3$. For the smaller and more damaging fine suspended particulates (FSP or PM$_{2.5}$), our situation is even worse. The highest annual concentration of PM$_{2.5}$ in Hong Kong was reported by EPD as 45 µg/m$^3$, more than four times
the corresponding WHO AQG of 10 µg/m³. Hence, we certainly have a serious air pollution problem. The improvement of PM and NOx over the past decade showed clearly that targeted air pollution control measures can help improve air quality, we have to work harder on it.

**PM2.5 Distribution and Health Exposure**

In Hong Kong, we have traditionally relied on in-situ data measured at the ambient air quality monitoring stations and applied epidemiological studies to estimate the population health exposure to ambient air pollutants. These in-situ measurements are very bulky and expensive, and hence they cannot be deployed extensively. We currently only have 15 air quality monitoring stations in Hong Kong. However, because of our complex topography and densely built-up urban and commercial areas, there must be substantial spatial variations in pollutant concentrations within Hong Kong. Depending on the local urban morphology and traffic density, the pollutant concentrations within the same district can be quite different. Hence, the average concentrations obtained from the air quality monitoring stations are probably too coarse in spatial coverage for describing the health exposure impacts of the air pollutants at different districts.

Recent advances in satellite remote-sensing techniques have allowed us to estimate the ground-level PM2.5 concentration. Figure 2(a) shows the satellite-derived ground-level PM2.5 concentration at 1km x 1km resolution over Hong Kong for 2013; also shown in Figure 2(b) is the population density mapped to the same spatial resolution. Figure 2 shows that there are substantial spatial variations in PM2.5 concentration and population density in Hong Kong. The orange and red areas in Figure 2(a) are areas with annual average PM2.5 concentration higher than 35 µg/m3 (our current Air Quality Objective and WHO Interim Target-1 for PM2.5). Two factors contribute to the PM2.5 variations in Hong Kong: first, there is a general northwest (higher) and southeast (lower) concentration gradient of PM2.5, associated with regional transport of pollutants from the PRD; second, there is also increases in PM2.5 concentration in areas with higher population density, probably related to enhanced traffic emissions at these more densely populated areas.

Taking advantage of the new remote-sensing data, we have plotted, in a bar chart (Figure 3) the population weighted average concentration of PM2.5 (blue) and the spatial average concentration of PM2.5 (green) for the 18 districts in Hong Kong. A few observations can be made from Figure 3. Firstly, not all districts are the same; some are cleaner than others. The districts with the highest population-weighted PM2.5 concentrations are Kowloon City, Yau Tsim Mong, Yuen Long, Wong Tai Sin, Shum Shui Po and Kwun Tong. Secondly, we note that, for the same district, the population-weighted concentration (blue bar) are typically higher than the simple spatial average (green bar) – this shows the pollution concentration increases with increasing population density. Thirdly, while most of the city have PM2.5 concentrations less than 35 µg/m³ (Figure 2(a)), on average, close to 50% of our population are living in the areas with PM2.5 exceeding WHO IT-1 concentrations. Last but not least, all districts in Hong Kong have annual PM2.5 concentrations higher than 25 µg/m³ (WHO IT-2), and we have a long way to go from the WHO AQG. For more effective protection of public health, more control efforts must be targeted at these high-pollution high-population areas. Beyond the tail-pipe solutions targeting individual emission sources, we also need to consider urban-planning and demand-side management measures (e.g. ventilation assessment in planning, electronic road pricing, low-emission zone, pedestrianisation, etc.) to improve ventilation and reduce traffic emissions in the most densely populated part of our city.

**Summary**

Hong Kong has a serious air pollution problem, contributed by both regional and local emission sources. With concerted efforts by the Hong Kong and Guangdong governments, the air quality trend has turned its corner and started a decreasing trend starting from 2006/2007. However, our current pollutant concentrations are still significantly higher than those recommended by the WHO, and a large portion of our population is still living in areas with PM2.5 concentrations higher than the minimum WHO IT-1 values. More air control efforts must be targeted at these high-pollution high-population density areas to help more effectively protect public health and reduce the overall population exposure to the air pollutants.

**References**

When it matters most against MRSA¹
New evidence, new hope

ZEPHyR —
the largest prospective trial of MRSA nosocomial pneumonia (NP) to date²

Linezolid demonstrated:

- Statistically superior clinical efficacy over dose-optimized vancomycin in MRSA NP¹²
- Statistically significant microbiologic success vs vancomycin in MRSA NP²

¹ Primary end point of ZEPHyR trial: clinical success at end of treatment (EOT) and end of study (EOS) in per protocol (PP) populations.
² Secondary end point of ZEPHyR trial: clinical success at end of treatment (EOT) and end of study (EOS) in per protocol (PP) and modified intent-to-treat (mITT) populations.

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3. INDICATIONS: a) Vancomycin-Resistant Enterococcus faecium infections, including cases with concurrent bacteremia; b) Nosocomial pneumonia (NP) caused by Staph. aureus (methicillin-resistant and - sensitive strains); Strep. pneumoniae (multi-drug resistant strains); c) Complicated skin and skin structure infections (CSSSI), including diabetic foot infections, without concurrent osteomyelitis, caused by Staph. aureus (methicillin-resistant and - sensitive strains); Strep. pyogenes, or Strep. agalactiae; d) Uncomplicated skin and skin structure infections caused by Staph. aureus (methicillin-resistant only) or Strep. pyogenes; e) Community-acquired pneumonia (CAP) caused by Strep. pneumoniae (multi-drug resistant strains), including cases with concurrent bacteremia or Staph. aureus (methicillin-resistant strains only).
4. DOSAGE: a) CAP, NP: For 10-14 days: 12mg/kg IV or PO q12h; pediatric patients (birth - 11 yrs): 15mg/kg IV or PO q12h. b) Vancomycin-resistant Enterococcus faecium infections: For 14-28 days: 12mg/kg IV or PO q12h; pediatric patients (birth - 11 yrs): 15mg/kg IV or PO q12h. c) Uncomplicated skin and skin structure infections: For 10-14 days; Adults: 400mg PO q12h; Adolescents: 600mg PO q12h; 5-11 yrs: 10mg/kg PO q12h; <5 yrs: 5mg/kg PO q12h.
5. CONTRAINDICATIONS: Known hypersensitivity to linezolid or any of the excipients; MAOI: Potential interactions producing elevation of BP; Potential serotoninergic interactions.
6. WARNINGS & PRECAUTIONS: Myelosuppression (incl. neutropenia, thrombocytopenia, and anemia); Mortality imbalance in an investigation study in patients with catheter-related bloodstream infections, including catheter-site infections; Clostridium difficile associated diarrhea (CDAD); Lactic acidosis; Serotonin syndrome; Peripheral & Optic neuropathy; Convolutions (Please refer to the full Prescribing Information for details).
7. INTERACTIONS: Adrenergic and serotonergic agents, strong CYP450 inducers.
8. PREGNANCY AND LACTATION: Pregnancy category C — should only be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. Caution should be exercised when administered to a nursing woman as it is not known whether linezolid is excreted in human milk.
9. SIDE EFFECTS: Most common: diarrhea, headache and nausea. Other reported in clinical studies include: oral moniliasis, vaginal moniliasis, hypertension, dyspnea, localized abdominal pain, pruritus, and tongue discoloration. Reference: HK PI (version date/LPD date) June 2010 Date of preparation: March 2011

Identifier number: ZYVOX0111 FULL PRESCRIBING INFORMATION IS AVAILABLE UPON REQUEST.

Air Pollution and Child Health

Dr Alfred TAM

MBBS(HK), FRCP(Edin., Lond., Glasg.), FHKCPaed, FHKAM(Paediatrics)
Specialist in Paediatrics

The effects of ambient air pollution (AP) on children have been documented over many decades. Children are considered more vulnerable because of the relative immaturity of their body systems, the fact that they spend more time outdoors, and that they have a relatively higher respiratory rate and surface area, increasing their contact with pollutants in the air. Respiratory effects of AP have been well documented. Besides, some studies have shown its relationship with infant mortality, childhood cancer, and brain development, especially related to environmental mercury and lead. This article will focus on the effects of AP on the developing lungs and respiratory system, with special references to studies done in Hong Kong.

Hong Kong Air Pollution Studies on Children

Two studies have shown a causal relationship between AP and mortality in general and cardiorespiratory mortality in particular, affecting adults as well as children. Both studies have shown a dose-response relationship between the increase of NO2, O3, PM10 and SO2 concentration and cardiorespiratory mortality. Respiratory symptoms ranging from sore throat, frequent or chronic cough, phlegm in the morning, chronic phlegm, wheezing to asthma have been found to be more frequent in children residing in areas with higher pollution levels, with odds ratios (OR) ranging from 1.74-3.84. Furthermore, respiratory illnesses resulting in doctor consultations were also increased by elevations of NO2, SO2, O3 and PM10 levels. Again a dose-response relationship was found. Two studies have shown lower lung function in children exposed to higher levels of pollutants. A small but significantly decrease in FVC and FEV1 was reported in one study, affecting girls to a larger extent. The other study reported lower FEV1, FEF25-75 and FEF75 in exposed children, with little difference between the sexes. A higher prevalence and degree of bronchial hyperreactivity was also found in exposed children in one study in the early 90’s. This was shown to have improved together with a decrease of respiratory symptoms after the government enacted laws to decrease the sulphur content in fuel, causing a drastic improvement of SO2 pollution in the territory. This timely study confirmed the health benefits that will result from proper and effective control of air pollutants.

Traffic Air Pollution and Asthma

Most studies on the health effects of AP in Hong Kong have focused on ambient AP. Recent international literature has emphasised the importance of traffic AP in relation to respiratory diseases, especially asthma and allergic sensitisation. Large cohort studies from Europe and the US have shown that children living in homes less than 50m to main roads with heavy traffic or were exposed to higher levels of PM2.5 have a higher chance of diagnosed asthma and hay fever at 6 years of age (OR 1.56-1.66). A higher chance of sensitisation to allergens, especially to outdoor allergens, was also found in those who were exposed to AP or living less than 50m to main roads (OR 1.30-1.52). Also, those children in home addresses with high PM2.5 levels had a higher chance of diagnosed asthma or wheezing at 8 years of age (OR 1.12-1.28). These epidemiological data have highlighted a possible role of AP, especially traffic AP in the aetiology of asthma and allergic sensitisation. More clinical studies are necessary to confirm the relationship. Traffic AP is mainly produced by vehicle emissions, especially from unclean or poorly maintained diesel engines. The emissions are rich in PM2.5 and NOx. In Hong Kong, roadside air monitoring stations, as opposed to general air monitoring stations which are situated on top of buildings, give a reflection of the seriousness of traffic AP. Moreover, our large, active but outdated diesel vehicle fleet, the narrow streets and our weather characteristics all contribute to function in the long term. However, this benefit may be attenuated by exposure to AP. A comparative study of lung function and VO2max in young people showed that not only did those exposed to AP have lower VO2max, in those who regularly exercise in polluted air, their improvement in VO2max was lost as compared to those who exercised in cleaner air. AP has also been shown in a study to increase influenza hospitalisation (0.24% increase in hospitalisation per 10ug/m3 increase of O3), suggesting that a potentiating effect to vulnerability of infection. The relationship between AP and asthma exacerbation and admission has been well documented by 4 studies. For every 10ug/m3 increase in NO2, SO2, O3 and PM10 hospital admission for asthma increased by 1.9-8%. A lag phase of up to 4 days was also documented in one study. The local studies described above have convincingly shown a causal relationship between AP and respiratory morbidity, including respiratory symptoms, doctor consultations, asthma and influenza admissions, poorer lung function, increased bronchial hyperreactivity and suppressed cardiorespiratory fitness.
an increasing effect of traffic AP in the heart of the city. There is reason to believe that the effects of traffic AP reported elsewhere in the world are likely to be causing more problems in our children. However, similar studies are difficult in Hong Kong as a control group will be difficult to find. Apart from living in some parts of the New Territories, most families in Hong Kong will have difficulties living more than 50m away from heavy traffic. Perhaps the only solution is to stay in a higher floor apartment. Even then, other factors like wind directions and weather conditions may affect the surrounding air quality.

What advice should we give to parents?

1. Be informed. The Air Quality Health Index (AQHI)\textsuperscript{25}, available as an Apps on smartphones and the internet, and discussed elsewhere in this series, is important information that reflects the short-term AP situation in various parts of Hong Kong. This information should be available to all parents and individuals, especially those who spend a significant amount of time outdoors or by the roadside. This will allow the individual to make informed decisions whether or not to travel or exercise in the light of AP conditions. Future improvement on the application may enable city dwellers to avoid areas of high AP when traversing from one place to another.

2. Minimise infections. As AP possibly increases individual vulnerability to infections and perhaps makes it worse (at least for influenza infection), parents should consider not sending their children to school or nursery too early, and practise infection control measures at home and when their children are in day care, nursery or kindergarten. Influenza and pneumococcal vaccinations should be taken up appropriately.

3. Treat nose allergy. Keeping the nose functioning normally, especially in children with atopic tendency, will lessen the chance of allergens and large particulates from reaching the lower airways and hence decrease the chance of sensitisation.

4. Exercise in clean air. Improvement in cardiorespiratory fitness will only happen with exercise conducted in relatively clean air. So the child should be brought to open clean air to exercise. Failing that, exercise should be conducted in a clean indoor environment.

5. Avoid environmental tobacco smoke. The detrimental effect of ETS is at least as serious as that or AP. So every effort should be made to control ETS in the child’s environment.

6. Anti-oxidants. There is some limited evidence to show that anti-oxidant intake can attenuate the effects of AP in both controlled exposure studies and ambient exposure studies\textsuperscript{26}. Anti-oxidants studied included Vitamins C, E and beta-carotene. However, little real-life studies have been done with enough power to permit clear clinical recommendations.

7. Air filters. Some data exist to show that high efficiency particulate aerosol (HEPA) filters with enough power may attenuate effects of AP on allergic rhinitis symptoms and asthma\textsuperscript{27}. However, again clinical study data are lacking to allow practical recommendations.

References


Air pollution: Its impact on adult patients with respiratory conditions

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Introduction

Respiratory ailments arising from the bad air in Hong Kong is an increasing observation in the doctor’s office. The possible link between air quality and the patients’ respiratory discomfort is usually apprehended by the patient whose asthmatic cough or chest tightness would improve or even disappear while travelling overseas, and yet such symptoms would return soon after their return to Hong Kong. It is high time that we scrutinise the untoward effects of air pollution on our adult respiratory patients.

Air pollution and asthma

Case presentation

A 40 year-old Caucasian, Hong Kong resident and ex-smoker, sought medical attention for 6 months’ history of episodic shortness of breath (SOB). He had been an amateur marathon runner for over 10 years, having participated in many local and regional marathons. He jogged regularly at the waterfront. Six months earlier, he experienced the first episode of SOB when he was 3-4 kilometres (km) into the Standard Chartered marathon and had to quit running at 5 km. Two months later, similar SOB developed at 2 km while he was in a 50-km marathon. The exercise-induced symptoms made it increasingly difficult for him to continue running. There was no history of atopy in the patient, nor family history of asthma. At presentation, the diagnosis of asthma was made based on physical finding of diffuse expiratory wheeze and on spirometric finding of mild airflow obstruction (FEV1/FVC ratio of 74%, normal being 82%) with excellent reversibility. Serum IgE was 13 (normal 87). The patient was started on combined bronchodilator and steroid inhalation therapy. He was also advised to refrain from running outdoors especially during peak traffic hours. His symptoms improved steadily.

Case Discussion

The development of adult-onset asthma in this patient without apparent atopy may be related to his earlier history of smoking, as well as his hobby as a marathon runner. Outdoor running in the urban environment can significantly increase the degree of exposure to roadside air pollutants.

Review of recent literature on air pollution and asthma

The idea that outdoor air pollution can cause exacerbations of pre-existing asthma has been firmly supported by an evidence base steadily amassing over several decades, while more recent studies have suggested a contribution to new-onset asthma as well.1 Exacerbations of asthma can manifest in increased hospital admission rates for asthma, or in increased emergency department (ED) visits. In a 2013 Canadian study involving 1.5 million patients with asthma on a provincial registry, daily air pollutant measures were obtained from 14 regional monitoring stations in Ontario. The Air Quality Health Index (AQHI) values were found to be significantly associated with increased use of asthma health services on the same day and on the following two days. A 1-unit increase in the AQHI was associated with a 5.6% increase in asthma outpatient visits, 2.1% increase in hospitalisation rate, and 1.3% increase in ED visit rate.2 Researchers in Shanghai also recently reported that an interquartile range (IQR) increase in the moving average concentrations of particulate matter with an aerodynamic diameter of <10 µm (PM10), sulphur dioxide (SO2), nitrogen dioxide (NO2) and black carbon (BC) on the concurrent day and previous day corresponded to 1.82%, 6.41%, 8.26% and 6.62% increase of asthmatic hospitalisation, respectively.3 There is no question that outdoor air pollution can lead to increased morbidity in asthmatic patients of all ages.

The causal relationship between outdoor air pollution and adult-onset asthma is less clear. A 2012 European review of the current evidence examined 7 publications from 5 study populations.4 Meta-analytic summaries were not possible because of the heterogeneity in the design of the studies. Six of these studies used markers of local traffic-related air pollution to characterise long-term exposure, and reported similar associations between asthma incidence and traffic-related air pollution. A 2012 UK systematic review of studies involving all age groups inclusive of some powerful meta-analysis led to the UK’s Committee on the Medical Effects of Air Pollutants to conclude causatively that “overall the evidence is consistent with the possibility that outdoor air pollution might play a role in causing asthma in susceptible individuals living very close to busy roads carrying lots of truck traffic.”5
The largest epidemiologic study of adult-onset asthma and ambient air pollution was just recently reported in the blue journal. As a spin-off of the Sister Study, there was a nationwide cohort of U.S. women (who were sisters of women with breast cancer without baseline symptoms, enrolled in 2003-2009, n=50,884), whose respiratory health, including self-reported wheeze, chronic cough, and doctor-diagnosed asthma, was followed up in 2008-2012 and correlated with annual average ambient PM2.5 and NO2 concentrations captured in their respective residential districts in 2006. For an IQR difference of 3.6 µg/m3 in estimated PM2.5 exposure, the adjusted odds ratio (aOR) was 1.20 for incident asthma and 1.14 for incident wheeze. For NO2, the aOR was 1.08 for incident wheeze per IQR of 5.8 ppb. This study suggests that PM2.5 exposure increases the risk of developing asthma and that PM2.5 and NO2 increase the risk of developing wheeze; the cardinal symptom of asthma, in adult women.

As for the pathogenesis of air-pollution-induced adult-onset asthma, Gowers et al proposed plausible complex interplay of the genes and various triggers, with air pollution potentially contributing to the asthmatic state via multiple mechanisms, including (1) oxidative stress and damage, (2) airway remodelling, (3) inflammatory pathways and immunological effects, and (4) enhancing respiratory sensitisation to allergens. In the prospective Atlanta Commuters Exposures (ACE-1) Study, 42 adults (21 with and 21 without asthma) were asked to undertake two two-hours scripted highway commutes during morning rush hour in the metropolitan Atlanta area. A suite of in-vehicle particulate components were measured in the subjects’ private vehicles. The study showed, at measurement time points within 3 hours after the commute, there were mild to pronounced elevations in exhaled nitric oxide, C-reactive protein, and exhaled malondialdehyde, indicative of pulmonary and systemic inflammation and oxidative stress initiation, echoing the mechanisms of damage proposed by Gowers as summarised above.

Air pollution and COPD

Case presentation

A 69 year-old Hong Kong Chinese was diagnosed to have chronic obstructive pulmonary disease (COPD) one year earlier. He had previously smoked 2 packs per day for 50 years, and had quit smoking 5 years earlier when he developed breathlessness. He presented to the clinic one year earlier for progressive breathlessness. A lung function test showed a normal FVC, a FEV1 of 1.06 L (43% of predicted), and a FEV1/FVC ratio of 34%. The TLC was 108% and RV 140% of predicted. The findings were compatible with severe COPD, at stage 3 of the GOLD classification. The additional finding of a DLCO at 43% and KCO at 59% of predicted confirmed the diagnosis of emphysema. His peak flow rate (PFR) was 180. His resting SpO2 was 97%. He walked 440 metres in 6 minutes with SpO2 plummeting to 89%. CT thorax confirmed emphysema. He was put on inhalational bronchodilators and over the course of the subsequent year, he developed 2 infective exacerbations requiring outpatient antibiotics. His best PFR achievable was about 280-290, and with exacerbations the reading would drop to 185-220. On 10 November 2014, he visited the clinic with 2 weeks’ history of much worsened breathlessness and more viscous sputum. He attributed the deterioration to the fact that the street where he resided had become part of bus re-routing (as a result of Occupy Central): his street had become a major conduit for 9 bus routes, with a bus stop stationed right in front of his building, where he lived on the third floor. On examination, his resting SpO2 was 95%, and his PFR was 210, as compared to a personal best of 290. His chest showed rather reduced air entry with quiet end-expiratory wheeze. The clinical impression was COPD exacerbation as a result of increased exposure to road-side air pollution. His inhaler therapy was stepped up accordingly.

Case Discussion

This patient suffered from severe COPD at Stage 3 of the GOLD classification. His clinical course appeared to be relatively stable with minor exacerbations which did not require hospitalisation. The recent exacerbation appeared to be temporally related to increased exposure to road-side air pollutants.

Review of recent literature on air pollution and COPD

COPD is a common preventable and treatable disease and is characterised by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles and gases. In 2005, COPD ranked second as a respiratory cause for hospitalisation and inpatient bed days in Hong Kong. In those >75 years of age, the hospitalisation rate for COPD was as high as 2,225/100,000. The prevalence of moderate COPD, using the spirometric reference of FEV1/FVC ratio of <70%, among 1,008 elderly Hong Kong Chinese (age ≥60 years) in the community, were 19.6% and 11.9% in the male and female subjects respectively.

The development of COPD is multifactorial, and the risk factors include both genetic and environmental factors. Tobacco smoking is an established risk factor for COPD. Indoor air pollution (such as second-hand smoking and biomass fuel combustion exposure) is associated with the development of COPD. There is currently no strong evidence that ambient air pollution leads to the development of COPD. A recent study included 6,550 subjects with assigned nitrogen oxides and 3,692 with particulate matter measures by Schikowski and colleagues found no strong association between these traffic-related ambient air pollutants and prevalence and incidence of COPD. Another large population-based English cohort study involving over 16,000 participants also noted inconclusive evidence for associations between ambient air pollution and COPD incidence.

Short-term exposures to air pollutants may have adverse effects on the lung function of COPD patients, but it is uncertain whether COPD patients are more susceptible to the effect of air pollution when compared to healthy subjects. A large scale study using the US Framingham Heart Study cohort found that increased ambient air pollution 24-48 hours before spirometry was associated with lower spirometric results. However, the effects of pollution were not greater in persons with asthma or COPD.
An association is generally observed between increased levels of outdoor air pollutants and increased doctors’ visit, hospital admissions, emergency department visits and higher mortality for patients with COPD. There is some evidence that temperature may modify the effect of air pollutants on these patients. A study in Taiwan found that COPD patients are more sensitive to air pollution and meteorology factors and required more outpatient patients’ consultations when compared to diseases including allergic rhinitis, asthma, and pneumonia. In Hong Kong, ambient concentrations of air pollutants have an adverse effect on hospital admissions for COPD patients, especially during the winter season. Increases in levels of SO2, NO2, ozone, PM10 and PM2.5 were all associated with increased risk for hospitalisation of COPD. A study from the United States found that even indoor air pollution is associated with increased respiratory symptoms and risk of COPD exacerbations. Increases in PM2.5 concentrations in the main living area were associated with increases in respiratory symptoms, rescue medication use, and risk of severe COPD exacerbations.

Apart from increasing hospital admissions and clinic visits, exposure to air pollutants are associated with increased mortality in COPD. A study from Spain that assessed the acute effects of air pollution among subjects suffering from COPD found that ambient air pollution is associated with increased mortality. The association of pollution and mortality was stronger for respiratory causes, but was not significant for cardiovascular causes. A study in Oslo, Norway that assessed the mortality among all inhabitants (143,842 participants) aged 51–90 years from 1992 to 1998 found that the effects of nitrogen dioxide, PM2.5 and PM10 on mortality were more apparent for COPD patients when compared with the rest of the population, and a linear effect of the air pollutants on COPD mortality was observed. For longer term exposure, a population based study that included a 5-years exposure period and a 4-years follow-up period in the United States found that an IQR elevation in BC concentrations was associated with a 7% increase in COPD mortality after adjustment for covariates.

The mechanism of air pollutants in increasing risk of exacerbation and mortality of COPD patients is not certain. It may be due to pollutants triggering increasing inflammation in the already inflamed airway. Systemic inflammation may also play a role. A recent study by Dadvand et al found that short-term exposures to NO2 was associated with increased levels of biomarkers of systemic inflammation (C-reactive protein, interleukin-8 and fibrinogen) and tissue repair (hepatic growth factor), in stable COPD patients, particularly in former smokers.

Conclusions

In conclusion, certain air pollutants, such as ozone, NO2, PM10 and PM2.5, have been observed to be associated with an increased risk of development of asthma. Smoking and biomass exposure are related to the development of COPD but there is no strong evidence that ambient air pollution increases the risk of development of COPD. Air pollution on the other hand is associated with increased exacerbations and morbidity in patients with asthma or COPD. Air pollution is a significant health hazard for patients with pre-existing airway disease.
MCHK CME Programme Self-assessment Questions

Please read the article entitled “Air pollution: Its impact on adult patients with respiratory conditions” by Dr Jane CK Chan and Dr Fanny WS Ko and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 31 January 2015. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please answer T (true) or F (false)

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<td>1. Emphysema is characterized by the lung function finding of airflow obstruction coupled with an impaired diffusion capacity/transfer factor.</td>
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<td>2. Chronic obstructive pulmonary disease (COPD) is characterized by the finding of complete reversibility after bronchodilator use.</td>
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<td>3. A good indicator of the severity of air pollution in a city or region is the measurement of concentrations of air pollutants at residential homes.</td>
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<td>4. Air pollution arising from roadside traffic contributes significantly to the increased morbidity in patients with pre-existing asthma.</td>
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<td>5. There is currently strong evidence that ambient air pollution leads to the development of COPD.</td>
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<td>6. Short-term exposure to air pollutants may have adverse effect on the lung function of COPD patients, but it is uncertain whether COPD patients are more susceptible to the effect of air pollution when compared to healthy subjects.</td>
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<td>7. An association is generally observed between increased levels of outdoor air pollutants and increased doctors’ visit, hospital admissions, emergency department visits and higher mortality in patients with COPD.</td>
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<td>8. PM10 refers to particulate matter with an aerodynamic diameter of &lt;10 um.</td>
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<td>9. Among particulate matter of all sizes, only PM10 appears to be contributing to adverse respiratory conditions.</td>
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<td>10. The UK’s Committee on the Medical Effects of Air Pollutants concluded in 2012 that overall the evidence is consistent with the possibility that outdoor air pollution might play a role in causing asthma in susceptible individuals living very close to busy roads carrying lots of truck traffic.</td>
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ANSWER SHEET FOR JANUARY 2015

Please return the completed answer sheet to the Federation Secretariat on or before 31 January 2015 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Air pollution: Its impact on adult patients with respiratory conditions

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Answers to December 2014 Issue

Expanded Newborn Metabolic Screening: Working towards a mandatory screening programme in Hong Kong

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REFERENCES:
1. Foster SmPC
5. Vois et al. Poster presentation ERS 2009

Further information is available on request

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Air Pollution and Cardiovascular Disease

Dr Archie Ying-sui LO

Air pollution — Fine particulate air pollution has been linked to cardiovascular (CV) disease and refers to particulate matters of less than 2.5 micron in aerodynamic diameter (PM$_{2.5}$). Most$^{1,9}$ but not all$^{10,11}$ studies have found positive associations between several different air pollutants and adverse cardiopulmonary events. The reasons for the discrepancy among observational studies are multifactorial, including variable characteristics of air pollutants, populations and areas studied, control of relevant confounders and uneven data qualities.

Increase in PM$_{10}$ and increase in Cardiopulmonary Mortality

An earlier but representative study looked at outdoor air pollution in 20 of the largest US metropolitan areas (which encompassed a total population of more than 50 million, 1987-1994) and estimated that an increase in the relative rate of deaths from cardiovascular and respiratory causes was 0.68% for each increase in the PM$_{10}$ level of 10 ug/m$^3$. PM$_{2.5}$ data were not available in this study.

An analysis of a national database comprising 204 US counties (population >200,000, with 11.5 million Medicare enrollees, 1999-2002) demonstrated that short-term exposures to PM$_{2.5}$ increased the risk for hospital admissions for cardiovascular and respiratory diseases.$^9$

Decline in PM$_{2.5}$ and increase in Life Expectancy

Earlier analysis of the Harvard Six Cities adult cohort study showed an association between long-term ambient PM$_{2.5}$ and mortality between enrollment in the mid-1970s and follow-up until 1990. Extended mortality follow-up was conducted for 8 years in a period of reduced air pollution concentrations. PM$_{2.5}$ exposure was associated with lung cancer (RR, 1.27) and CV deaths (RR, 1.28). Improved overall mortality was associated with decreased mean PM$_{2.5}$ (10 ug/m$^3$) between the periods (RR, 0.73).$^{12}$

Another study analysed the data on life expectancy for 211 county units in 51 U.S. metropolitan areas. Changes in socioeconomic, demographic variables and the prevalence of cigarette smoking were factored into regression analysis. A decrease of 10 ug/m$^3$ in the concentration of PM$_{2.5}$ was correlated with an estimated increase in mean life expectancy of 0.61±0.20 year (P=0.004).$^2$ The authors opined that reductions in air pollution accounted for as much as 15% of the overall increase in life expectancy in those study regions.

In a study of pollution-related mortality rates in Dublin, Ireland before and after a ban on coal sales, which led to a 70% average reduction in black smoke concentrations,$^{13}$ adjusted CV deaths decreased by 10.3% (p<0.0001) six years after the ban.

PM$_{2.5}$ increases Risk of first CV event

The Women's Health Initiative, also demonstrated that PM$_{2.5}$ was associated with CV disease.$^5$ This database of 65,893 postmenopausal women without prior CV disease in 36 US metropolitan areas (1994-1998) was analysed to evaluate the risk of long term exposures to air pollutants and the risk for a first CV event. Potentially confounding variables such as the body mass index, age, educational levels, household income, and conventional CV risk factors were also taken into consideration. For each 10 ug/m$^3$ increase in PM$_{2.5}$, there were significant increases in the risk of any CV event (hazard ratio 1.24), deaths from CV disease (hazard ratio 1.76), and of cerebrovascular accidents (hazard ratio 1.35).

Pollutants increase Risk of MI

In a systematic review and meta-analysis of data from 34 studies, carbon monoxide, nitrogen dioxide, sulfur dioxide, and small particulate matters (both PM$_{2.5}$ and PM$_{10}$) were all associated with an increased risk of myocardial infarction, with the overall population attributable risk ranging from 1 to 5%; ozone in this study was not.$^{14}$

PM$_{2.5}$, Ozone and Acute Coronary Syndrome

In the American Cancer Society Cancer Prevention Study involving 450,000 subjects, PM$_{2.5}$, but not ozone, concentration was significantly associated with the risk of deaths from CV causes (RR 1.2). The risk of deaths from respiratory causes associated with an increment in ozone concentration of 10 ppb was also increased (RR 1.040).$^{15}$

On the other hand, other studies have demonstrated that short-term exposures to ozone$^{16,17}$ were associated with cardiac admissions. Observational data confirmed that short-term ozone exposures within a duration of just 1 to 2 days was associated with acute coronary events in middle-aged adults without prior heart disease (for an
increase of 5 µg/m³ of ozone concentration, RR 1.05), while NO₂ and SO₂ were not. Subjects 55 to 64 years of age with no prior history of ischaemic heart disease were the most susceptible to suffer from an acute myocardial infarction (RR 1.14).¹⁸

Generally, an acutely increased risk for ischaemic heart disease events has been observed, even within 1 to 2 hours after exposure to elevated PM, as reported in case-crossover analyses.¹⁹ ²⁰ An analysis of out-of-hospital coronary deaths and air pollution in Rome (Italy) showed that air pollutants originating from combustion processes, including PM₂.₅, were related to fatal, nonhospitalised coronary events. The risk appeared higher among people over the age of 65.⁶

Mortality from all causes was higher among subjects with greater exposure to PM₂.₅ in survivors of acute coronary syndrome in England and Wales.²¹ In a Utah (US) study of 12,865 patients, a short-term increase of 10 µg/m³ in PM₂.₅ positively correlated with an increase in acute ischaemic coronary events, especially among subjects with pre-existing coronary artery disease.²² However, individuals with stable presentation and those with angiographically normal coronaries were not as susceptible to short-term particulate exposure.

**Traffic Proximity and Atherosclerosis**

Using thoracic aortic calcification as a marker for atherosclerosis, both long-term exposures to PM₂.₅ and night-time traffic noise were independently associated with subclinical atherosclerosis and might both contribute to the linkage between “traffic proximity” and atherosclerosis.²²

**Heart Failure**

In the Medicare enrollee study,⁹ a 10-µg/m³ increase in concurrent-day PM₂.₅ was associated with a 1.28% increase in heart failure (HF) admissions, and was the single largest cause for hospitalisation in this study. On the other hand, a decrease of PM₂.₅ by 10 µg/m³ was estimated to reduce HF admissions in those 204 counties by 3,156 cases per year.

A case-crossover study from Utah’s Wasatch Front area explored associations between PM₂.₅ and 2,628 HF hospitalisations. The strongest PM₂.₅ - HF associations were for elderly patients who had previously been admitted for HF.²³ A 14-day lagged cumulative moving average of 10 µg/m³ PM₂.₅ was associated with a 13.1% increase in HF admissions.

**How PM₂.₅ may increase risk of CV disease**

Possible mechanisms by which PM₂.₅ may increase the risk of CVD include:

1) An increase in sympathetic tone and/or the trigger for vasospasm. Researchers assessed the vascular response to the 2-hour inhalation of approximately 150 µg/m³ of concentrated ambient fine particles (CAP) plus ozone (120 ppb) versus the response to the inhalation of filtered air. Exposure to CAP plus ozone caused a significant brachial artery vasoconstriction compared with filtered air inhalation (-0.09/+0.15 mm versus +0.01/-0.18 mm, P=0.03).²⁴ To investigate the link between PM₂.₅ and blood pressure during 631 repeated visits for cardiac rehabilitation in 62 Boston residents with CV disease, data showed that for an increase from the 10th to the 90th percentile in mean PM₂.₅ levels during the 5 days prior to the visit, there was a 2.8-mm Hg increase in systolic, and a 2.7-mm Hg increase in diastolic blood pressure.²⁵ Other studies have likewise demonstrated an association between PM₂.₅ and increase in blood pressure.²⁶ Increased levels of PM₂.₅ were associated with lower heart rate variability, suggesting lower cardiac autonomic control.²⁷

2) An increase in the susceptibility to myocardial ischaemia. In 3,256 randomly selected men and women 25 to 64 years of age, high pollutant concentrations were associated with increased plasma viscosity,²⁸ which may aggravate myocardial ischaemia. Levels of particulate air pollution 2 days before exercise stress testing were associated with increased risk of ST-segment depression during exercise test.²⁹

3) Endothelial dysfunction. Endothelial function was impaired by ordinary levels of pollution in healthy young urban males and may be reduced by 50% between the least and the most polluted days.³⁰

4) Possible trigger for atherosclerosis.³¹, ³² One study correlated long-term exposures to fine particles with carotid intima-media thickness (CIMT). An increase in PM₂.₅ (4.2µg/m³), PM10 (6.7µg/m³), was associated with a 4.3% and 1.7% increase in CIMT, respectively.³² With the assumption that atherosclerosis is in part an inflammatory process, epidemiological data have linked PM exposures to an augmentation of systemic inflammation as evidenced by elevated C-reactive protein.³³

**AHA Scientific Statement on Air Pollution**

The first American Heart Association (AHA) Scientific Statement on “Air Pollution and Cardiovascular Disease” was of the opinion that the “overall evidence is consistent with a causal relationship between PM₂.₅ exposure and cardiovascular morbidity and mortality”.³⁴ This body of evidence has expanded and been substantiated further since and the 2010 update from the AHA stated that “PM₂.₅ exposure is deemed a modifiable factor that contributes to cardiovascular morbidity and mortality”.³⁵ New conclusions in the 2010 update also included the following: “Exposure to PM₂.₅ over a few hours to weeks can trigger cardiovascular disease–related mortality and nonfatal events; a longer-term exposure (e.g. a few years) increases the risk for cardiovascular mortality to an even greater extent than excesses over a few days and reduces life expectancy within more highly exposed segments of the population by several months to a few years; reductions in PM levels are associated with decreases in cardiovascular mortality within a time frame as short as a few years; and many credible pathological mechanisms have been elucidated that lend biological plausibility to these findings. It is the opinion of the writing group that the overall evidence is consistent with a causal relationship between PM₂.₅ exposure and cardiovascular morbidity and mortality”.

³³
Obviously, despite extensive data showing PM$_{2.5}$ to be causally related to CV diseases, there are still numerous unanswered questions. Further research will be needed to better document the time course and specific CV health benefits induced by reductions in PM; better define susceptible individuals/population; determine which adverse CV effects (both acute and chronic) at a population level can be avoided.

**Conclusion**

In summary, there is little dispute over whether air pollution, in particular PM$_{2.5}$, is causally related to an increase in the risk of CV disease. Our government should strive to decrease the impact of air pollution to improve the health of our society. All patients with CV diseases should be educated about the risks posed by air pollution.

**References**

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4. A statistically significant 14% reduction in the risk of mortality with Spiriva while patients received study medication. (P=0.006, on treatment analysis).
5. Effect extended to end of treatment period (day 1440), as defined by protocol (13% risk reduction. P=0.034, intention-to-treat analysis).
6. For the 30 days following the conclusion of treatment period (Day 1470) when, according to protocol, patients were discontinued from their study medication, the study revealed an 11% reduced risk reduction (P=0.066, intention-to-treat analysis).
7. In UPLIFT, tiotropium demonstrated reduced risk of COPD exacerbations and hospitalisations, improved quality of life and reduced mortality vs placebo.
8. In POET-COPD, tiotropium demonstrated reduced risk of COPD exacerbations and hospitalisations vs salmeterol.

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Is Air Pollution Carcinogenic??

Dr George Tak-jor AU

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Air Pollution is often separated into indoor and outdoor situations. Outdoor air pollution mainly deals with diesel exhaust in the air that we breathe and Indoor air pollution will include radon gas, cigarette smoke exposures and contribution from burning fuel for cooking and heating, which is a big issue in developing countries.

What is diesel exhaust? Diesel is a type of derivative from crude oil. Large engines including those used in trucks, buses, trains, construction and farm equipment, generators, ships and in some cars, run on diesel fuel. The exhaust from diesel engines is made up of 2 main parts: gases and soot. The gas portion is mostly, carbon dioxide, carbon monoxide, nitric oxide, nitrogen dioxide, sulfur oxides and polycyclic aromatic hydrocarbons (PAHs). The soot portion is made up of carbon particles, organic materials (including PAHs) and traces of metallic compounds. Exposure to diesel exhaust is widespread in modern world. Health concerns about diesel exhaust relate not only to cancer, but also to other health problems such as lung and heart diseases. People are exposed to diesel exhaust mainly by breathing in the smoke and gases, which then enter the lungs. This is particularly relevant in poor countries, where trucks, generators and farm and factory machinery routinely belch clouds of sooty smoke and fill the air with sulfurous particulates. The United States and many western countries have less of a problem because they require modern diesel engines to burn much cleaner than they did even a decade ago. Mining industries are regulated with limits on the amount of diesel fumes to which their workers may be exposed.

Landmark Study linking diesel exhaust to Lung Cancer and Possibly Bladder Cancer Non metal miners who were exposed to high levels of diesel exhaust appear to have a considerably higher risk of developing and dying from Lung Cancer compared to other people. This was reported in the Journal of National Cancer Institute in 2012. This study, part of the Diesel Exhaust in Miners Study was made by scientists from the National Cancer Institute and National Institute for Occupational Safety and Health. In June, 2012, the International Agency for Research on Cancer (IARC), which is part of the World Health Organization (WHO) declared after their meeting in Lyon, France that they classified diesel engine exhaust as carcinogenic to Humans (Group 1), based on sufficient evidence that exposure is associated with increased risk for lung cancer, as well as limited experience linking it to an increased risk of Bladder cancer. Diesel exhaust now shares the W.H.O. Group 1 carcinogen status with smoking, asbestos, ultraviolet radiation, alcohol and other elements that pose cancer risks. Other agencies, including National Institute of Health, (NIH), Centers for Disease Control (CDC), Food and Drug Administration (FDA) and Environmental Protection Agency (EPA) have classified diesel exhaust as a potential occupational carcinogen.

Public Health Implications and Government Regulation Increasing environmental concerns over the past two decades have resulted in regulatory action in North America, Europe and elsewhere with successively tighter emission standards for both diesel and gasoline engines. There is a strong interplay between standards and technology - standards drive technology and new technology enables more stringent standards. For diesel engines, this required changes in the fuel, such as marked decrease in sulfur content, changes in engine design to burn diesel fuel more efficiently and reductions in emissions through exhaust control technology. In the Unites States, miners are covered by the Mine Safety and Health Administration (MSHA), which currently enforces standards at exposure in underground metal and coal mines. Workers in agriculture, construction and maritime industries are covered by the Occupational Safety and Health Administration (OSHA). It is notable that many parts of the developing world lack regulatory standards and data on the occurrence and impact of diesel exhaust are limited. Much work remains to be done on this front.

Radon and Cancer

What is radon: Radon is a radioactive gas released from the normal decay of the elements uranium, thorium and radium in rocks and soil. It is an invisible, odourless, tasteless gas that seeps up through the ground and diffuses into the air. Radon gas usually exists at very low levels outdoors. However in areas without adequate ventilation, such as underground mines, radon can accumulate to levels that substantially increase the risk of lung cancer.

How is the general population exposed to radon? Radon is present in nearly all air. Everyone breathes in radon every day, usually at very low levels. Radon can enter homes through cracks in the floor, walls, or foundations and collect indoors. It can also be released from building materials or from water obtained from wells that contain radon. Radon levels can be higher in homes that are well insulated, tightly sealed and/or built on soil rich in elements uranium, radium or thorium.
The basement and first floors typically have the highest radon levels because of their closeness to the ground.

**How does radon cause cancer?** Radon decays quickly, giving off tiny radioactive particles. When inhaled, these radioactive particles can damage the cells that line the lung. Long term exposure to radon can lead to lung cancer, the only cancer proven to be associated with inhaling radon.

**How many people develop lung cancer because of exposure to radon?**
Cigarette smoking is the most common cause of lung cancer. Indoor exposure to second hand smoking is therefore hazardous and can cause lung cancer. Radon represents a far smaller risk for this disease but it is the second leading cause of lung cancer in the United States. Scientists estimate that 15,000 to 22,000 lung cancer deaths in the United States each year are related to radon. Exposure to the combination of radon gas and cigarette smoke creates a greater risk of lung cancer than exposure to either factor alone. The majority of radon related cancer deaths occur among smokers. However, it is estimated that more than 10 percent of radon related cancer deaths occur among nonsmokers.

**How did scientists discover that radon plays a role in the development of lung cancer?** Radon was identified as a health problem when scientists noted that underground uranium miners who were exposed to high radon levels died of lung cancer at alarmingly high rates. The results of miner studies have been confirmed by experimental animal studies which show higher rates of lung cancers among rodents exposed to high radon levels.

**Household Air Pollution and Cancer/Health**

In March 2014 World Health Organization (WHO) released information showing around 3 billion people in the world cook and heat their homes using open fires and simple stoves burning biomass (wood, animal dung, crop waste) and coal. Over 4 million people die prematurely from illness attributable to the household air pollution from cooking or heating with solid fuels. Such inefficient cooking fuels and technology produce high levels of household air pollution with a range of health damaging pollutants, including small soot particles that penetrate deep into the lungs. In poorly ventilated dwellings, indoor air pollution can be 100 times higher than acceptable levels for small particles. Exposure is particularly high among women and young children, who spend the most time near the domestic hearth. Approximately 17% of annual premature lung cancer deaths in adults are attributable to exposure to carcinogens from household air pollution caused by cooking with solid fuels like wood, charcoal or coal. The risk for women is higher, due to their role in food preparation. Pneumonia, stroke, ischaemic heart disease and chronic obstructive pulmonary disease are other causes of premature deaths as a result of household air pollution. There have been reports in parts of China with high incidence of Lung Cancer and other health problems relating to the use of coal for heating and cooking.

**World Health Organisation’s RESPONSE WHO**
WHO is leading efforts to evaluate which new household cooking technologies and fuels produce the least emissions and thus are most optimal for health. WHO is also drawing new indoor air quality guidelines for household fuel combustion. WHO is also providing technical support to countries in their own evaluations and scale-up of health promoting stove technologies.

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The Mission and Vision of the Air Quality Health Index

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Background

The task of reviewing the old air pollution index (API) reporting system and developing a new one started in 2008, with a tender by the Environmental Protection Department (EPD). This was awarded to our multidisciplinary research team of experts on environmental health, air pollution science, epidemiology, and statistics, from the Chinese University of Hong Kong and the Hong Kong University of Science and Technology.

We first reviewed the API, a widely used system first developed by the United States Environmental Protection Agency (US EPA), together with recent advances in air pollution reporting. The API is based on compliance (or otherwise) of national air quality standards (AQS) or air quality objectives (AQO) for selected criteria air pollutants, with a cut-point of 100 at the short-term AQOs, and another at 50 for the more stringent annual AQS. In Hong Kong, the criteria pollutants that were used in calculating the API were: carbon monoxide (CO), nitrogen dioxide (NO2), sulphur dioxide (SO2), ozone (O3), and particulate matter with an aerodynamic diameter less than 10 micrometres (PM10).

It is important to note that even though the APIs in different countries are calculated using similar methods, the values of the API cannot be compared across countries, because of the different criteria pollutants and standards used in different jurisdictions. A Hong Kong API of 100 does not mean the same air quality as an API of 100 in China or the US.

One defining feature of the API is that it is based on the concentration of whichever air pollutant is highest relative to its own AQS, irrespective of the other pollutants. Hence, the API represents the level of the single worst-performing air pollutant. This does not go well with our current understanding that all air pollutants exert some adverse impact on health. Another disadvantage of the API is that, for some air pollutants (namely, NO2, PM, and SO2), it is calculated using concentrations averaged over the past 24 hours. For example, a sudden surge in the concentration of PM (which occurs episodically, through regional air pollutant transportation from the Pearl River Delta) would not be reflected as a rise in the API until many hours later, after the average 24-hour concentration breaches the Hong Kong AQO. Thus, the public would not receive timely warnings about the high PM levels and the API would have failed in its task of risk communication. Finally, while the API informs the public about the level of a given air pollutant relative to its AQO, it does not truly reflect the health risk posed by all the pollutants present in the air.

Developing the AQHI

Canada was the first country to adopt a new method of reporting air quality. Stieb et al (2005 & 2008) developed an air quality health index (AQHI) based on Canadian health and air pollution data. They derived the relative risks (RR) of different air pollutants using a time series approach, and calculated the excess mortality risk based on an assumption of zero risk if the air pollutant is at zero concentration (a hypothetical situation). They then summed up the risks ‘caused’ by each air pollutant, and expressed those as a summary excess risks (ER), given in terms of a percentage increase per unit increase in air pollutant concentrations. This range of excess risk was then divided into categories, with 1–3 as low, 4–6 as medium, 7–10 as high, and 10+ as very high.

Our team saw the advantage of this new system and developed our AQHI based on the Canadian approach, with some modifications. To begin with, we used hospital admissions data for respiratory and cardiovascular diseases instead of mortality data. There were two reasons for this change: firstly, we have comprehensive and high-quality hospital data provided by the Hospital Authority; and secondly, morbidity represents a wider sector of the population than mortality, which reflects only the effect on the most susceptible.

The air pollutants CO and SO2 were not used to compile the Canadian AQHI, as their ambient concentrations in Canada were low. For the Hong Kong AQHI, we also left out CO due to low ambient concentrations, but we chose to retain SO2, so that any future increase in SO2 concentrations can thus be reflected. The other pollutants we used in calculating our AQHI are O3, NOx, and PM10.

Like the Canadian system, we derived the RR of hospital admissions using local data, calculated the %ER of each pollutant and expressed a summed %ER. However, the cut-points of our five categories (low, moderate, high, very high, and serious), further divided into 11 bands (bands 1 to 10 and band 10+), were different from the Canadian approach. Our cut-points were based on the summed %ER equivalents to the short-term air

1 Air Quality Objective’ is the term used in Hong Kong; it is equivalent to the AQS used elsewhere.
2 The time series study is the standard methodology used to determine the short-term health impact of air pollution.
quality guidelines\(^3\) (AQG) recommended by the World Health Organization. A %ER higher than this value was labelled as ‘very high risk’ (bands 8–10), while a %ER modified for the vulnerable groups (children and those aged 65 years and above) was labelled as ‘high risk’ (band 7). Categories with lower %ER values were termed ‘low’ (bands 1–3) and ‘moderate’ (bands 4–6), while the category with even higher %ER values (band 10+) was labelled as posing a ‘serious risk’ to health.

There are three main advantages of the new Hong Kong AQHI over the old API system: (i) it takes into account the effects of all four major air pollutants; (ii) it has a relatively short-lag time, as we use a 3-hour moving average to calculate the index, and (iii) it reflects genuine health risk, rather than compliance with an AQO. In addition, the scale of the %ER is linear, and the five categories (each denoted by a colour code) and eleven bands are more easily interpretable than a number that may range from below 50 to well above 100.

**The Mission of the AQHI and How It Should Be Used**

The AQHI is a tool for communicating, to the general public, the short-term risks to health that arise from exposure to the levels of air pollutants found in the outdoor environment. The purpose of reporting it is to change people’s behaviour. Thanks to the AQHI, people can make informed decisions on their physical activities and health. Healthy people may wish to refrain from outdoor physical activities and strenuous exercises on days when the air quality is poor. Those suffering from heart or lung illnesses, and who are on regular medication, may want to consult their doctors on adjusting the dose they take.

Health advice is provided for healthy individuals in each of the five categories of health risks. There is also targeted advice for vulnerable individuals such as young children, senior citizens, and people with existing heart or lung diseases. These different groups of citizens are advised on how they should respond to each health risk alert level. The bands indicate the risk in greater detail than do the alert levels. Within the same alert level, an AQHI in a higher band denotes a higher health risk, but the health advice is the same.

The AQHI was developed using group data. It cannot be tailored to each individual, as each of us is different in our health status and sensitivity to air pollution. Instead, the AQHI represents the health risk to particular groups. Someone belonging to a vulnerable group would still have to adjust his / her response to air pollution, based on his / her own health condition.

**Our Vision for the AQHI**

Since we wrote our report in 2009, we have received many comments on the methodology of the AQHI and its validity as a health risk communication tool. Our report has been reviewed and supported by experts in Canada and the World Health Organization. Following our work, researchers in Shanghai and Taipei have started developing their own AQHI, perhaps for eventual use in national reporting systems; and in recent years, several more studies on the AQHI have been published (Cairncross et al, 2007; Sicard et al, 2011).

We recently performed a validation study (also commissioned by the EPD) that used a longer time series (12 years’ worth of data). The results were similar to our previous findings, confirming the robustness of the AQHI model. In addition, we found an independent effect of coarse particulates on health. In the future, we would consider enhancing the AQHI by incorporating coarse particulates into the calculations, and using RRs derived from a longer time series. Besides these technical aspects, we would also like the government and the medical community to step up efforts to publicise the AQHI, promote the various AQHI tools already available for smartphone and desktop computer, and – most importantly – to improve public understanding of the AQHI, so that people can learn how to protect their own health from air pollution.

**Acknowledgements**

We would like to acknowledge the other members of our team for their contributions towards the development of the AQHI: Prof. Alexis Lau and Prof. Ignatius Yu for their constructive comments; Prof. Wilson Tam for statistical modelling; Mr Simon Ng for his literature review; and Mr David Yeung for programming work. We also thank the Environmental Protection Department for providing funding, air pollution data, and logistical support for this project.

**References**


\(^3\) The WHO AQG for NO$_2$ (with a 1-hour averaging time) was modified to a 3-hour value, and the lower 95% confidence limit was used.
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Improving Air Quality of Hong Kong

Mr Sik-wing PANG
Principal Environmental Protection Officer (Air Science)
Environmental Protection Department

Like many other places, Hong Kong’s air quality is affected by our own emissions as well as the emissions of our neighbouring areas. To improve our air quality, we have been reducing local emissions as well as working in concert with Guangdong (GD) to reduce emissions in the Pearl River Delta (PRD) region.

In March 2013, the Environment Bureau released “A Clean Air Plan for Hong Kong” (http://www.enb.gov.hk/en/files/New_Air_Plan_en.pdf) to outline comprehensively and clearly the challenges Hong Kong is facing with regard to air quality. It gives an overview of the relevant policies, measures and plans to tackle our air pollution as well as collaboration between Guangdong and Hong Kong to deal with regional pollution.

New Air Quality Objectives and New Air Quality Index

Air Quality Objectives (AQO) are the goal of our air quality management programme. A new set of AQOs (Table 1) came into effect in January 2014. The new AQOs, which are benchmarked against a combination of interim and ultimate targets in the World Health Organization’s Air Quality Guidelines, are broadly comparable to those adopted by the European Union and the United States. To attain the new AQOs, the Government has put forward a wide range of new air quality improvement measures.

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<tr>
<th>Pollutants</th>
<th>Averaging Time</th>
<th>Concentration limit (µg/m³)</th>
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<tr>
<td>Sulphur Dioxide (SO₂)</td>
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<td></td>
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<td>Ozone (O₃)</td>
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<td>Lead (Pb)</td>
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Air pollution communication plays also an important role in helping the public to reduce the risk arising from air pollution exposure. On 30 December 2013, we replaced the Air Pollution Index with a new Air Quality Health Index (AQHI). The AQHI, which reports the health risk associated with air pollution by a figure from 1 to 10 and 10+ under five health categories, is available on EPD’s website (http://www.aqhi.gov.hk/tc.html) and app (HK AQHI). It allows the public to make informed decisions on their outdoor physical activities, especially the susceptible groups such as children and the elderly, people with existing heart and respiratory illnesses to take precautionary measures when necessary.

Reducing Roadside Air Pollution

Among others, roadside air pollution poses the greatest public health concern, which is mainly caused by vehicle emissions trapped in our street canyons. To improve roadside air quality, we have taken a number of measures to reduce vehicle emissions, including:

(i) An incentive-cum-regulatory scheme was launched in March 2014 to progressively phase out some 82,000 pre-Euro IV diesel commercial vehicles by the end of 2019 with an ex-gratia payment amounting to HK$11.4 billion to assist affected vehicle owners. The service life of newly registered diesel commercial vehicles will be limited to 15 years starting from 1 February 2014 to facilitate timely replacement of these vehicles in the long run for better air quality.

(ii) The one-off subsidy scheme to assist owners of about 21,000 LPG taxis and light buses to replace their worn-out catalytic converters and oxygen sensors was completed in April 2014. The replacement can help reduce NOx emissions from these vehicles. Starting from September 2014, the control of emissions from petrol and LPG vehicles has been strengthened through the use of roadside remote sensing equipment and dynamometers for emission testing.

(iii) Franchised bus companies are working to retrofit their Euro II and III buses with selective catalytic reduction devices to reduce their emissions. We are also funding their purchase for trial of six hybrid buses at a total cost of $33 million and 36 electric buses (including charging facilities) with $180 million earmarked.

(iv) A $300 million Pilot Green Transport Fund was set up in March 2011 to encourage the testing out of green and low-carbon transport technologies by transport operators.

(v) Active promotion is being made for the wider use of electric vehicles (EVs) via financial incentives such as waiving their first registration tax up to the end of March 2017, close liaison with vehicle suppliers and manufacturers to encourage them to introduce EVs to Hong Kong; and provision of charging facilities and technical support, etc.

Reducing Emissions from Power Sector

Electricity generation is another major local air pollution source. The Government has adopted an aggressive strategy to control their emissions. We have banned all
new coal-fired power plants since 1997 and imposed stringent emission caps on all power plants since 2005. In 2008, we have amended the law to impose the emission caps for 2010 and beyond through statutory Technical Memoranda (TM). Four TM have now been promulgated for taking effect from 2010, 2015, 2017 and 2019 respectively with progressively stringent emission caps. In response to the TM, power companies had retrofitted their major coal units with emission control devices, including flue gas desulphurization systems and de-NOx systems, and increased the use of natural gas and low emission coal for electricity generation to reduce emissions. As a result, the emissions from the power sector have been reduced by 38% to 73% from 1997. Compared to the First TM, the Fourth TM will further tighten the emission caps of the emissions by about 40 to 60%.

Since 90% of our electricity is consumed in buildings, improving their energy efficiency is important for reducing emissions from power plants. To enhance building energy efficiency, we have made it a statutory requirement for both new buildings and existing buildings undergoing major retrofitting works to comply with the Building Energy Codes. Commercial buildings are further required to conduct energy audits once every ten years. To promote energy efficiency and conservation, we are developing a district cooling system in the Kai Tak Development and we have put in place both voluntary and mandatory Energy Efficiency Labelling Schemes.

Cutting emissions from vessels
The success in reducing the emissions of land-based sources has made vessels the greatest emission contributor in Hong Kong. To combat emissions from vessels, we introduced on 1 April 2014 a statutory cap on the sulphur content of marine diesel at 0.05%, a 90% reduction from the previous ceiling of 0.5%. A new legislation is in the pipeline to mandate ocean-going vessels at berth to switch to cleaner fuel, starting in 2015. Also, we are exploring the options to install onshore power supply facilities in Kai Tak Cruise Terminals and work with the Guangdong Government to jointly implement “fuel switch at berth” at PRD ports and, in the longer term, set up an Emission Control Area in PRD waters.

Other Emission Sources
Volatile organic compounds (VOC) could react with nitrogen oxides emitted from vehicles and other sources under sunlight to form ozone and photochemical smog. To reduce VOC emissions, we have been controlling petrol vapour emissions from petrol filling stations, regulating the VOC content of a wide-range of products including paints, printing inks, adhesives, sealants and consumer products.

In addition, we have mandated the use of ultra-low sulphur diesel for industrial and commercial processes. We are also drafting a legislation to control the emissions from non-road mobile machinery, which are mainly used in construction sites, the airport and container terminals to further reduce our local emissions.

Regional Collaboration
Our air quality could be under significant regional influence. We have attached great importance to cooperation with GD to reduce emissions in the PRD region.

Back in 2000, we set up a Joint Working Group on Sustainable Development and Environmental Protection with our GD counterpart to tackle our regional environmental issues. A consensus was reached in 2002 to jointly reduce emissions of the major air pollutants in the PRD region by 20% to 55% by year 2010, using 1997 as the base year. To further improve regional air quality, a new set of PRD emission reduction targets was agreed for 2015 (-5% to -25%) and 2020 (ranges set between -15% and -75%), as compared with 2010 emission levels. Both governments will conduct a mid-term review in 2015 to assess the state of socio-economic development at the time and the progress made in emission reduction, with a view to finalising the emission reduction targets for 2020.

To encourage and facilitate Hong Kong-owned factories in the PRD region to adopt cleaner production technologies and practices, thereby contributing to improving the regional air quality, we have also been implementing a Cleaner Production Partnership Programme since April 2008 with the GD authority.

To strengthen the collaboration in the PRD region, we have signed with GD and Macao in September 2014 a Co-operation Agreement on Regional Air Pollution Control and Prevention. Among others, three parties will:
- jointly release regional air quality information;
- take forward air pollution control and prevention measures;
- co-operate in scientific environmental studies; and
- fostering exchanges and promotion activities on environmental technology among three sides.

Improvement in air quality trend
Our control efforts have started to bear fruits. From 2006 to 2013, the ambient levels of sulphur dioxide ($SO_2$) and respirable suspended particulates (RSP) measured at our local air quality monitoring network reduced by 41% and 13% respectively while the ambient level of nitrogen dioxide ($NO_2$) increased slightly by 4%. During the same period, the concentration of $SO_2$ and RSP at roadside showed a reduction of 48% and 24% respectively while the concentration of $NO_2$ increased by 25%. Control of $NO_2$ therefore, is now our priority. With the completion of the additional measures mentioned above to target at the roadside $NO_2$ problem and the continued effort with GD to improve regional air quality, we expect that our roadside $NO_2$ levels would be brought down significantly.

Way Forward
Air pollution will affect public health. The Government is fully committed to improving our air quality to safeguard public health. In addition to the end-of-pipe reduction efforts, we are also considering how to help people reduce their daily exposure to air pollutants. In this connection, we look forward to a closer collaboration with the health and medical professionals in providing more useful information and advice to the public, in particular, those who are vulnerable to air pollution, to minimise their health risk arising from air pollution.
To be a smart citizen in health protection and combating air pollution

Ms Sum-yin KWONG

Chief Executive Officer, Clean Air Network

Hong Kong’s air quality has been worsening since the mid to late nineties, and in present day harmful levels of pollutants still prevail in the city’s densely populated urban areas, putting citizens at risk as a result. Air pollution is the city’s biggest public health crisis, killing more than 3,000 people a year in average. In 2013 the city’s air pollution caused at least eight deaths a day and in 2012 there were only 69 safely breathable days in the year. These could be avoided by focusing on reducing the emissions Hong Kong generates.

Air pollution is hurting us in more than one way; it is hurting us financially, economically and physically. Financially, it is draining our pockets. In 2013 air pollution cost Hong Kong almost $39.4 billion. The reason it has come to this amount is from the productivity that was lost due to sick days, doctor/hospital visits and avoidable deaths. This is the result of the rising problem of air pollution.

Secondly, air pollution is also hurting Hong Kong’s competitive economy. In terms of quality of life, Singapore consistently ranks above Hong Kong, and is the result of such pollution. This has hurt Hong Kong’s ability to attract and retain locals or even expatriates. A 2011 survey showed that one in four people in Hong Kong had considered moving away due to the harmful air quality, and these individuals were predominantly highly-educated professionals.

The most important of the three is public health. Air pollution affects everyone daily, be it inside a building or the outside environment, it will still affect our health. Some examples of its effects on health can vary from asthma to an increase risk of cancer and suffering a stroke.

A common misconception some people think is that almost all of the air pollution comes from Southern China, however a study by the HK University of Science and Technology shows that 53 percent of the time, the significant portion of the dirty air in Hong Kong is locally generated and not from Southern China.

Hong Kong’s own air pollution is mainly contributed by motor vehicles, marine vessels and power plants. There are two challenges Hong Kong faces, local street-level pollution and regional smog. Street-level pollution is usually caused by diesel vehicles, trucks, buses and light buses and the smog is caused by a combination of pollutants mainly from motor vehicles, industrial factories and power plants located in Hong Kong and the Pearl River Delta. These factors are what worsen the air quality in Hong Kong.

The citizens who do not know what can harm them are usually the most affected, in order to protect yourself and the people around you; gaining knowledge on the new AQHI system is the first step towards widening your perspective on air pollution.

The new Air Quality Health Index (AQHI) was introduced on December 30th 2013 to replace the Air Pollution Index (API). A joint study by public health and air science experts from CUHK and HKUST, as well as the EPD showed that replacing the API with a health risk-based AQHI system can provide better communication on health risk and information on the possible risks to health from exposure to different levels of air pollution in the outdoor environment. The AQHI system takes into account the Air Quality Guidelines (AQG) for air pollutants, developed by the World Health Organization (WHO), as well as the local hospital admissions risk.

The AQHI is a method of communicating health risks posed by air pollution to the general public and can provide the essential information on the effects of health from exposure to different amounts of pollution. It has many strengths in comparison to the old system, for example, the AQHI takes into account the combined effects of the air pollutants in Hong Kong and uses a 3-hour dynamic average calculating the pollutant concentrations, this allows it to closely follow the air quality and provide accurate health risk warnings to the public.

There are a number of ways the public can receive hourly forecasts on air quality, phone apps like EPD’s AQHI app or AQHI alert wizard can be downloaded onto mobile devices or you can also download Clean Air Network’s real-time air quality overview (HKCAN) from iTunes.

These apps collect air pollution data hourly and are analysed to give real-time levels of risk; during days of high pollution the AQHI will calculate the risk level and should it pass a certain level, warnings and advice are given to mobile devices recommending less exercise outdoor and to steer clear of heavy traffic areas.

To combat air pollution in Hong Kong, the population must know the cause and effect of these harmful emissions. A singe NGO is not enough to urge the government to opt for a policy change, what is required are individuals of the public voicing their concerns for air pollution and the effects on their families and friends. With enough public support, the possibilities of encouraging the government to change policies for the better will be great.

On 28 March 2013, The Environmental Bureau released “A Clean Air Plan for Hong Kong” which set out in detail various measures to mitigate and lessen emissions from transport vehicles, marine vessels and power
plants. Furthermore the Bureau has also strengthened its collaboration with Guangdong in order to counter regional pollution.

One significant policy aimed to lessen vehicle emissions was providing HK$10 billion to subsidise the owners of outdated vehicles, and was given two options to either submit their vehicle for scrapping or, to receive a higher amount to replace the old vehicle with a newer less polluting vehicle. The plan aims to remove 88,000 polluting vehicles off the roads by 2019, with pre-Euro and Euro I vehicles by 2016 and Euro II by 2017.

Whilst this is a good attempt to reduce emissions, it is still considered an end-of-pipe solution; a method implemented at the last stage of a process before waste is either disposed or delivered. The Government needs to shift away from this habit and focus on fixing the initial problem instead of ignoring the cause and only lessening the end result. An example of non-end-of-pipe methods would be to reorganise bus routes, which results in shorter travel time, easy interchanges and good service, so as to improve roadside air quality.

Low Emission Zones (LEZ) is also a good step towards improving air quality; it is designed to restrict or deter heavy polluting vehicles from busy districts. Since 2011, franchised bus companies have been supported to use cleaner buses for running through the preliminary LEZ in Central, Causeway Bay and Mong Kok. Another project being formulated is the “Green Oasis” in Central; Des Voeux Road is recommended to be transformed into a tram-pedestrian precinct. The roadside air quality in Central is very poor as there are no trees along the road in addition to a lack of greening, and a very distinct canyon effect created by the tall buildings from both sides which can circulate air in the same area, accumulating dirty pollution released by vehicles; this contributes to a hot and suffocating concrete urban jungle. Freeing the district from all transport except for trams will encourage walking thus improving air quality.

Clean Air Network is an independent NGO that aims to inform the population in Hong Kong about the effects of air pollution in order to gain support and urge the government to accept policies related to air quality. Raising awareness is one of our main objectives, and it is done through regular use of social media to share updates on Hong Kong’s current air pollution-related issues. Additionally CAN also gives talks at educational institutions, maintaining close relations with student groups at many universities.

CAN also encourages the involvement of public health officials in devising air quality policy by bringing the issue of air quality onto the campaign agendas of politicians. Hong Kong as we know it has very bad air quality, at CAN we strive to share such important information to the population and widen their horizons on the air crisis we are fighting today.

However, CAN is not able to voice their opinion onto everyone, and this is where we are asking the citizens of Hong Kong to help raise awareness, to be self-aware about the harm air pollution is causing them, and to enlighten others of the effects as well as gaining insight on possible policies to combat air pollution that can urge the government to make the necessary changes.

Together, we can push for policy change for the better of Hong Kong and its citizens.

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**Dermatological Quiz**

**Dr Lai-yin CHONG**

MBBS(HK), FRCP(Lond, Edin, Glasg), FHKCP, FHKAM(Med)

Specialist in Dermatology

This 70-year-old Chinese man had rapid onset of pruritic hyperpigmented skin lesions over the face, neck and intertriginous areas for three months (Figs. 1-2). There were also involvements of his palms and oral mucosal area. Recently he also noticed anorexia, weight loss and malaise. Past health was good otherwise.

**Questions:**

1. What is your diagnosis of his skin lesions?
2. What are the two main groups of underlying causes of this cutaneous disease?
3. Which underlying cause is more likely in this patient and why?

*(See P.40 for answers)*
How to fight air pollution: The London experience

Prof Frank J. KELLY

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Introduction

Air pollution has been a serious problem in London since the 12th Century with the introduction of bituminous coal into the city. This affordable and abundant form of energy was initially used for manufacturing before becoming a common domestic fuel. By the late 1700’s, coal was fuelling the industrial revolution in addition to millions of domestic fires. As a consequence, London’s appalling mixture of fog, smoke and sulphur dioxide emissions became world-famous and in 1905, during a public health lecture by Harold Des Voeux, a London physician and prominent advocate for smoke abatement, the lethal mix earned the term “smog”. Concern over the health effects of London’s poor air quality also dates back over many centuries. In 1661 the diarist John Evelyn presented Charles II with a treatise on the problem, suggesting that smoke pollution would shorten the lives of Londoners. Nearly two hundred years later, an article in The Lancet stated that ‘The air of this great city is, as all know too well, polluted by a variety of noxious gases and vapours diffused or held in solution’. The devastating health effects brought about by smog became evident in December 1952, when a toxic combination of London’s cold, motionless smoggy air brought about the worst pollution disaster in history. This great killer smog episode claimed an estimated 4,000-12,000 premature deaths and increased morbidity from cardiorespiratory causes. It was also the impetus for the 1956 Clean Air Act, the major focus of which curtailed domestic coal burning in London and other major cities in the UK. The implementation of smokeless zones, controls imposed on industries, increased availability and use of natural gas and changes in the industrial and economic structure of the UK led to a considerable reduction in concentrations of smoke and sulphur dioxide between the 1950’s and the present day.

Air Pollution in London in more recent times

In December 1991 a severe wintertime air pollution episode occurred in London, characterised by unprecedented levels of benzene, carbon monoxide (CO), oxides of nitrogen (NOx) and in particular, nitrogen dioxide (NO2) - all components of motor vehicle exhaust. In response to this, new air quality monitoring sites were established in and around London and the equipment base of existing sites extended. Continuous monitoring of particles with an aerodynamic diameter of 10 µm or smaller (PM10) began to replace the original BS measurements. During the 1990’s and early 2000’s the airborne particulate and lead concentrations declined steadily following the phase-out of lead in petrol, whilst CO, benzene and 1,3-butadiene also fell dramatically owing to the mandatory implementation of three-way catalysts and evaporative canisters in petrol-engined vehicles. In turn, the reduction in volatile organic carbon (VOC) emissions produced a decline in the peak intensity of photochemical episodes. In contrast, the annual percentage reductions in NOx levels (again achieved through the implementation of three-way catalysts on petrol-engined motor vehicles) were substantially less than those achieved for CO and VOCs. This is because of the substantial and growing contributions to NOx emissions from diesel-engined motor vehicles. The increasing use of diesel-engined vehicles also means that PM is still of major concern despite the reduction in BS levels. Concentrations of PM10, in fact, declined during the 1990’s but these trends have slowed down and during the 2000’s, levels have remained constant. So despite the air quality gains achieved in previous decades, like many other large cities around the world, London continues to experience high levels of air pollution, owing to a combination of mobile sources and regional background.

Air quality strategy

In view of widespread public concern about the health effects of air pollution, in 2002 the Mayor of London launched an Air Quality Strategy, entitled Cleaning London’s Air. This strategy set out policies to move towards the point where pollution no longer poses a significant risk to human health. The primary focus of the strategy is the reduction of pollution from road traffic since this is the main source of the pollutants of concern from within the city. In 2003, emissions from road transport contributed to approximately 40% of NOx emissions and to 66% of PM10 emissions in the Inner London area. A reduction in London’s road traffic emissions was to be achieved in two ways. First, through a decrease in the number of vehicles on the road, and second through reduced emissions from individual vehicles (i.e. modernisation of the fleet vehicle stock). To help achieve the first aim, the Mayor introduced a Congestion Charging Scheme (CCS) in central London in February 2003. One of the measures to tackle the second aim was a London-wide Low Emission Zone (LEZ), which was introduced in February 2008.
The congestion charging scheme in London

The congestion charging scheme (CCS) in London is a vehicle-charging scheme covering approximately 22 km² or 1.4% of the Greater London Area, and containing some of the most congested conditions in the capital. Vehicles crossing a cordon line to enter the congestion charging zone (CCZ) between the weekday hours of 07:00-18:00, termed the congestion charging hours (CH), pay a daily charge of what was originally £5 (60HKD), but in July 2005 this was increased to £8 (96HKD) and then in July 2013 £10/day (120HKD). Assisted by revenue from the CCS, parallel improvements in the fleet of public transport vehicles and traffic management have been implemented to accommodate the shift in travel patterns following the introduction of the charging scheme as well as continued growth in demand. The principal traffic and transport objectives have been met, mirroring the effectiveness of similar schemes in Singapore, Stockholm and Norway.6-8 Similar road pricing schemes have been considered for other UK cities and it is likely that traffic zonal payment schemes will become more common elsewhere in the world. Indeed, Milan introduced such a scheme on a test basis at the beginning of 2007 to address the city’s severe air pollution and traffic problems, whilst New York came close to becoming the first major American city to introduce a traffic congestion charge in 2008. The CCS in London is therefore a likely forerunner in what may become a powerful and widely adopted approach to traffic demand management.

The CCS and air quality in London

There is considerable interest in determining whether the reduction in congestion and traffic achieved in London following the implementation of the CCS has had a positive impact on the air quality. In principle the CCS, in reducing congestion and the numbers of vehicles crossing the cordon, should reduce emissions and improve air quality both inside and outside the CCZ. However, one would not expect the CCS to elicit more than a small effect on air quality within the zone, considering the charge has brought about a relatively moderate effect on traffic (approx 20% reduction in vehicles entering the CCZ) in a small (1.4% Greater London) area of London. This is particularly the case when one considers the host of other contributors to emissions both within London and from Regional background. For example, changes in association with the CCS in traffic flows and vehicle speeds have the potential to produce both increases and decreases in PM and NOx emissions. In addition, improvements in public transport vehicles (and with this, an increase in the number and flows of diesel-powered buses and taxis entering the CCZ), the introduction of traffic management measures and the magnitude and location of road works are also likely to have air quality impacts. In fact the CCS has resulted in no or only a very small improvement in air quality. 9

The London-wide Low Emission Zone

The key driver for the introduction of the LEZ was the need to improve the health and quality of life of people who live and work in London through improving air quality.10 In addition, the scheme was designed to move London closer to achieving national and EU air quality objectives for 2010. The LEZ was designed to tackle these objectives by restricting the entry of the oldest and most polluting vehicles across Greater London, an area of 2,644km².10 The scheme operates 24 hours a day, 365 days a year, using cameras to identify the registration numbers of vehicles and the Driver and Vehicle Licensing Agency (DVLA) database to identify a vehicle’s emissions. The LEZ applies to diesel-engine heavy goods vehicles (HGVs), buses and coaches, larger vans and minibuses. The LEZ initially targeted vehicles with disproportionately high emissions (lorries over 12 tonnes), obliging them to meet modern Euro emission standards which set limit values for exhaust emissions for new vehicles; see (http://www.tfl.gov.uk/roadusers/lez/vehicles/2535.aspx). This first phase of the LEZ was followed by the inclusion of lighter HGVs, buses and coaches in July 2008, while large vans and minibuses were to be included from October 2010. However, Mayor Johnston decided to delay this 3rd phase by 18 months due to economic conditions. Specifically the LEZ required heavy duty vehicles to meet the Euro III emission standard for PM10, and then Euro IV in 2012. A standard for NOx was not included as part of the scheme as there were too many unresolved issues about certification and testing of NOx abatement equipment. Cars are not included in the scheme but may be at a later date.

The stick and the carrot

In order to deter high-polluting vehicle use, and provide an incentive to operators to upgrade their vehicles, operators of vehicles that do not meet the LEZ standards are required to pay a charge of £200 (2,400HKD; lorries, coaches and buses) or £100 (1,200HKD; vans and minibuses) for each day they are driven into the zone. Furthermore, should an operator of a non-compliant vehicle not pay the daily charge, then following the service of a penalty charge notice, penalty charges of £1,000 (12,000HKD; lorries and buses) or £500 (6,000HKD; vans and minibuses) apply; reduced by 50% if paid within 14 days or increased by the same increment if not received within 28 days.

LEZ’s elsewhere in the world

Low emission zones for freight vehicles have already been successfully implemented in Sweden since 1996 when Stockholm, Gothenburg and Malmo introduced ‘Environmental Zones’ in their city centres, to improve air quality and reduce noise.11 Tokyo has been a Low Emission Zone since October 2003 and again has been successful in reducing emissions of HGVs. More recently Berlin has implemented a LEZ in the central city area while Oxford, UK introduced a scheme for bus operators. Such schemes are also being considered by other UK and European cities. The London LEZ is the largest such zone in the world and the first to use a charging mechanism rather than an outright ban.

Potential health and environmental impacts of the LEZ

Poor air quality is known to have marked effects on
certain, sensitive individuals. It also seems that the young and the elderly are generally more sensitive to air pollution. In that the LEZ was implemented across the whole of Greater London, an area in which more than 8 million people live and work, suggests the scheme has the potential to bring about a range of health benefits for London residents. When introduced the LEZ was predicted to reduce the emission of PM$_{10}$ in London by 64 tonnes (or 2.6%) in 2008, and in turn, decrease the area of Greater London that exceeds the EU air quality limit values for PM$_{10}$ of 40µg/m$^3$. In practice the benefits have been smaller than expected and difficult to identify as EURO emissions databases have been found to be inaccurate in reflecting real world driving conditions. Notwithstanding these issues the LEZ and the CCS together provide the means by which mobile emissions can be controlled in urban areas and when more rigorously applied should lead to better air quality in London and, in doing so, provide a health benefit to Londoners.

References
3. The Lancet, Saturday 1856:139-140.
孟澈雅製：現代竹木文玩

何孟澈醫生

唐代王維，官居右丞，辋川別業，蕩然無蹤，獨以詩存。宋代范仲淹岳陽樓記，歐陽修醉翁亭記，至今學子，琅琅上口；二公位列宰相，政績湮沒，而以文存。有元一季，帝國雄踞，未及一世紀，金甌瓦解，唯忽必烈「瀆山大玉海」，今於北京團城存焉。清史稿目錄，所載名公鉅卿，十居其九，聞所未聞，三數百年耳，何消亡之速也。

余不敏，習泌尿外科，手作之術，施諸血肉之軀，三五十年間，亦將灰飛煙滅。有慮及此，故自上世紀九十年代，工餘擷取古今名蹟，初承北京王世襄先生指點，特倩藝術家，精製文玩，上用敝款敝章，庶幾來日，如王羲之蘭亭序云：「後之視今，亦如今之視昔」，余名得賴竹木以存，不亦幸矣。

王世襄先生題水仙留青竹臂擱成對	范遙遙青刻 (1997)

歲朝清供，水仙獨佔鰲頭，「金盞玉臺、紫宸重器」，實非過譽。曾見宋趙孟堅水仙圖卷，及明剔紅水仙圓盒，因思曷不依式製竹筆筒，可作案頭之用。王世襄先生 ( 王老 ) 覆示，以為新製竹筆筒易裂、不如取金北樓先生刻水仙臂擱拓本、一刻水仙，一刻自書「憶江南」水仙詞一闋，作臂擱更為可取。

王老與余，遂具函常州范遙遙青先生，請刻臂擱，三月而成。北樓先生水仙臂擱，原為陰刻；范先生改作留青陽刻。畫書合璧，精雅無比，水仙下用印「回春」，因余業醫，語帶雙關也。

水仙留青竹臂擱及拓本 范遙遙刻 (1997) 傅萬里拓

宋代趙孟堅水仙圖紫檀筆筒 傅稼生刻 (2012)

近年，又得紫檀筆筒，不虞開裂，遂請北京榮寶齋傅稼生先生，刻宋代趙孟堅水仙圖，姿卓約，直如宋劉邦直詩云：「仙風道骨今誰有，淡掃蛾眉參一枝」。

王老藏有清代甘士調指畫松鷹圖，與雪界翁畫不相伯仲，余甲申歲曾呈敬觀儷松居藏松鷹圖七絕一首：

昂首不鳴歇楚庭
松颠峭立影伶仃
長空奮翅三千里
直擊九天勇摘星

此詩亦可為雪界翁畫鷹寫照也。
飛天伎樂竹筆筒 周漢生刻 (2011)

20 世紀 90 年代，於倫敦維多利亞阿爾拔博物館，見北魏石刻飛天，反彈琵琶。後又見青州龍興寺遺址出土北魏佛像背光，伎樂飛天羅列，氣勢不凡。

2008 年於武漢周漢生先生府上，見有大筒材。兩年後，忽有靈感，求刻竹筆筒。先生覆函曰：「擬將飛天圖案集中於筆筒腰部作一圈陷地深刻（如古埃及、古中國摩崖石刻均有此作法），其上下天地頭保留原材不動，雖仍為高浮雕卻不致傷材，亦較能體現石刻原作的拙味。」余欣表贊同。

一月後漢生先生又附上三個飛天初稿，一彈琵琶，一撥箜篌，一吹排簫；「前後相隨可循環往復觀賞…又間以魏常見的蓮花紋、雲紋，力求畫面有些主從相屬的層次感。」

筆筒設計，漢生先生捨易取難，用兩節竹筒成之，「刻時節內竹絲上下俱反順為逆，須時時小心變換用刀方向，此亦前人所以不用有節之材的原因。」獅子提象，信是力作。

元代倪瓚竹枝圖花梨筆筒 傅稼生刻 (2007)

二零零九年香港回歸假日，晴窗之下把玩筆筒，偶得四句：　

窗外雲林粉本 　倪瓚，號雲林。　
案頭枝葉分明 　「世說新語、任誕」：王子猷嘗暫寄人空宅住，便令種竹，成問暫住何煩爾，王嘯咏良久，直指竹曰：何可一日無此君。　
獨愛此君勁節 　伴余天雨天晴　
　
黃花梨大筆筒，購得多年，闊置架上。紋理較粗，刻此墨竹，最為相宜。唐張頵歷代名畫記，載謝赫論畫之六法，其五曰「經營位置」。倪畫之上，原有乾隆御詩，礙人眼目，故移置倪題之後，則畫面更趨統一完美，惟此舉若在二百年，可坐大逆不敬之罪，定株連甚廣。御印六枚：「乾隆御覽之寶」、「古稀天子」、「三希堂精鑑璽」、「宜子孫」、「乾」、「隆」，皆稼生先生精心之作。　

黃花梨大筆筒，購得多年，闊置架上。紋理較粗，刻此墨竹，最為相宜。唐張頵歷代名畫記，載謝赫論畫之六法，其五曰「經營位置」。倪畫之上，原有乾隆御詩，礙人眼目，故移置倪題之後，則畫面更趨統一完美，惟此舉若在二百年，可坐大逆不敬之罪，定株連甚廣。御印六枚：「乾隆御覽之寶」、「古稀天子」、「三希堂精鑑璽」、「宜子孫」、「乾」、「隆」，皆稼生先生精心之作。
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- **Monday, January 15**: HKMA Kowloon West Community Network – The Update Management on Atopic Dermatitis
- **Monday, January 16**: HKMA Tai Po Community Network – Bariatric and Metabolic Surgery: Does it Work?
- **Monday, January 17**: HKMA Council Meeting
- **Tuesday, January 18**: HKMA New Territories West Community Network – Male LUTS Beyond BPH Management
- **Tuesday, January 19**: HKMA Kowloon East Community Network – What’s New on COPD Management?
- **Tuesday, January 20**: HKMA Hong Kong East Community Network – What’s New on COPD Management?
- **Wednesday, January 21**: HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2013 – Bleeding Tendency
- **Thursday, January 22**: HKMA Kowloon City Community Network – (1) Update on Pharyngitis Management; (2) Understanding on Acid Pocket
- **Thursday, January 23**: HKMA Shatin Doctors Network – The Regenerative Power of Stem Cell in Cancer Therapy
- **Saturday, January 3**: Refresher Course for Health Care Providers 2014/2015 – Speech therapy in primary care
<table>
<thead>
<tr>
<th>Date / Time</th>
<th>Function</th>
<th>Enquiry / Remarks</th>
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<tbody>
<tr>
<td>2 FRI 8:00 AM</td>
<td>Joint Surgical Symposium - New Development in Colorectal Surgery</td>
<td>Department of Surgery, Hong Kong Sanatorium &amp; Hospital Tel: 2835 8698 Fax: 2892 7511 1 CME Point (Active) Ms. Nancy CHAN Tel: 2527 8898</td>
</tr>
<tr>
<td>6 SUN 1:00 PM</td>
<td>HKMA Kowloon West Community Network – The Update Management on Atopic Dermatitis</td>
<td>Miss Hana YEUNG Tel: 2527 8285 1 CME Point</td>
</tr>
<tr>
<td>8 THU 1:00 PM</td>
<td>HKMA New Territories West Community Network – Male LUTS: Beyond BPH Management</td>
<td>Miss Hana YEUNG Tel: 2527 8285 1 CME Point</td>
</tr>
<tr>
<td>9 FRI 1:00 PM</td>
<td>HKMA Shatin Doctors Network – The Regenerative Power of Stem Cell in Cancer Therapy</td>
<td>Ms. Mandy LO Tel: 2859 8759 1 CME Point</td>
</tr>
<tr>
<td>10 SAT 2:15 PM</td>
<td>Refresher Course for Health Care Providers 2014/2015 – Speech therapy in primary care</td>
<td>Ms. Clara Tsang Tel: 2354 2440 2 CME Points</td>
</tr>
<tr>
<td>13 TUE 1:00 PM</td>
<td>HKMA Central, Western &amp; Southern Community Network – Diagnosis and Treatment of Axial-Spondylarthropathy (Axial-SpA)</td>
<td>Miss Hana YEUNG Tel: 2527 8285 1 CME Point</td>
</tr>
<tr>
<td>15 THU 8:00 AM</td>
<td>FMSHK Executive Committee Meeting</td>
<td>Ms. Nancy CHAN Tel: 2527 8898</td>
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<tr>
<td>16 FRI 1:00 PM</td>
<td>HKMA Kowloon City Community Network – (1) Update on Pharyngitis Management; (2) Understanding on Acid Pocket</td>
<td>Miss Jess LAU Tel: 2507 9969 1 CME Point</td>
</tr>
<tr>
<td>20 TUE 1:00 PM</td>
<td>HKMA Kowloon West Community Network – Update on Management of Hyperuricaemia and Gout</td>
<td>Miss Hana YEUNG Tel: 2527 8285 1 CME Point</td>
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<tr>
<td>22 THU 1:00 PM</td>
<td>HKMA Kowloon East Community Network – Update on Management of Hypertension</td>
<td>Ms. Candice TONG Tel: 2527 8285 1 CME Point</td>
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<tr>
<td>5 MON 7:30PM</td>
<td>A man with recurrent scrotal tumour</td>
<td>Tammy Hung Tel: 9609 6004 1 CME Point</td>
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<tr>
<td>6 TUE 1:00 PM</td>
<td>HKMA Kowloon West Community Network – The Update Management on Atopic Dermatitis</td>
<td>Miss Hana YEUNG Tel: 2527 8285 1 CME Point</td>
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<tr>
<td>6 TUE 1:45 PM</td>
<td>HKMA Tai Po Community Network – Bariatric and Metabolic Surgery: Does it work?</td>
<td>Ms. Ashley CHAN Tel: 8209 9604 1 CME Point</td>
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<tr>
<td>6 TUE 8:00 PM</td>
<td>HKMA Council Meeting</td>
<td>Ms. Christine WONG Tel: 2527 8283</td>
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<tr>
<td>8 FRI 1:00 PM</td>
<td>HKMA New Territories West Community Network – Male LUTS: Beyond BPH Management</td>
<td>Miss Hana YEUNG Tel: 2527 8285 1 CME Point</td>
</tr>
<tr>
<td>8 FRI 1:00 PM</td>
<td>HKMA Kowloon East Community Network – What’s New on COPD Management?</td>
<td>Miss Hana YEUNG Tel: 2527 8285 1 CME Point</td>
</tr>
<tr>
<td>8 FRI 2:00PM</td>
<td>HKMA Structured CME Programme with Hong Kong Sanatorium &amp; Hospital Year 2015 – Bleeding Tendency</td>
<td>HKMA CME Department Tel: 2527 8452 1 CME Point</td>
</tr>
<tr>
<td>9 FRI 1:00 PM</td>
<td>HKMA Shatin Doctors Network – The Regenerative Power of Stem Cell in Cancer Therapy</td>
<td>Ms. Mandy LO Tel: 2859 8759 1 CME Point</td>
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<td>10 SAT 2:15 PM</td>
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<td>Ms. Clara Tsang Tel: 2354 2440 2 CME Points</td>
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<td>Miss Hana YEUNG Tel: 2527 8285 1 CME Point</td>
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<tr>
<td>15 THU 8:00 AM</td>
<td>FMSHK Executive Committee Meeting</td>
<td>Ms. Nancy CHAN Tel: 2527 8898</td>
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**Calendar of Events**

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<tr>
<th>Date</th>
<th>Time</th>
<th>Function</th>
<th>Enquiry / Remarks</th>
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<tr>
<td><strong>22 THU</strong></td>
<td>8:00 PM</td>
<td><strong>FMSHK Foundation Meeting</strong>&lt;br&gt;Organiser: The Federation of Medical Societies of Hong Kong; Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong</td>
<td>Ms. Nancy CHAN&lt;br&gt;Tel: 2527 8898</td>
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<tr>
<td><strong>23 FRI</strong></td>
<td>1:00 PM</td>
<td><strong>HKMA Shatin Doctors Network – (1) Update on Pharyngitis Management; (2) Understanding on Acid Pocket</strong>&lt;br&gt;Organiser: HKMA Shatin Doctors Network; Chairman: Dr. MAK Wing Kin; Speakers: Dr. CHAN Yap and Dr. NG Ka Yan; Venue: Jasmine Room, Level 2, Royal Park Hotel, 8 Pak Hok Ting Street, Shatin</td>
<td>Miss Jess LAU&lt;br&gt;Tel: 2507 9969&lt;br&gt;1 CME Point</td>
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<tr>
<td><strong>24 SAT</strong></td>
<td>7:00 PM</td>
<td><strong>10th HKMA Sports Night</strong>&lt;br&gt;Organiser: The Hong Kong Medical Association; Chairpersons: Dr. CHAN Hau Ngi, Kingsley and Dr. IP Wing Yuk; Venue: The Grand Hall, 28 Harbour Road, Wanchai, Hong Kong</td>
<td>Mr. Andie HO&lt;br&gt;Tel: 2527 8285</td>
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<tr>
<td><strong>30 FRI</strong></td>
<td>1:00 PM</td>
<td><strong>HKMA Yau Tsim Mong Community Network – Right Treatment, Right Patient and Right Prostate</strong>&lt;br&gt;Organiser: HKMA Yau Tsim Mong Community Network; Chairman: Dr. CHENG Kai Chi; Speaker: Dr. WONG Tak Hing, Bill; Venue: Diamond Ballroom, 1, B1, Eaton, Hong Kong, 380 Nathan Road, Kowloon</td>
<td>Ms. Candice TONG&lt;br&gt;Tel: 2527 8285&lt;br&gt;1 CME Point</td>
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**Federation News**

**Public Talk for Overcoming Depression and Suicide Prevention**

The Public Talk for Overcoming Depression and Suicide Prevention was held at the Federation Lecture Hall on 30 Nov 2014. It was our pleasure and privilege to invite Dr Wai-him CHEUNG, Specialist in Psychiatry, who delivered a talk on “如何掃走抑鬱” and Ms Susanna CHOY, Clinical Psychologist at Princess Margaret Hospital, who delivered a talk on “痛苦可以走過如何預防自殺”. The participants’ active questioning in the Q&A section helped to complete a very successful & interactive seminar. The event concluded with Dr Yin-kwok NG, 2nd Vice President of the Federation, thanking the two speakers with souvenir presentations by Dr Raymond LO, President of the Federation.
Certificate Course on Healthcare Mediation

**Objectives:**
- To promote mediation skills in healthcare sector
- To reduce misunderstanding between healthcare workers and patients through the application of mediation skills
- To improve the interpersonal skills through systematic learning
- To understand the concept of mediation, win-win and interest-based resolution
- To communicate better with patients, relatives and colleagues

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<thead>
<tr>
<th>Date</th>
<th>Topics</th>
<th>Speakers</th>
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<tbody>
<tr>
<td>7 Jan</td>
<td>Mediation and healthcare</td>
<td>Dr. TSOI Chun Hing Ludwig</td>
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<td>Senior Medical Officer</td>
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<td>14 Jan</td>
<td>Negotiation skills and empowerment</td>
<td>Mr. LAI Tsz Kin Jacky</td>
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<td>Barrister-at-law</td>
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<td>21 Jan</td>
<td>Listening skills and use of body language</td>
<td>Prof. LAI Bo San Paul</td>
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<td>28 Jan</td>
<td>Perception check, paraphrasing and summarizing skills</td>
<td>Dr. DAI Lok Kwan David</td>
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<td>4 Feb</td>
<td>Reframing and facilitative skills</td>
<td>Dr. ONG Kim Lian</td>
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<td>11 Feb</td>
<td>Deadlock and impasse</td>
<td>Dr. CHAN Lap Wa</td>
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<td>Accredited Mediator</td>
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**Dates:** 7, 14, 21, 28 January and 4, 11 February, 2015 (Every Wednesday)
**Time:** 7:00 pm - 8:30 pm
**Venue:** Lecture Hall, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong
**Language Media:** Cantonese (Supplemented with English)
**Course Fee:** HK$750 (6 sessions)
**Certificate:** Awarded to participants with a minimum attendance of 70%
**Enquiry:** The Secretariat of The Federation of Medical Societies of Hong Kong
  Tel: 2527 8898    Fax: 2865 0345    Email: info@fmshk.org

CME / CPD (including HK Mediation Centre) Accreditation in application
A total of 9 CNE points for the whole course and the points will be awarded according to the number of hours attended
Application form can be downloaded from website: http://www.fmshk.org
Answers to Dermatological Quiz

1. Acanthosis nigricans (AN) The clinical appearance of these hyperpigmented lichenified papillomatous velvety lesions over the neck and intertriginous areas is typical of AN. Often multiple acrochordons (skin tags) are present as well.

2. Endocrinopathy and malignancy

   80% of AN are idiopathic or benign. The associated endocrinopathy includes diabetes, acromegaly and Cushing’s disease. Although the precise mechanism is unknown, insulin plays a significant role in the benign form, in which excess insulin is bound to IGF (insulin-like growth factor) receptors. In obese patients, it is also called pseudo-acanthosis nigricans and is a reliable cutaneous marker of hyperinsulinemia and insulin resistance. In patients with AN and sudden onset of acrochordons, 60-80% eventually turn out to be diabetic. In 2000, the American Diabetes Association also established AN as a risk factor for the development of diabetes in children.

   Malignancy-associated AN is a well established cutaneous marker of the paraneoplastic syndrome. At least 80% of the patients with malignant associated AN are over 40 years old. The association of it with gastric adenocarcinoma is well known, accounting for 50-60% of cases in this group. Therefore it is mandatory to have a detailed diagnostic work up to search for an underlying malignancy, especially gastric or bronchial carcinoma. Though the mechanism is even more unclear, it is postulated that a transforming growth factor-α produced by the underlying tumour may be related.

3. Underlying malignancy

   In practice, it is essential to differentiate between the benign and malignant forms. The useful clinical clues include age of onset, progression and extent, mucous membrane involvement and palmar involvement. In this patient, the advanced age, rapid course, oral mucosal involvement and palmar involvement (also known as triple palm with accentuated palmar dermatoglyphic ridges) raised a strong suspicion of an underlying malignancy. An upper gastro-intestinal endoscopy and biopsy was performed which showed adenocarcinoma of stomach in this patient.

Dr Lai-yin CHONG
MBBS(HK), FRCPE(Lond, Edin, Glasg), FHKCP, FHKAM(Med)
Specialist in Dermatology
Certificate Course on
Common Geriatric Problems

Objectives:
To update on practical approach to common problems in elderly patients

<table>
<thead>
<tr>
<th>Date</th>
<th>Topics</th>
<th>Speakers</th>
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<tr>
<td>12 Mar</td>
<td>Cognitive Impairment</td>
<td>Dr. AU YEUNG Tung Wai</td>
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<td>Specialist in Geriatric Medicine</td>
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<td>Convener, Brain Health Special Interest Group</td>
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<td>The HK Geriatrics Society</td>
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<td>19 Mar</td>
<td>Movement Disorder and Functional Impairment</td>
<td>Dr. Stanley TAM</td>
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<td>Specialist in Geriatric Medicine</td>
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<td>Co-convener, DM Special Interest Group</td>
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<td>The HK Geriatrics Society</td>
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<tr>
<td>26 Mar</td>
<td>Falls and Fractures</td>
<td>Dr. CHAN Tak Yeung</td>
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<td>Specialist in Geriatric Medicine</td>
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<tr>
<td>2 Apr</td>
<td>Blood Pressure Abnormalities</td>
<td>Dr. Stephen WONG</td>
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<td>Specialist in Geriatric Medicine</td>
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<tr>
<td>9 Apr</td>
<td>Chronic Obstructive Pulmonary Disease</td>
<td>Dr. KO Chi Fai</td>
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<td>Specialist in Geriatric Medicine</td>
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<td>16 Apr</td>
<td>Diabetes Mellitus</td>
<td>Dr. YEUNG Kwan Mo</td>
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<td>Specialist in Geriatric Medicine</td>
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Date: 12, 19, 26 March and 2, 9, 16 April 2015 (Every Thursday)
Time: 7:00 p.m. – 8:30 p.m.
Venue: Lecture Hall, 4/F., Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong
Language Media: Cantonese (Supplemented with English)
Course Fee: HK$750 (6 sessions)
Certificate: Awarded to participants with a minimum attendance of 70%
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CME / CPD Accreditation in application
A total of 9 CNE points for the whole course and the points will be awarded according to the number of hours attended.
Application form can be downloaded from website: http://www.fmshk.org
Help your COPD patients do more

Vannair™ pMDI (budesonide/formoterol)

- significantly improves lung function in 5 minutes\(^1,2\)
- shows faster onset of bronchodilation than salmeterol/fluticasone\(^1\)
- makes a significant improvement in breathing\(^2\)
- effectively prolongs time to exacerbation\(^2\)
- eases bronchoconstriction and controls inflammation\(^3\)