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Room 603
Hong Kong Academy of Medicine Jockey Club Building
99 Wong Chuk Hang Road
Aberdeen
Hong Kong

Tel 2871 8766
Fax 2556 9047
email enquiry@hkcp.org
College Website http://www.hkcp.org

The President´s Annual Report 2012

Patrick CK Li
President, HKCP

The Hong Kong College of Physicians is the statutory organisation responsible for overseeing physician training and setting the standard of internal medicine practice. Over the past year, the Education and Accreditation Committee has continued to refine and update the training requirements and assessment framework. Through the Professional and General Affairs Committee and the Specialty Boards, the College has worked closely with the Academy and our sister Colleges to provide technical advice to the Government on issues such as elderly care, dementia service and fitness to drive and promulgate accurate information on topics of health concern such as harmful use of alcohol and pain management to members of the public. The College has also maintained collaboration with overseas professional organisations through the National and International Committee for international benchmarking of our professional standard and the Joint HKCP Intermediate - MRCP (PACES) examination held twice yearly locally continued to be of high standard under the coordination of the Examination Committee. The Annual Scientific Meeting organised by the Scientific Committee is firmly established as the major event of the College and not only provides the opportunity to keep abreast of the latest medical advances but serves as the forum for outstanding trainees and Fellows to present their research work. The Membership Committee has admitted a total of 51 Members and 67 Fellows in the past year and the total number of Fellows now stands at 1509. With the crucial role of internal medicine in providing care for an ageing population with multiple chronic medical illnesses, the College will need to maintain its momentum in training young doctors in a career in internal medicine and its specialties.

Following last year's public attention on the heavy workload and suboptimal career progression for physicians working in the public sector, the Hospital Authority has introduced a number of measures to pacify the prevailing grievances and low morale among the doctors. These include an enhanced promotion scheme taking reference of the duration of specialist practice experience and a tiered on-call honorarium to recognise the hardship of long working hours for some specialties. While these measures have for the moment boosted staff morale and stabilised the wastage rate, the effect may not be sustainable and could result in intensified frustrations in the longer term. This is in particular the case for internal medicine which has among the greatest backlog of staff awaiting promotion.

Under the new scheme, the majority of the newly promoted Associate Consultants in Internal Medicine are still working as first-line doctors with many still having to take overnight calls. In addition, the new promotion scheme has deprived outstanding doctors of the opportunity of excelling themselves in their career progression. The College will continue to impress upon the Hospital Authority and the Food and Health Bureau the critical role of Internal Medicine in the future delivery of medical service to ensure their continued support to physician training and work with them to develop an appropriate manpower planning and service model that would enable physicians to provide quality care to their patients.

In 2011, the College has simplified the assessment framework for physician training to reduce hardship to the trainees who are already facing excessive workload. On the other hand, the criteria for passing the interim and exit assessment have continued to be refined to maintain the high standard of training requirement. Through close monitoring of the trainer eligibility, institution performance, timely remedial action for deficiencies identified during the interim assessment, and the fair and structured format of the exit assessment, the College has maintained a high pass rate in all the specialties. In recognition of the importance of a broad-based foundation to specialty training, the College has decided that all specialty trainees will be required to undertake dual training with either Advanced Internal Medicine or Geriatrics. In addition, physician specialists will be required to have achieved accreditation in either Advanced Internal Medicine or Geriatrics before they can be eligible to serve as trainer in their respective specialty. Completion of the Self Learning Tool jointly developed with the Hospital Authority is now a mandatory requirement for higher physician trainees to consolidate the clinical risk management component of Advanced Internal Medicine training.

The College places considerable emphasis on promoting and facilitating research by trainees and Fellows, including those working in the academic, public and private sectors. Through the input of the Neurology Specialty Board, the College had provided advice to the Food and Health Bureau on the thematic priorities for the newly established Health and Medical Research Fund. The College has also strongly voiced out its concern to the Department of Health about the possible negative impact of the proposed amendment to the Pharmacy and Poisons Ordinance on investigator-initiated clinical research. The College has for many years recognised original research through the Distinguished Research Paper Award for Young Investigators. This year, the Research Committee has launched the Young Investigator Research Grant which was awarded to 5 Members/Fellows of the College to facilitate them in embarking on their career in clinical research.

In view of the important function of the College in overseeing the progress of trainees and maintaining the standard of training, the process of registration as trainee, Member, Fellow and trainer of the College has been formalised during different milestones of the physician career. The Basic Physician Board, the Specialty Boards, the Membership Committee as well as the College Secretariat have maintained efficient processing of applications from trainees/specialist to ensure timely registration and commencement of the next phase of their career.

The College has also maintained communication with Fellows and Members through the Synapse and the College website. To enhance their opportunity of active participation in College affairs and sense of ownership, younger Fellows are appointed as Assistant Programme Director or Observer SMO/AC in the Specialty Boards, or recruited into the standing committees and different working groups. This will serve the purpose of familiarising them with the operations of the College and priming them to take up more important positions within the College. The College has also decided on a succession planning process with defined tenure and structured turnover in the Membership of the Specialty Boards.

I wish to extend my sincere appreciation to the Chairpersons and Members of the Committees and Boards for their contributions towards upholding the standard of physician training and supporting the various responsibilities and functions of the College. My sincere gratitude also goes to our two Vice-Presidents who have worked hard to review and update our training guidelines and assessment framework and maintain communication and collaborations with our international counterparts respectively. I am extremely grateful to our Honorary Treasurer for monitoring and maintaining the healthy financial position of the College. I would also like to thank our Council Members, in particular our Immediate Past President and Senior Advisor for their unfailing support and invaluable advice over the past year. Finally, I wish to thank our Honorary Secretary and the hardworking and dedicated secretarial staff in maintaining smooth operation of the College.

The President´s Address to New Fellows at the Hong Kong College of Physicians Conferment Ceremony 2012

Patrick CK Li
President, HKCP

Dr. Ko Wing Man, Secretary for Food and Health, Prof. Raymond Liang, President of the Hong Kong Academy of Medicine, Dr. Neil Dewhurst, President of the Royal College of Physicians of Edinburgh, President of Sister Colleges and their representatives, Fellows and Members of the College, Invited guests, ladies and gentlemen.

Today is an important occasion for the newly admitted Fellows and Members. On behalf of the College, I extend to you my congratulations on your achievement, which is a reflection of your dedication and hard work over the past years. I am pleased that you can share the joy with your family and loved ones. I hope that you will all continue to strive for excellence and professionalism in your future practice.

Today marks an important milestone in your career, as most of you are now accredited in a specialty in internal medicine, and are eligible for inclusion in the Specialist Register of the Medical Council. However, another equally important milestone lies ahead, as you will need to complete your training in advanced internal medicine before you can be eligible to serve as trainer in your specialty area.

Our College is unique within the Academy in having a dual higher physician training programme. I can only have admiration for our predecessors in the College Council who had the wisdom and foresight in designing this training pathway and the courage to stand firm in their belief in the face of dissenting views from many of our sister Colleges within the Academy at that time. I would like to take this opportunity to share with you my views on why dual training in advanced internal medicine together with your chosen specialty is important for your clinical practice and the future of medical service in Hong Kong.

When I undertook my physician training many years ago, most of my senior colleagues took pride in their competence in wide areas of internal medicine. They were able to independently diagnose and manage patients with disorders affecting different organ systems and were proficient in performing various diagnostic and therapeutic procedures such as endoscopy and cardiac pacing. However, the practice of internal medicine has transformed considerably since then. There has been an exponential rate of increase in our understanding of human physiology and pathogenesis of medical diseases and consequently the therapeutic options that are available have become increasingly sophisticated. Such rapid pace of medical advances makes it impossible for any individual clinician to maintain current knowledge in a wide range of medical specialty areas. There is thus a growing need for physicians to devote their career towards developing and maintaining competence in defined specialty areas.

With the formation of the Specialty Boards and development of

structured training programme, there are a growing number of accredited specialists. Over the past two decades, we have witnessed the evolving trend towards general physicians with special interest in a specialty area, and increasing number of them are devoting a considerable proportion of their practice to their respective specialty area. Some physicians have even undertaken sub-subspecialisation. For example, cardiology practice has diversified into interventional cardiology, cardiac electrophysiology, echocardiography and adult congenital heart disease. Likewise, many neurologists have focused their practice in stroke service, epileptology, movement disorders, neurophysiology or neuromuscular diseases. Such degree of focused expertise would undoubtedly be welcomed by patients with complicated medical conditions requiring tertiary care. With the increased focus on patient safety and call for credentialing for competence in diagnostic and interventional procedures, the trend towards specialisation will be expected to gather further momentum.

specialisation taken to the extreme could only be detrimental to patient care and the healthcare system

However, specialisation taken to the extreme could only be detrimental to patient care and the healthcare system. We are already witnessing increasing trend of cross referrals to other specialists within internal medicine. While complicated clinical conditions and highly technical therapeutic procedures are best undertaken by trained specialists, division of responsibility by organ system risks fragmentation of the patient care process. Each doctor would be treating the organ system instead of providing holistic care to the patient. Focusing solely on confined and technical aspects of care will diminish the art of medical practice. Unnecessary cross-referrals would generate duplication of work, result in delay in implementation of treatment, ultimately leading to deterioration in patient satisfaction.

This consideration is especially important with an ageing population, where the majority of patients have multiple co-existing acute and/or chronic medical problems. It would require broad experience in internal medicine to fully appreciate the interplay of multiple medical diseases, the interaction of different therapeutic interventions and to consider in the right perspective the individual medical problems in the overall context of the patient's physical status and prognosis.

In addition, the cost and efficiency of the healthcare system would be adversely affected by over-specialisation. Many overseas studies have demonstrated that for similar medical conditions, care by specialists is often more costly than that delivered by general physicians. We have also witnessed the manpower strain with resultant high call frequencies and long working hours for some highly specialised clinical departments with relatively restricted staffing numbers. To counter this, the Hospital Authority is considering service models that pool together doctors from related clinical departments for call duties to manage the doctor work-hours problem. Medical Departments should take note of such experiences when planning their service provision in the process of further specialisation.

the need to strike a balance in the training and provision of general physicians versus specialists

There has been ongoing discussion and debate around the world regarding the need to strike a balance in the training and provision of general physicians versus specialists. For example, during the International President's Working Luncheon at the Royal Australasian College of Physicians Congress in May this year, many countries have reflected on the need to attract young doctors into a career in general medicine. Dr. Neil Dewhurst in his Presidential Address during the Roll-Signing Ceremony yesterday also commented on the importance to strike a balance between the number of general internists and specialists. The role of the general physicians is especially crucial in district and community hospitals where a high degree of specialisation would be undesirable and clinicians who can independently manage a wide range of medical problems can more effectively deliver the necessary patient care. In a letter submitted to the Synapse, our College newsletter, back in 1995, I had called for appropriate manpower planning by the College and the Hospital Authority to strike a balance in the number of general physicians and specialists and to develop structured training in advanced internal medicine with defined scope of practice to encourage aspiring young doctors to undertake a career in general medicine.

In Hong Kong, the situation is different from many other countries in that the majority of acute medical services are provided at large regional hospitals with substantial bed complement. Most departments of internal medicine would have sufficient staffing and economies of scale to undertake a high degree of specialisation. In addition, the population is very concentrated in urban areas and there is little geographical barrier limiting access to specialist services. Thus there may seem to be

relatively limited service need for specialists in general medicine.

Furthermore, there is prevalent public perception of superiority of specialists over the general physicians, and many young doctors feel that specialty practice is intellectually more challenging and possibly associated with greater financial reward. All these factors make it difficult to attract trainees of high calibre to devote their career solely to general medical practice. On the other hand, physician specialists are still working within departments of internal medicine and most of them continue to have general ward and emergency call responsibilities.

through a concurrent advanced internal medicine training programme that the trainees could appreciate the specialty practice in the wider context of the functional status of other organ systems and the patient as a whole person

It is with these considerations that our College has designed a dual higher physician training programme such that our trainees would have the breadth of experience from their training in advanced internal medicine and depth in expertise from their specialty training. The basic physician training programme would lay the foundation for subsequent specialty training. However, it is only through a concurrent advanced internal medicine training programme that the trainees could appreciate the specialty practice in the wider context of the functional status of other organ systems and the patient as a whole person. Our physicians would therefore be able to maintain a broad perspective and adopt a holistic approach in their specialty practice. They would be competent in handling the majority of patients with different medical problems without necessarily having to refer them to specialists of the respective organ system.

To strengthen the emphasis on advanced internal medicine training, our College has within the past year decided that it will be mandatory for specialty trainees to undertake dual training with a broad-based programme, either Advanced Internal Medicine or Geriatrics. In addition, physician specialists will be required to have achieved accreditation in either Advanced Internal Medicine or Geriatrics before they can be eligible to serve as trainer in their respective specialty. Advanced Internal Medicine training is further strengthened by the mandatory requirement for completion of the Self Learning Tool, developed jointly with the Hospital Authority, which serves to consolidate their awareness of clinical risks in a wide range of specialty scenarios. The criteria for passing the exit assessment in Advanced Internal Medicine are also under constant review to ensure that a high standard is maintained.

What about the growing trend for sub-subspecialisation? If one considers the need for a critical patient and service volume in order to maintain the proficiency of highly specialised skills, such categories of specialists should be limited in number and concentrated at designated quaternary centres of excellence. Moreover, in line with the common principle that a broad base of medical knowledge would be beneficial to holistic patient care, such highly specialised physicians should maintain their competence in their parent specialty, and preferably in Advanced Internal Medicine as well.

More than a decade ago, our College had submitted a proposal for dual accreditation in Advanced Internal Medicine and a specialty but unfortunately it was not supported by the Academy. I am pleased to inform you that the Academy will be revisiting the issue following a motion by our Senior Advisor during a recent meeting of the Past President Advisory Committee. I would like to emphasise that dual accreditation is not about professional prestige but to foster the breadth and depth in training so that physicians can deliver holistic care to their patients. After a decade, there is a higher likelihood that dual accreditation could be supported by the Academy as more Colleges are embarking on specialisation. I believe that the principle of a broad-based training as the foundation to specialisation and specialty practice is equally relevant to the other Colleges.

Finally, following accreditation in Advanced Internal Medicine, it will be equally important for all of us to remain up to date concerning the key advances in different specialties in order to maintain one's breadth of perspective. Our Annual Scientific Meeting is one such forum to help us keep abreast of the latest developments outside our own specialty areas. More importantly, we should maintain our competence as general physicians through our daily practice. We should exercise gate-keeping function in judicious referral of our patients to other specialists. We should mentor and serve as role models for our trainees in delivering holistic care to our patients.

I would like to close by calling on all of you to support the practice of Advanced Internal Medicine, for the benefit of our patients, for holistic care and to strengthen the art of medicine. May I wish all of you a promising career in your specialty that is strengthened through the practice of Advanced Internal Medicine.

The 17th AJS McFadzean Oration 2012

Making Money from Orphans

Timothy M Cox
Professor of Medicine, Addenbrooke's Hospital, University of Cambridge

It is a special honour to be invited to deliver the 17th McFadzean Oration but the reputation and image of Alexander McFadzean are intimidating. Born in the year that he came to Hong Kong from Glasgow, I never met Professor McFadzean - but I have been privileged to know several of his immensely distinguished representatives on this earth. I have moreover seen what he achieved and the inspiration he provided those who ultimately came to be the Founders of this College and the Academy of Medicine in Hong Kong. By these means, all present are the embodiment of strongly independent institutions in Hong Kong which represent Medicine internationally at the highest professional and academic standard. From today's ceremony it is obvious that the precepts of medicine in Hong Kong are treated with formal respect; and it clear that this respect carries with it the expectation of continued progress through education and discovery. These dual activities are the best equipment we have to shape our future.

It would be invidious to name those many persons who took the torch from McFadzean, with his commitment to clinical medicine and teaching that have been shaped within special context of Hong Kong, but I cannot fail to mention your Founding President - of the Academy and of the College - Professor Sir David Todd. It is an immense pleasure to see him here in the company of many admiring friends. Leonardo da Vinci has been credited with a moving expression: "Unfortunate is the teacher who is not surpassed by his pupils". Although Alexander McFadzean retired to Scotland and died tragically thereafter at the age of only 60, he clearly knew that his teaching had been fortunate.

The subject of my oration is rare, so-called orphan diseases. Many thousands of rare conditions identified in the registries of disease are known to affect tens of millions of people across Europe and America, and occur without 'respect' for any ethnic group. Eighty percent of the >6000 rare diseases so far identified have a genetic basis, and one half of these affect children. In sum, the burden of rare diseases is very large, particularly on families who need to provide for the long-term care and support of young children and infants affected by them; at the same time, this burden has little prospect of relief because of the general lack of incentives for companies (and it must be accepted, investigators) to develop treatments. While we struggle to understand the function of the 22,000 or so human genes, and especially their interactions and regulation, the advice of William Bateson, Mendel's amanuensis,

"Treasure your exceptions!"[1], has never been truer. Now, with the tools of molecular analysis, informative mutants - represented by patients rather than other experimental organisms – provide an unprecedented level of understanding of biochemical pathways, control processes and signalling networks at work in the living person. After years of neglect, rare disorders have rightfully attracted attention; but I would suggest that this productive work should not be confused with the explosion of cynical marketing pitched at the trendy catch-all - Personalized Medicine. Medicine in the proper sense has always been 'patient-centred' and deeply personalized.

The emotive term, 'orphan diseases' emphasises the human cost of neglecting rare conditions and, as the nemesis of civilisations, the cost of failing to provide for them. Orphan diseases are variously defined in different countries **(see Table 1)**. However, with the inception of the Orphan Drug Act in 1983, the US Food & Drug Administration (FDA) a new furnace by which to heat the crucible of drug development for rare diseases with very small markets was ignited. At the initiative of the National Organization of Rare Disorders, this legislation gives pharmaceutical companies no competition over a 7-year period and clinical trial tax incentives for companies with agents in late-phase development. It took years for this initiative to be taken up in Europe but in 2001, largely driven

Table 1 Definitions of Orphan Diseases in different Countries

Population	Number affected individuals	Prevalence per 104
EU	< 250,000	< 5.0 *
United States	< 200,000	< 7.5
Australia	< 2,000	< 1.1
Japan	< 50,000	< 4.0
UK (ultra-orphan)	< 1,000	< 0.2

Prasad, S & James, E (2009)
Brit. J .Med. Procur. Touch briefings, 43-8.




by the European Organization for Rare Disorders (EURORDIS), similar legislation brought: a fee waiver for marketing approval; a 10-year sales monopoly for the first company with approval; central authorization within the European Community through the European Medicines Agency (EMA), and protocol assistance in the design of clinical trials. Since 2007, the FDA and EMA have a common application process for companies seeking licensing and clinical development of orphan drugs. Recent reviews have confirmed the emerging success of these policy initiatives[2, 3].

Largely unknown in the West, has been the signal success over millennia of authentic scientific developments from Chinese materia medica. With many other important matters to hand, the modern history of mainland China not been characterized by drug development. But what has been celebrated, has been the painstaking development of the first-line anti-malarial, artemisinin, first obtained from the Chinese herb, Qinghao, at the China Academy of Chinese Medical Research in Beijing[4]. Professor Tu Youyou, who led the team, won the 2011 Lasker Award in Clinical Medicine at the age of 81 years. With its activity as a febrifuge and initial extraction described in a pharmacopoeia by Ge Hong (284-346 CE) entitled "Handbook of Prescriptions for Emergencies", Professor Tu and her team were able to effect the purification of Qinghaosu from Artemisia annua L., and create a highly effective modern antimalarial drug- a signal and enlightened achievement for human health across the globe[5].

While malaria is, unfortunately, not a rare condition, Hong Kong has provided a recent inspiring example of medicinal development the field of orphan disorders - also based on the extraordinary richness of ancient Chinese medicine. Arsenic in several forms has been a staple of traditional Chinese medicine for more than five millennia but its astounding revival as a cure for acute promyelocytic leukaemia during 1970s, accompanied by the brilliant unravelling of its mode of action by Chinese scientists in Harbin and Shanghai, is a paradigmatic example of translational medicine[6]. Pioneering molecular investigations over the next two decades led by Professor Wang ZY, who had also investigated the action of all-trans retinoic acid differentiation therapy in acute promyelocytic leukaemia, established the role of these specific therapies in this highly malignant leukaemia[7, 8].

Arsenic also reflects the interplay between Asian traditions and Western science as a rare and propitious outcome of medical empiricism. The advent of Thomas Fowler's solution (1% w/v As_2O_3 in potassium bicarbonate) in 1786 encouraged inorganic arsenicals in occidental medicine – followed by the organic arsenicals with antiprotozoal and antimicrobial activity, such as arsphenamine (Salvarsan 606), the first effective treatment for syphilis, marketed in 1910, and with it the concept of chemotherapy. In effect,

the colonial medical schools, returned medicinal arsenic to China. Part of the medicinal history of arsenic has been further documented by Dr Wing-Yan Au of the Department of Medicine at Hong Kong University [9] and will be well-known to you in this college. Repetition ('confirmation') of the effect of intravenous arsenic trioxide supplied from China in the United States led to the filing of a patent for arsenic trioxide by the Cephalon Company of Philadelphia. In 2011 Teva Pharmaceuticals in Israel acquired Cephalon for $6.8 billion, inflating the prices for Trisenox, which had gained a licence as an orphan agent at a cost of $410 for 10mg/d for 4-9 weeks, a full course of Trisenox for leukaemia was of the order of US $50,000 – and thus beyond the reach of most Chinese patients. The local rediscovery of oral liquor arsenicalis, depended on the recollection by Sir David Todd of its use at Queen Mary Hospital in the 1954 [10]. But the agent familiar in Hong Kong as Fowler's solution, long before the inception of its first University in 1911 founded on The Hong Kong College of Medicine for Chinese in 1887 – the colonial heyday. Subsequent development of simple oral arsenic trioxide as a stable preparation by Dr Au and colleagues is inspiring – involving as it did, exploration of the hospital pharmacy archive by R Mack, and Professor C R Kumana's studies to compare the pharmacokinetics and bioavailability of oral and parenteral arsenic trioxide [11]. Thus a small local team, with direct links to the McFadzean era has, in effect, pioneered the first orphan drug from Hong Kong – using the marketplace expertise of the technology transfer office of the University of Hong Kong, a US patent was awarded in 2010 for arsenic trioxide to its commercial arm, Versitech Limited. Dr Au, Professor Todd and their colleagues have promoted use of a convenient formulation to replace intravenous arsenic in Hong Kong and avoid exorbitant charges: at least 150 patients with acute promyelocytic leukaemia have been successfully treated with this first-line therapy utilizing its well-characterized selective molecular actions in this otherwise fatal leukaemia.

Orphan diseases and the development of cognate drugs is a burgeoning political topic; these issues that will not go away, especially with the initiatives of patient advocacy groups – with international Rare Disease Day, on 29th February 2012, a strong precedent has been established. In the West, the horizon is fast changing with the perceived success of orphan drug legislation with a conflation of favourable circumstances. In the 20 years since the inception of the regulations, in excess of 1200 applications have been received, and more than 850 of these have been designated as orphan medicinal products; applications continue to rise [2,3]. At the same time, there has been a major crisis in drug discovery by major pharmaceutical companies. This crisis has inspired courageous investment in informative rare diseases and febrile acquisition of smaller discovery companies by large biopharmaceutical corporations, with obvious synergies in some cases. Smaller biotech companies are awash with numerous scientists and ambitions for discovery and development; but without the expertise and resources to introduce these treatments into the clinical arena, many potentially valuable discoveries tend to remain on an empty platform - orphans unadopted.

More than 95% of rare diseases lack specific treatment – in many cases despite successful exploratory therapeutic studies in cognate animal models [12]. However, perceptions of this bleak field of therapeutics have been radically altered by some unusual examples. Therapeutic development in one very rare disease – ultra-orphan - Gaucher disease is an extreme case [13]. In this respect, the experiment of nature has provided treasure that is tangible as well as intangible. Gaucher disease, an inborn error of metabolism transmitted as an autosomal recessive trait, was first described by Phillipe CE Gaucher in his MD thesis of the University of Paris in 1882. The condition occurs with a birth frequency ≈1 in 100,000 live births or less and is most familiar in its visceral form as principally a macrophage disorder. The large pathognomic cells filling the sinusoids of the spleen in a 34 year-old woman with massive hepatosplenomegaly are so characteristic that they have been immortalized in the eponym. In the 1930s, pathological accumulation of a sphingolipid, β-glucosylceramide was identified in tissues from patients with the disease; and in 1964, the causal deficiency of the acid hydrolase, β-glucocerebrosidase (a lysosomal acid β-glucosidase which maps to human chromosome 1q) was co-discovered in Gaucher disease in 1964 by Des Patrick and Roscoe Brady. Glucosylceramide is a major component of membranes and a precursor of many complex cellular glycosphingolipids. As predicted by the discoverer of the lysosome, Christian de Duve, inherited deficiencies of acid hydrolases in this cellular recycling compartment would cause their cognate macromolecular substrates to accumulate – thus giving rise to the so-called lysosomal storage disorders. De Duve also recognized that, with its ready access to the extracellular fluid phase, functional defects of this organelle should be readily corrected by supplying the complementing factor externally [14]. However, it was to take about 20 years before the simple concept of obtaining and supplying the corrective moiety, acid β-glucosylceramidase, an intrinsic protein of the lysosome showed any beneficial effect in patients with Gaucher disease. Research into Gaucher disease was frustrated by the absence of animal models; and it took many years to understand that therapeutic targeting the pathological macrophage depended on a specific receptor pathway – the mannose receptor. Thus the glycoprotein, glucocerebrosidase (acid β-glucosylceramidase), heroically purified from human placentae, required sequential treatment with three exoglycosidases to reveal mannose residues that mediate endocytosis of the enzyme by macrophages more than an order of magnitude more avidly than unmodified glucocerebrosidase. Enzyme therapy given by intravenous infusion first emerged with a licensed preparation of mannose-terminated β-glucocerebrosidase extracted from human placentae (alglucerase, Ceredase™); latterly the corrective

protein has been obtained from genetically-engineered rodent cells as a recombinant human product, imiglucerase (Cerezyme™). Infusion of mannose-terminal glucocerebrosidase induces marked benefit in patients with Gaucher disease with evident reversal of disease. [15, 16].

Despite its long evolution, Cerezyme, a 'biologic', has been a triumph of modern biotechnology in medicine: as a product of the Genzyme Corporation, licensed in the US under the orphan drug legislation in 1994, it was a global best-seller in ultra-orphan therapeutics. By 2009 more than 6000 patients received the drug worldwide, with about 270 in the United Kingdom. The drug was the single largest source of revenue of the company, was a true 'blockbuster' with annual sales exceeding US$ 1.3 billion [16]. We now know that palliative splenectomy, formerly required life-saving for many patients with florid hypersplenism that is so characteristic of Gaucher disease, is associated with the worsening outcome of the disease in the skeleton. Here, painful osteonecrosis, as well as fragility fractures due to osteoporosis, and lytic lesions due to expansion of the bone marrow space containing pathological macrophages, greatly reduce the quality of life for patients. In most cases, prompt introduction of enzyme therapy shrinks splenomegaly and precludes the need for splenectomy. In worldwide follow-up studies, enzyme therapy has a dramatic effect on the disease, reversing many of the key manifestations in the haematopoietic system, preventing osseous complications and decreasing the size of the often grossly enlarged abdominal viscera, with an improvement in life quality and symptoms of fatigue, as well as the consequences of pancytopenia[13]. In vivo studies confirm the striking targeting of mannose-terminated recombinant human glucocerebrosidase in those organs and sites of disease infiltrated by pathological macrophages [15-17].

An international disease Registry has reported repeatedly on the effectiveness of enzyme therapy in Gaucher disease. However, the individual cost for patients in the order of US$ 200,000-300,000 per year for full licensed doses in adults has attracted a great deal of attention and controversy [16, 18]. By all accounts nonetheless, treatment of the ultra-orphan condition Gaucher disease has been an overwhelming success, not only for the Genzyme company, but as an example of the effects of orphan drug legislation on utility and progress - at least in the occident.

The competitive bubble has naturally excited competition, and by 2010 two other enzyme preparations were in late-phase clinical development: velaglucerase-alfa, also long in clinical development, formerly with Transkaryotic Therapies, now owned by Shire Human Genetic Therapies, is a recombinant glucosylceramidase expressed by gene activation in a human fibrosarcoma cell line and obtained with exposed terminal mannose residues by expressing the recombinant glycoprotein in the presence of a specific inhibitor of glycan processing during biosynthesis, thus leaving an immature lysosomal glycoprotein with exposed mannose residues. A further product, taliglucerase-alfa is a human glucocerebrosidase expressed as a recombinant product in a plant-cell line; in this case the protein has been manipulated for expression in the cell vacuole with terminal mannose residues [16-17, 19]. This agent has been approved by the Food & Drugs Administration of the US but not in Europe.

In 2002 another agent, a novel, orally active iminosugar (N-butyldeoxynojirimycin) as an inhibitor of the first-committed biosynthetic step in the formation of glycosphingolipids that accumulate in Gaucher disease, was given marketing approval by the FDA and EMA. This drug is approved as an orphan second-line treatment for mild to moderate Gaucher disease in adults who are unwilling or unable to take repeated infusions of enzyme protein. Regulatory approval of miglustat stimulated Genzyme itself to develop its own highly selective inhibitors of glucosylceramide biosynthesis. The mode of action resembles that of the statins, where the active agent serves as a potent competitive inhibitor of the first committed biosynthetic step in a metabolic pathway (formation of mevalonate) where failure of degradation leads to disease (cholesterol). Eliglustat tartrate is in late-phase development for Gaucher disease as an orally active agent with a unique chemistry, based on its structural similarity to the ceramide moiety of glucosylceramide [20].

Shortly after de Duve's prediction that the lysosome was accessible to the fluid phase, and that inherited disorders affecting the lysosome might be susceptible to therapy externally, Elizabeth Neufeld later showed that specific cell-surface mannose 6-phosphate receptors could mediate the uptake of glycoproteins and their delivery to lysosomes; this system seems to mirror the intracellular route taken by nascent lysosomal proteins after biosynthesis on the endoplasmic reticulum, but also favours complementation of heritable deficiencies of lysosomal function [21]. Of the 70 or so lysosomal diseases now known, with a net birth frequency of about 1 in 5000 live births, several targeted recombinant enzyme preparations for the treatment of these disorders are approved internationally for marketing. The cost of these ultra-orphan agents for lysosomal disorders other than Gaucher disease for individual patients is extremely high; and despite the orphan drug legislation that promoted their development, their lower effectiveness compared with the biologics available for Gaucher disease, continues to provoke acrid debate in relation to reimbursement from public funds. The generally modest efficacy may be attributable to the lower abundance of the plasma membrane mannose 6-phosphate receptors that mediate the secretion and recapture of lysosomal proteins.

Over-represented in Ashkenazi Jews and with a severe form occurring in a genetic isolate in Norrbottnia, Northern Sweden, Gaucher disease is a panethnic disorder; several patients, mainly children, are treated with imiglucerase in Hong Kong and China. Recent reports show that the

condition is found widely amongst the Chinese and other peoples, including the Thai, in South-East Asia [22-23]. Many of the more disabling alleles of the human glucocerebrosidase gene that occur amongst Chinese lead to a complex and severe phenotype, often associated with neurological manifestations as a result of the impaired lysosome housekeeping of endogenous glycosphingolipids in neural cells, as well as macrophages. Naturally the rarity of the disease is no consolation for those who suffer from it; but the global occurrence of Gaucher disease and the exceptional costs of the treatment and care of the individual suffering from it, provide an illuminating case for debates about the relationship of the individual to their peers, utilitarian attitudes to healthcare provision - and the concept of societal justice. The McFadzean oration is perhaps not the place to explore such complex ethical issues in depth, but the underlying theme I have chosen to discuss adds an egalitarian flavour to the more familiar extremes of clinical medicine, namely the exceptional patient. It was William Bateson, the anglophone amanuensis of the first geneticist, Gregor Mendel, who in my view best captured the sayings of many sages, including in Western medicine, William Harvey and Francis Bacon, about the value of studying rare forms of disease [1].

This brings me to discuss recent findings in a more familiar condition – namely Parkinson's disease, described by the polymath James Parkinson, who worked at The London Hospital, Whitechapel, and published his essay on "The Shaking Palsy" in 1817. Parkinson's disease is no respecter of who you are or where you come from, and there are some notable examples in history, including Deng Xiao Ping, Mao Zedong, Mohammed Ali, Salvador Dali, Generalissimo Francisco Franco, the Reverend Billy Graham, Pope John Paul II - and even Adolf Hitler. In the past, the condition was thought to be of a low prevalence in China, but recent studies show that the prevalence in Beijing, Xian and Shanghai are similar to that in developed countries and that at least 1.7 million people in China over the age of 55 years suffer from this disorder. There has been much interest in the genetic factors in predisposing to Parkinson's disease, and the two largest case control studies published in 2011, supported by the Wellcome Trust in the Parkinson's Disease Genomics Consortium examined more than 12,000 cases and 21,000 control subjects. In the Consortium too there was a combination of an independent replication data set involving more than 3000 cases of Parkinson's disease, and 20,000 controls. This second study rather triumphantly added five risk loci in addition to those eleven previously identified. However there appears to have been a significant oversight. With only one precedent of an isolated case report from Belgium in the 1930s, observant clinicians in Israel and Italy in 1996 anoted an excess occurrence of Parkinson syndrome in patients with otherwise non-neuronopathic Gaucher disease. This coincidence was supported by finding an increased frequency of mutations in the cognate glucocerebrosidase gene in Parkinson's disease in Ashkenazi Jews. By 2009, a multi-centre study of glucocerebrosidase mutations in Parkinson's disease including nearly 5,700 Parkinson patients (780 Ashkenazi Jews) and nearly 4,900 healthy individuals (387 Jews) was reported. Among the Jews, more than 15% of those with Parkinson's disease had a mutant allele of the glucocerebrosidase gene, compared with only 3.4% of healthy Jews. 3.2% of non-Jews with Parkinson's disease had at least one of the mutant alleles, compared with 0.6% of healthy non-Jews – more than a fivefold increase. By definitive sequencing of the human *GBA1* gene in 1,880 non-Ashkenazi Parkinson's patients, mutations were present in 7% (a tenfold increased risk). These findings have been replicated rapidly all over the world – including centres in Asia, where when present in heterozygote form, mutations in the *GBA1* gene responsible for Gaucher disease, confer high susceptibility to Parkinson's disease. Studies in Korea and Taiwan, two separate Chinese populations, report that heterozygous pathogenic variants in *GBA1* confer a high of Parkinson's disease, which is associated with familial clustering and early onset.

The striking relationship between Parkinson's disease and mutations in *GBA1* have stimulated a review of the molecular cell pathology of these disorders – as well as intensive efforts to obtain suitably preserved post-mortem brain samples from patients with haematological and visceral manifestations of Gaucher disease, but without overt neurological features. In a study of patients with extrapyramidal disorders associated with Lewy body deposition, in many instances confocal microscopy showed that apart from the ubiquitin and alpha-synuclein aggregates, the core of the Lewy bodies contained immuno-reactive glucocerebrosidase. Further, in those patients harbouring the N370S mutant of *GBA1*, more than 80% of Lewy bodies showed imunoreactivity against glucocerebrosidase. Whether this can be interpreted as showing that Parkinson's disease is a protein aggregation disorder has yet to be settled; but it is clear that yet again, an ultra-orphan disorder, Gaucher disease, is highly instructive in general application.

Although never directly instructed by Alexander McFadzean, I know that he belonged to that thoughtful group of physicians who, after the clinical consultation, always asked: "So, what did you learn". In Gaucher disease, we have seen a relentless condition which can not only be definitively treated, but dramatically reversed in many of its manifestations – a triumph of the orphan drug legislation and associated initiatives. We see also that competition is an essential feature of human activity and therapeutic development – even though the orphan drug legislation offers the promise of success by providing anti-competitive incentives. We learn also that exceptional drug costs challenge global health systems in their organisation and their provision in society. The concept of societal justice is particularly challenged by the occurrence of disabling and very rare disorders with a strong genetic basis which just will not go away – for as recessive disorders, even the worst

excesses of eugenic practice by the Nazis failed to target heterozygote carriers. The 1000 human genome project shows that, on average, we each harbour about 100 mutations which cause disease when present on both parental alleles.

Finally, following William Bateson's dictum, exceptions clarify general molecular pathophysiology as the counterpart of the normal workings of the human body and its constituent elements. Gaucher disease continues to repay careful investment and can be considered like the Rosetta stone of antiquity (published on 27th March, 196 BCE). The black granite of this stone carries a text inscribed in a hierarchy of languages. Ancient Egyptian hieroglyphs, were inscribed in the top register: the most elevated writing and the exalted language of the Gods. Rare diseases are exceptions to be treasured and, like hieroglyphics, to be patiently translated and understood; for unlike the demotic of common disorders, their study often provides universal insights - and, occasionally, immense worldly wealth.

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ANNUAL SCIENTIFIC MEETING (13-14 October 2012)

The 2012 meeting featured "Medical emergencies' as its main theme. Held at the Hong Kong Academy of Medicine Building, this attracted a packed lecture hall of College trainees and fellows. Local experts provided updates in the management of cardiac, gastrointestinal and respiratory emergencies. The Gerald Choa Memorial Lecture by Professor Kar Neng Lai led the audience to contemplate on medical education and the challenges of teaching humanity.

On the second day of the meeting, winners of the College's most prestigious prizes for scientific research presented their work. The Sir David Todd Lecture Medal was presented to Professor Man Fung Yuen for his influential research on hepatitis B infection. Fellows selected for the Best Thesis Award and the Distinguished Research Paper Award for Young Investigators 2012 also presented their papers.

The ASM 2012

PROF CC SZETO WITH PROF R YU

PROF YL KWONG (CENTRE), CHAIRMAN OF THE ASM ORGANIZING COMMITTEE

DR PATRICK LI, DR WS WONG AND PROF PHILIP LI

PROF KUMANA AND DR MATTHEW TONG

Symposium on Cardiological Emergencies

CHAIRED BY PROF CM YU

DR NS MOK

PROF BRYAN YAN

PROF BERNARD CHEUNG

Symposium on Gastrointestinal Emergencies

CHAIRED BY DR ST LAI

PROF WK LEUNG

DR SC NG

PROF VINCENT WONG

Symposium on Respiratory Emergencies

CHAIRED BY DR THOMAS MOK

PROF KENNETH TSANG

DR CM CHU

DR WM CHAN

25TH ANNUAL GENERAL MEETING, 14TH CONGREGATION AND ANNUAL COLLEGE DINNER

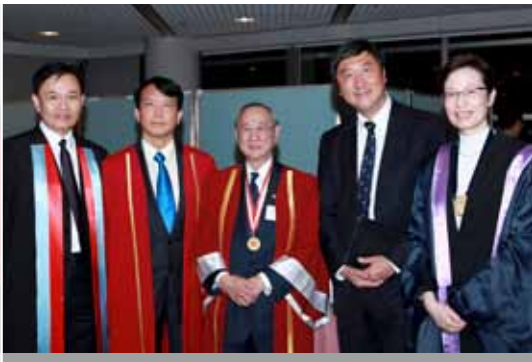
At the AGM held on the 13 October 2012, Dr Patrick Li delivered the Presidential report which summarized the work and achievements of the college during the past year. He paid tribute to members of the various subcommittees for their continuing contributions rendered to the College. The official ceremony proceeded with the conferral of Fellowships and Memberships in the presence of a dignified platform party. This year, Honorary Fellowship was conferred to Dr Neil G Dewhurst. Dr Dewhurst is currently the President of the Royal College of Physicians of Edinburgh. His citation was delivered by Professor Matthew Ng, Vice President of the Hong Kong College of Physicians.

The Annual College Dinner was well attended by new and old Fellows alike, together with their family members. Professor Timothy Cox from the University of Cambridge delivered the AJS McFadzean Oration entitled "Making money from orphans". The President presented all the College awards for best academic achievements in the year 2011-2012.

THE FELLOWSHIP CONFERRAL CEREMONY AND ANNUAL COLLEGE DINNER 2012

PRESIDENT WITH THE OFFICIATING PLATFORM PARTY

(L TO R): PROF L CHAN, PROF R YU, PROF KN LAI, DR CH LEONG, DR CS LI



(L TO R): DR PY LEUNG, PROF P LI, PROF R YU, PROF J SUNG, DR J KHOO

BACK (L TO R): PROF KN LAI, PROF TIM COX, MRS E DEWHURST, PROF J SUNG, PROF R YU, PROF V CHAN, DR D SIU AND DR WM KO
FRONT (L TO R): DR DEWHURST, PROF SIR D TODD, PROF TK CHAN AND MRS S COX

(L TO R): DR PY LEUNG, DR WM KO, PROF F CHAN

PROF J SUNG, PROF L CHAN, DR WM KO AND PROF P LI SHARING A LAUGH TOGETHER

DR WM KO WITH HKCP COUNCIL MEMBERS AND OTHER DISTINGUISHED GUESTS

NEW FELLOWS WITH DR WM KO AND DR PATRICK LI

BACK (L TO R): PROF KN LAI, PROF L CHAN, PROF P LI, PROF R YU, PROF TK CHAN, PROF SIR D TODD AND DR L YAM
FRONT (L TO R): PROF AND MRS YW KAN

THE GERALD CHOA MEMORIAL LECTURE 2012

Medical Education: Can we introduce humanity without a textbook or syllabus?

Professor Kar Neng Lai
Past President, HKCP

THE AJS MCFADZEAN ORATION 2012

Making money from orphans

Professor Timothy Cox
University of Cambridge

SIR DAVID TODD LECTURE

Research on hepatitis B infection – A revelatory course

Professor Man Fung Yuen
Department of Medicine,
Queen Mary Hospital,
The University of Hong Kong

DISTINGUISHED RESEARCH PAPER AWARD FOR YOUNG INVESTIGATORS 2012

The following doctors received the awards at the Annual Scientific Meeting.

ENTECAVIR MONOTHERAPY IS EFFECTIVE IN VIRAL SUPPRESSION AFTER LIVER TRANSPLANTATION FOR HEPATITIS B

James Yan Yue Fung Department of Medicine, Queen Mary Hospital

Fung J, Cheung C, Chan SC, Yuen MF, Chok KS, Sharr W, Dai WC, Chan AC, Cheung TT, Tsang S, Lam B, Lai CL, Lo CM. Gastroenterology. 2011 Oct;141(4):1212-9

SIGNIFICANCE OF HBV DNA LEVELS AT 12 WEEKS OF TELBIVUDINE TREATMENT AND THE 3 YEARS TREATMENT OUTCOME

Walter Wai Kay Seto Department of Medicine, Queen Mary Hospital

Seto WK, Lai CL, Fung J, Wong DK, Yuen JC, Hung IF, Yuen MF. J Hepatol. 2011 Sep;55(3):522-8.

RISK OF INTRACEREBRAL HEMORRHAGE IN PATIENTS WITH CEREBRAL MICROBLEEDS UNDERGOING ENDOVASCULAR INTERVENTION

Yannie Oi Yan Soo Department of Medicine & Therapeutics, Prince of Wales Hospital

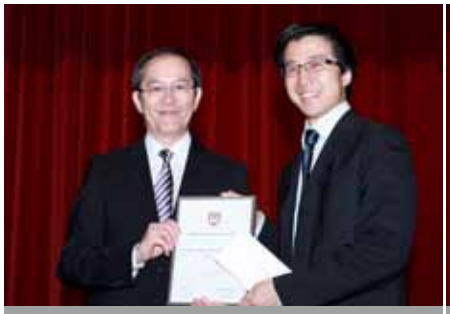
Soo OY, Siu DY, Abrigo J, Yu S, Ng N, Ahuja AT, Wong LK, Leung TW. Stroke. 2012 Jun;43(6):1532-6.

PREVALENCE OF NON-ALCOHOLIC FATTY LIVER DISEASE AND ADVANCED FIBROSIS IN HONG KONG CHINESE: A POPULATION STUDY USING PROTON-MAGNETIC RESONANCE SPECTROSCOPY AND TRANSIENT ELASTOGRAPHY

Wai Sun Wong Department of Medicine & Therapeutics, Prince of Wales Hospital

Wong WS, Chu WC, Wong GL, Chan RS, Chim AM, Ong A, Yeung DK, Yiu KK, Chu SH, Woo J, Chan FK, Chan HL. Gut. 2012 Mar;61(3):409-15.

The winner for the best oral presentation was Wong Wai Sun.



DR WALTER WAI KAY SETO

PROF WAI SUN WONG

DR YANNIE OI YAN SOO

YOUNG INVESTIGATOR RESEARCH GRANT 2012

The following doctors received a research grant from the HKCP to complete their respective projects as named. Applications for 2013 will be advertised in March 2013. Selection will be decided by a scientific panel headed by Professor KS Wong.

HONG KONG SPINOCEREBELLAR ATAXIAS REGISTRY

Anne Yin Yan Chan
Department of Medicine & Therapeutics, Prince of Wales Hospital

THE ROLE OF INSULIN GROWTH FACTOR (IGF) LEVEL ON CACHEXIA AND TREATMENT OUTCOME IN ADVANCED PANCREATIC CANCER PATIENTS

Joanne Wing Yan Chiu
Department of Medicine, Queen Mary Hospital

WAVELET ANALYSIS OF INTRACRANIAL HIGH-FREQUENCY OSCILLATIONS OF CHINESE REFRACTORY EPILEPSY PATIENTS

Ho Wan Leung
Department of Medicine & Therapeutics, Prince of Wales Hospital

HLA-DP AND IL28B GENETIC VARIANTS AND THEIR ASSOCIATION WITH HEPATITIS B SURFACE ANTIGEN (HBSAG) SEROCLEARANCE IN CHRONIC HEPATITIS B

Walter Wai Kay Seto
Department of Medicine, Queen Mary Hospital

CIRCULATING INSULIN-LIKE GROWTH FACTORS AND RELATED PROTEINS, IN ADDITION TO TUMOR EXPRESSION OF INSULIN-LIKE GROWTH FACTOR RECEPTOR-1, DURING NEOADJUVANT CHEMOTHERAPY FOR BREAST CANCER

Hilda Hiu Yan Wong
Department of Medicine, Queen Mary Hospital


DR ANNE YIN YAN CHAN


DR JOANNE WING YAN CHIU


DR HO WAN LEUNG

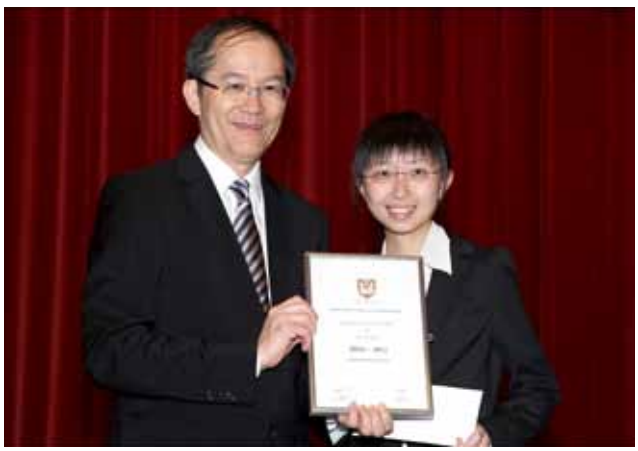

DR WALTER WAI KAY SETO

THE HKCP GOLD MEDAL FOR ADVANCED INTERNAL MEDICINE EXIT ASSESSMENT 2011-2012

Kam Wing Woo
Caritas Medical Centre

THE HKCP GOLD MEDAL FOR MRCP PACES EXAMINATION 2011-2012

Jessica Ka Yan Poon
Ruttonjee Hospital



The HKCP Council 2012-2013

President	*Dr Li Chung Ki Patrick*
Vice-Presidents	*Prof Matthew Ng* *Dr Yam Yin Chun Loretta*
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Co-opted Council Members	*Prof Chan Ka Leung* *Dr Tsoi Tak Hong*
Founding President	*Prof Sir David Todd*
Past President	*Prof Lai Kar Neng*
Senior Advisor	*Prof Yu Yue Hong Richard*

The Council appointed the following Chairmen of various Committees:

National and International Liaison Committee
Prof Matthew Ng

Education and Accreditation Committee
Dr Loretta Yam

Professional and General Affairs Committee
Dr CP Wong

Scientific Committee
Prof YL Kwong

Membership Committee
Dr CS Li

Examination Committee
Prof CS Lau

Administration and Finance Committee
Dr TF Tse

Working Group in Traditional Chinese Medicine
Dr TF Tse

Research Committee
Prof Lawrence Wong

Synapse
Dr Carolyn Kng

SIR DAVID TODD LECTURE

Research on Hepatitis B Infection – A Revelatory Course

Man Fung Yuen
Department of Medicine, Queen Mary Hospital, The University of Hong Kong

Synopsis

In spite of well established universal vaccination for hepatitis B infection in over 170 countries, it is estimated that 400 million people are still infected with chronic hepatitis B (CHB) globally. Of these, 25 – 40% persons will die of cirrhosis and/or hepatocellular carcinoma (HCC).[1, 2] The disease morbidity and mortality are especially high in Asia and Africa where CHB carrier rate is higher than 8%.

Through research that spans over two decades, we have made substantial contributions to the knowledge and clinical management of hepatitis B disease, resulting in much improved clinical outcomes of patients. This synopsis highlights our research findings in elucidating the risk factors for disease progression and increasing our understanding of HBsAg-negative hepatitis B.

A. Natural history of chronic hepatitis B – Risk factors for disease progression

We have identified new risk factors that predict disease progression These factors can be classified into 3 main categories - viral factors, longitudinal serologic profile, and the host response to the infection.

Viral factors These include the virus genotype,[3-9] naturally occurring viral mutations at the enhancer region (nt1653), precore (nt1896), core promoter (nt1753/ 1762/ 1764) regions,[9-11] and also S genome (pre-S deletion).[12, 13] Since these viral factors may have different and confounding effects on disease progression, studies have been performed to elucidate the deleterious effect of each factor. Furthermore, we have demonstrated the synergistic risk escalation when these independent risk factors co-exist.[8] In addition to these mutations, the replication capacity of the virus as reflected by the HBV DNA level has been shown to be the single most important risk factor on the development of cirrhosis and/or HCC.[14] We have demonstrated that HBV DNA levels should preferably be less than 2000 IU/ mL in order to achieve risk reduction for the development of cirrhosis and/ or HCC.[15-19]

Longitudinal serologic profile The serologic profile that evolves during the long disease course has two landmarks. The first serologic landmark is the seroconversion from being hepatitis B e antigen (HBeAg) positive to antibody-to-HBeAg (anti-HBe) status. We have definitively demonstrated that achieving HBeAg seroconversion alone does not confer disease remission.[20, 21]

Host response We have proposed a new cut-off value for alanine aminotransferase (ALT) level, which has been confirmed by other investigators. We showed that patients with ALT over half of the conventional upper limit of normal are associated with a significant risk for the development of cirrhosis-related complications.[14, 21] We also showed that the presence of cirrhosis is itself an important risk factor of disease progression.14 Also, this can now be measured without the risk of a liver biopsy by measuring the transient elasticity of the liver.[22-25] We have shown that this new measurement can now be used to predict the outcome of CHB disease.[25]

B. HBsAg-negative hepatitis B disease

While most research focuses on classical, i.e. HBsAg-positive chronic hepatitis B disease, our team is one of the pioneering research teams to study a mysterious disease entity of hepatitis B disease, the HBsAg-negative hepatitis B. Occult hepatitis B (OHB) and CHB with HBsAg seroclearance are the two main, but different categories of HBsAg-negative hepatitis B disease. The former group of patients has no prior history of CHB infection and they are usually younger. The diagnosis is made by detecting HBV DNA in the serum. The latter group of patients has a definite history of CHB and subsequently loses the HBsAg during follow-up. These patients usually have undetectable HBV

DNA in the serum although the HBV DNA is always detectable in the liver.

Occult hepatitis B

Studies of OHB are difficult to perform because of the requirement of highly sensitive assays to detect the extremely low HBV DNA levels (often below 20 copies/mL) in the sera of the patients. Another difficulty in studying this disease is the requirement for reproducibility of the assays, i.e. consistently detectable low HBV DNA levels, to avoid false positive results. It is therefore not surprising to note that there is scarcity of studies on the exact prevalence of this disease entity. Although it is generally believed that regions with high prevalence of CHB should have a higher prevalence of OHB, large population studies are lacking to confirm this postulation.

According to several cohort studies conducted in different countries, the prevalence of OHB is usually less than 1%.[26] We have conducted the first large population study in Hong Kong, where the prevalence rate for CHB is 8-10%, to document the exact prevalence of OHB in this highly endemic region of CHB. This study recruited a large retrospective cohort (n=3,044) and even larger prospective cohort (n=9,990) of blood donors.[27] The prevalence rates of OHB were 0.13% and 0.11% respectively. This study did not find any difference in identifying OHB by two different surveillance strategies, i.e. screening by nucleic acid testing (NAT) followed by HBsAg or by HBsAg followed by NAT. The latter however has the obvious advantage of a lower screening cost. The HBV DNA levels of the OHB persons (n=11) were confirmed to be extremely low with a range from 0.6 to 14 IU/mL. They had normal liver biochemistry and basically normal liver histology indicating minimal inflammation and fibrosis. Of the six subjects with adequate liver biopsy samples for virological study, 4 had demonstrable HBV DNA and none had covalently closed circular (ccc) DNA (the viral replication template) in the liver. Because of these favorable clinical parameters, these persons should have a very good long-term prognosis.

There is another aspect of OHB which is of obvious importance, i.e. whether HBV can be transmitted if the blood products of these subjects are given to recipients. This is an important health care issue if blood donation centres do not routinely adopt NAT for all blood donations. To answer this important question, we have conducted a separate study of HBV transmissibility using animal and human models.[28] It has been shown that sera from OHB donors are able to infect the liver of chimeric mice whose livers are re-populated with human hepatocytes. Hepatitis B core antigen and cccDNA are detectable by immunostaining and PCR assays respectively in the human hepatocytes inside the chimeric mice. For the human study, by meticulous donor-recipient pair tracing, revelation of viral sequence and establishment of virus transmission from donors to recipients through phylogenetic analysis, we found that there was a 2.2% chance of transmitting HBV to recipients who received the blood products of donors with OHB. The rate of transmission is however very low in OHB donors who were positive for antibody against HBsAg (anti-HBs).

Chronic hepatitis B with HBsAg seroclearance

This second entity of HBsAg-negative hepatitis B is an uncommon event with an occurrence of less than 1% annually in CHB patients. It is generally taken as the most favourable outcome for CHB patients in term of the viral activity and clinical progress. However, the serological, virological, histological and clinical aspects are less known owing to the scarcity of studies. We have conducted studies to address several questions which are of great interests for this group of patients.

Can one predict HBsAg seroclearance?

Factors predicting HBsAg seroclearance can be divided into two categories, namely, the host and the viral factors. For the host factors, increasing age is associated with a higher chance of HBsAg seroclearance. The median age of HBsAg seroclearance is 50 years.[29] In addition, patients who have HBeAg seroconversion at an earlier age have a higher chance of HBsAg seroclearance subsequently.[30] There is no data to suggest gender predilection for HBsAg seroclearance. Western populations seem to have a higher chance of HBsAg seroclearance compared with Asian populations. It is most likely due to the early childhood acquisition of CHB infection in the latter group in contrast to the adolescent acquisition of CHB infection in the former. There are preliminary data from our centre suggesting that host genomic constitution, in particular, the HLA DR, may have an important role on HBsAg seroclearance in CHB patients (data on file).

For the viral factors, we have shown that among the Asian population, HBV genotype B is associated with a higher chance of HBsAg seroclearance compared with HBV genotype C.[30] In addition, we have now shown that HBsAg level < 200 IU/mL is higher predictive for subsequent HBsAg seroclearance within 3 years of follow-up.[31] A log reduction of 0.5 log IU/mL per year is also predictive for HBsAg seroclearance.

Although HBsAg seroclearance is observed in patients receiving interferon-alpha and nucleotide analogue therapy,[32, 33] no case control studies with control groups not receiving treatment have been performed.

Are patients with HBsAg seroclearance really negative for HBsAg?

Documentation of HBsAg positivity is usually performed by the standard enzyme linked immunosorbent assay (ELISA) to detect the HBsAg in patient serum. These assays detect only one epitope, i.e. the common determinant 'a'. An innovative chemiluminescent enzyme immunoassay (CLEIA), called linearized HBsAg assay has been developed recently. In addition to targeting the 'a' determinant, it also targets the epitope embedded inside the lipid bilayer of the viral envelope.[34] It is therefore 10 times more sensitive than the existing HBsAg assays. Another new assay measuring the hepatitis core-related antigen (HBcrAg) circulating in the patient serum has also been developed.[35] We are the first group to find out that, 26% and 21% of patients who were tested HBsAg negative by conventional assays were positive for linearized HBsAg and HBcrAg respectively.[34] Over 40% of sera from patients with HBsAg seroclearance were positive for either linearized HBsAg or HBcrAg. These results suggest that transcription of viral proteins still exists even when serum HBsAg is undetectable by the conventional HBsAg assays.

The liver and viral replication activity in patients with HBsAg seroclearance

The liver biochemistry is normal including the albumin, bilirubin and ALT levels in around 70- 80% of patients with HBsAg seroclearance.[29] Abnormal liver biochemistry observed in these patients is usually due to other causes of chronic liver diseases e.g. fatty liver. Data of the liver necroinflammation and fibrosis/ cirrhosis are lacking because of the reluctance of performing liver biopsies in patients with normal liver biochemistry. Our study revealed that nearly all patients with HBsAg seroclearance had no significant necroinflammation and also with only minimal fibrosis.[29, 30] Our study also showed that there were decreasing

proportions of patients with detectable serum HBV DNA levels within 1 year, between 5 to 10 years and more than10 years after HBsAg seroclearance, being 13.4%, 6.1% and 3.7% respectively. This suggests that viral replication decreases with time after HBsAg seroclearance. In spite of this, intrahepatic HBV DNA and cccDNA were still detectable in 100% and 80% of patients respectively. 79-100% of the intrahepatic HBV DNA were in the form of cccDNA indicating that the virus is quiescent and not replicating. This is further supported by the lack of mRNA expression in the liver.[29] Nevertheless, the presence of cccDNA in the liver signifies the potential for reactivation of viral replication. This is best exemplified by hepatitis B reactivation in HBsAg-negative and HBV marker-positive (e.g. antibody to hepatitis B core antigen) subjects receiving potent immune modulating agents e.g. anti-CD 20 monoclonal antibody (rituximab) in haematological conditions.

Impact of HBsAg seroclearance on the risk of developing hepatocellular carcinoma

Owing to the favourable biochemical, serological, histological and virological parameters mentioned above, it is expected that patients with HBsAg seroclearance should have a low chance of development of hepatocellular carcinoma (HCC). According to our two studies, patients with HBsAg seroclearance have a lower risk of development of hepatocellular carcinoma (HCC) only when the HBsAg seroclearance occurs before the establishment of cirrhosis30 and before the age of 50 years.[29] It is likely that when the disease has progressed to the late stage and/or has been there for over 50 years, development of HCC may continue even with the very low level of viral replication after HBsAg seroclearance.

To understand better the role of HBV in HBsAg-negative patients with HCC, a detailed documentation of the presence of HBV inside the liver of patients with HCC without identifiable causes including chronic hepatitis C and alcoholic liver disease is necessary. Two studies from our centre examining patients with cryptogenic HCC demonstrated that majority of these HCCs (73 – 100%) are related to HBV.[36, 37] cccDNA was detectable in 26% of liver tissues of these patients with detectable HBV DNA inside the liver. In addition, pre-genomic RNAs were detectable in 52% of HCC patients with detectable HBV DNA.[37]

Conclusions

Through research efforts by our team and others, the knowledge on the natural history of chronic hepatitis B infection has undergone a revelatory advancement over the recent two decades. We are now able to identify patients who are at high risk of developing cirrhosis or hepatocellular carcinoma for intervention with long-term viral suppressive therapy, which has proven efficacy in reducing the risk of complications.

Occult hepatitis B and chronic hepatitis B with HBsAg seroclearance are the two major entities constituting HBsAg-negative hepatitis B disease. Our research findings have narrowed the knowledge gaps in these hitherto mysterious entities. We now know that HBsAg seroclearance have better clinical outcome if the HBsAg seroclearance occurs early in the course of the disease, although practically all of them are still habouring HBV inside the liver with quiescent viral activity. Indeed both conditions are still at risk of HBV reactivation, with potential for liver decompensation and mortality, such as in the setting of immunosuppression.

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Best Thesis Award

Gold Award Winner

Assessment of liver fibrosis with transient elastography (TE) and non-invasive clinical formulae in patients treated with methotrexate for psoriatic arthropathy and rheumatiod arthritis

By **Wai Chung Chan**
Department of Medicine & Geriatrics, Kwong Wah Hospital

Background

Methotrexate is often used in the treatment of psoriasis and rheumatoid arthritis. There are ongoing controversies in the prevalence and severity of its hepatotoxic side-effects. Risk factors which have been identified to be associated with methotrexate hepatotoxicity include psoriasis, type 2 diabetes, obesity, excessive alcohol use and total cumulative dose. Liver biopsy remains the 'gold standard' for liver fibrosis evaluation. However, it is invasive and many patients do not accept its inherent risks. On the other hand, there are evolving evidence on the use of non-invasive methods in assessing methotrexate hepatotoxicity.

Methods

In this prospective study, patients with chronic viral hepatitis B and C were excluded. Transient elastography (FibroScan) and three clinical prediction formulae: AST to platelet ratio index (APRI), AST to ALT ratio (AST/ALT) and FIB4 were used to evaluate liver fibrosis in psoriasis and rheumatoid arthritis patients on methotrexate. The effect of different risk factors in causing significant liver fibrosis (defined as FibroScan ≥7.9kPa) and determinants for liver stiffness were studied. Histological features of liver biopsy in patients with significant liver fibrosis were reported.

Results

A total of 128 subjects were included in this study. The prevalence of significant fibrosis by FibroScan ≥7.9kPa, APRI >0.5, AST/ALT >0.8 and FIB4 >1.3 were 4.9%, 7.8%, 85.2% and 35.2% respectively. Clinical prediction formulae did not show a positive correlation with FibroScan value. In evaluating the six patients with FibroScan ≥7.9kPa, all patients had cumulative dose of methotrexate more than 1500mg, but on multivariate analysis, hypertension was the only independent risk factor associated with significant liver fibrosis (OR = 11.02, 95%CI: 1.130-107.503, p = 0.039). Determinants of liver stiffness were age (β = 0.058; p < 0.001), psoriasis (β = 0.947; p = 0.022), methotrexate regime adjusted due to abnormal liver biochemistries (β = 1.188, p = 0.003) and insulin resistance (β = 0.815; p = 0.043). Liver biopsy was performed on 2 patients with FibroScan ≥7.9kPa with findings of mild peri-portal fibrosis and mild steatosis (Roenigk grade IIIa).

Conclusion

Significant liver fibrosis and cirrhosis are not common in methotrexate-treated patients. The cumulative dose of methotrexate is probably not associated with significant fibrosis but this requires further elucidation with a larger sample. Patients with risk factors should be monitored closely with non-invasive methods with good performance in diagnosing significant fibrosis.

Best Thesis Award

Silver Award Winner

Health related quality of life in Chinese patients with systemic sclerosis (SSC) relationship with the extent of skin involvement and severity index

By **Pak To Chan**
Department of Medicine & Geriatrics, Tuen Mun Hospital

Background

Systemic sclerosis (SSc) is a multisystemic connective tissue disease with unknown etiology. It is characterized by a triad of immune dysfunction, vasculopathy and fibrosis. It causes skin hardening and tightening, digital ulceration, arthritis, joint contractures and dysfunction of essential internal organs, leading to morbidity and mortality. It also causes significant disability and impairment in the health related quality of life (HRQoL).

Hypothesis: Patients with more severe skin involvement and organ manifestations have poorer HRQoL and greater disability.

Objectives

To evaluate the HRQoL and disability scores of a group of Chinese SSc patients and compare these parameters with age-matched healthy controls. The relationship between HRQoL and disability and the extent of skin disease and severity of organ manifestations were also studied.

Methods

Systemic sclerosis (SSc) patients who fulfilled the 1980 American College of Rheumatology (ACR) preliminary criteria for the classification of systemic sclerosis or the LeRoy and Medsger classification criteria, who were hospitalized or attended the outpatient rheumatology clinics of Tuen Mun and Pok Oi Hospitals were recruited for a cross sectional study of the HRQoL and disability. The clinical records of these patients were reviewed and a full physical examination was performed for each participant to assess for the extent of skin and organ involvement. The severity of skin involvement was assessed by the Modified Rodnan skin score (mRSS). Disease severity was assessed by the Medsger severity scale. Two sets of questionnaires, the Medical Outcomes Study Short Form 36 (SF-36) and the Health Assessment Questionnaire Disability Index (HAQ-DI) were used to assess the HRQoL and disability of patients, respectively. An equal number of age and gender matched healthy controls were also recruited for the completion of these two questionnaires. The SF36 scores and HAQ-DI score of SSc patients and healthy controls were compared by the Students' t-test. Linear regression analyses were used to find out the factors associated with poorer HRQoL and greater disability in SSc patients.

Results

Seventy-eight Chinese SSc patients were recruited. The mean age was 50±12 years and the mean disease duration was 7.8±6.5 years. Sixty-eight (87%) of them were women. Sixty-three (81%) patient had limited cutaneous systemic sclerosis (lcSSc) while the remaining had diffuse cutaneous systemic sclerosis (dcSSc). The median mRSS of the patients was 8 (IQR 4-14). Raynaud's phenomenon was the most common clinical features (97%) followed by sclerodactyly (82%), arthritis (68%) and joint contracture (47%). Patients with SSc had a significantly higher HAQ-DI score than the healthy controls (0.69±0.69 vs 0.04±0.18; $p < 0.001$) and lower SF36 score in all domains ($p < 0.05$ in all). Linear regression analyses revealed that the mRSS was inversely correlated to the physical component score (PCS) and mental component score (MCS) of the SF36 (Beta = -0.39; $p = 0.001$) and (Beta = 0.27; $p = 0.031$) and positively correlated to the HAQ-DI score (Beta = 0.51; $p < 0.001$), after adjustment for the age, sex and duration of the disease. The SF36 and HAQ-DI scores also significantly correlated with the Medsger severity scale in the general, peripheral vascular, skin and tendon/joint domains of the Medsger severity scale.

Conclusion

Chinese SSc patients had poorer HRQoL and greater disability than matched healthy subjects. Patients with higher skin scores, more peripheral vascular system damage and tendon / joint contracture were associated with greater disability and poorer health related quality of life.

Best Thesis Award

Bronze Award Winner

Correlation of genotypic and phenotypic features of Haemoglobin H disease in Hong Kong Chinese

By **Hoi Yan Chan**
Department of Medicine, Pamela Youde Nethersole Eastern Hospital

Background

The alpha thalassaemias are one of the most common inherited disorders of haemoglobin (Hb) synthesis in Southeast Asia. Deletions or mutations of three of the four alpha globin genes located on chromosome 16 give rise to Hb H disease, an intermediate clinical form of alpha thalassaemia. Hb H disease shows a wide spectrum of clinical phenotypes, ranging from asymptomatic to transfusion-dependent anaemia. The clinical variability is contributed by the wide genetic heterogeneity as well as environmental factors. We studied the clinical features and the α-globin gene abnormalities in Hong Kong Chinese patients with Hb H disease, and the genotype-phenotype correlation as well as the effects of iron overload in these patients.

Methods

The clinicopathological features including haematological parameters, serum ferritin levels, liver function and endocrine functions of adult Chinese patients with Hb H disease were studied in a single hospital in Hong Kong from January 1995 to July 2010. Genotypic analysis of the two pair of α-globin genes was performed. Cardiac/liver magnetic resonance imaging studies were performed in a subset of patients enrolled in oral chelator therapy study.

Results

Hb H disease in 82 of the 95 patients (86%) was due to the deletional type and 13 patients (14%) had the non-deletional type. All patients with deletional type were compound heterozygous for two α-globin gene deletion of the Southeast Asia type and a single α-globin gene deletion on the other chromosome: -3.7-kb rightward deletion (64%), and 4.2-kb leftward deletion (21%). In patients with non-deletional type of Hb H disease, the Quong Sze variant accounted for the majority, followed by hemoglobin Constant Spring. The present study demonstrated correlation between genotype and phenotype of Hb H disease in Chinese. Patients with the non-deletional type of Hb H disease had more severe anaemia, larger spleen size, higher serum ferritin levels and were more likely to require transfusions. Raised serum ferritin and the presence of hepatic iron overload were common among adult Hb H patients who were not transfusion dependent. Age, transfusion and genotype are among the factors that affect iron overload in these patients.

Conclusions

In Chinese patients with HbH disease, most have relatively mild anaemia and do not require regular transfusion. The majority of patients have the deletional type of disease and they have milder clinical phenotype than those with the non-deletional type.

Pass Rates for MRCP (Part I) examination

	Sitting	Pass
Sep 02	100	33 (33%)
Jan 03	124	55 (44%)
May 03 (SARS Special)	21	7 (33%)
Sep 03	54	29 (54%)
Jan 04	93	39 (42%)
Sep 04	29	16 (55%)
Jan 05	96	68 (70.8%)
Sep 05	24	15 (62.5%)
Jan 06	95	74 (80%)
Sept 06	21	13 (62%)
Jan 07	87	67 (77%)
Sep 07	23	12 (52%)
Jan 08	56	38 (68%)
Sept 08	47	32 (68%)
Jan 09	59	47 (80%)
Sept 09	47	28 (60%)
Jan 10	45	28 (62%)
Sept 10	62	39 (63%)
Jan 11	44	23 (52%)
Sept 11	64	49 (77%)
Jan 12	45	28 (62%)
Sept 12	80	59 (74%)

Pass Rates for the Joint HKCPIE/MRPC(UK) Part II (Written) examination

	Sitting	Pass
2 Jul 2002	53	27 (51%)
13 Nov 2002	50	24 (48%)
13 Aug 2003	110	62 (56%)
10 Dec 2003	54	31 (57%)
28 Jul 2004	65	42 (65%)
8 Dec 2004	46	32 (70%)
13 Apr 2005	32	15 (47%)
27 Jul 2005	76	56 (74%)
7 & 8 Dec 2005	26	16 (62%)
12&13 Apr 2006	29	13 (45%)
26 & 27 Jul 2006	91	68 (75%)
6 & 7 Dec 2006	33	18 (55%)
11 & 12 Apr 2007	34	22 (65%)
25 & 26 Jul 2007	80	70 (88%)
5 & 6 Dec 2007	19	13 (68%)
9 & 10 Apr 2008	21	13 (62%)
30 & 31 Jul 2008	47	36 (77%)
3 & 4 Dec 2008	17	10 (59%)
8 & 9 Apr 2009	32	25 (78%)
29 & 30 Jul 2009	50	43 (86%)
25 & 26 Nov 2009	12	7 (58%)
7 & 8 April 2010	41	34 (83%)
28 & 29 July 2010	25	19 (76%)
24 and 25 Nov 2010	8	2 (25%)
6 and 7 April 2011	45	35 (78%)
23 and 24 Nov 2011	32	25 (78%)
28 and 29 March 2012	55	43 (78%)
12 and 13 December 2012	57	44 (77%)

Pass Rates for the Joint HKCPIE/MRCP (UK) Part II PACES examination

October 2001	36/72 = 50%	June 2007	44/74 = 59%
February 2002	34/74 = 46%	October 2007	36/55 = 65%
October 2002	29/72 = 40%	March 2008	36/74 = 49%
February 2003	30/69 = 43%	October 2008	29/65 = 45%
October 2003	27/59 = 46%	February 2009	39/75 = 52%
March 2004	39/64 = 61%	October 2009	24/72 = 33%
October 2004	26/69 = 38%	March 2010	33/75 = 44%
March 2005	35/75 = 47%	October 2010	40/74 = 54%
October 2005	28/75 = 37%	February 2011	23/66 = 35%
March 2006	36/75 = 48%	October 2011	34/70 = 49%
October 2006	16/73 = 22%	February 2012	32/74 = 43%
March 2007	44/74 = 59%	October 2012	32/74 = 43%

Pass List for the Joint HKCPIE/MRCP (UK) Part II PACES Examination October 2012

Chan Chi Him Simon
Chau Ling Kit Nicholas
Chow Lai Man Vivian
Ho Kim Wah*
Kong Hoi Yan Harriet
Lam Kwan Wai
Leung Chun Yu
Leung Yuen Ling Erica
Ma Siu Pang
Natarajan Deepa
Shum Kin Cheong
Tam Ho Chi
Tang Sze Wan
Tse Chit Ming
Wong Hing Wing
Yeung Wing Yin
Chan Shung Kay Samuel
Cheng Hiu Man
Chua Nicholas Yul Chye*
Hui Ting Hin Adrian
Kwong Ying Yui
Lee Ying Cheong
Leung Sai Chau
Li Cho Shan
Mak Oi Ki Ankie
Sakhrani Navin
Soekojo Cinnie Yentia*
Tan Nee Hooi*
Teo Kay Cheong
Wai Tsz Yan
Yan Wing Shan Betty
Yip Pui Lun

* Not HKCP registered trainees

Single specialty trainer in Dermatology and Venereology

At its 249th Meeting of 28 June 2012, the Council confirmed that, apart from AIM and Geriatric Medicine, single specialty Trainers in Dermatology and Venereology is accepted in our College. Hence, Fellows in Dermatology and Venereology are entitled to apply for Trainer status even if they have not undergone concurrent training in AIM or Geriatric Medicine.

Statistics on No. of Trainees in all Specialties

Updated in October 2012

SPECIALTY	TRAINEES TOTAL (PP/DH/HA/ OTHERS)	TRAINEES						
		HONG KONG EAST CLUSTER			HONG KONG WEST CLUSTER			
		PYNEH	RH	TWEH	FYKH	GH	QMH	TWH
		YEAR			YEAR			
CARDIOLOGY	24	1 0 2 3 4 *5*	1 1 2 3—I 4 *2*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *5*	1—I 3 2 3—II 4 *6*	1 0 2 3 4 *0*
CLINICAL PHARMACOLOGY & THERAPEUTICS	1	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 1 2—I 3 4 *1*	1 0 2 3 4 *0*
CRITICAL CARE MEDICINE	7	1 1 2—I 3 4 *4*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1—I 2 2—I 3 4 *3*	1 0 2 3 4 *0*
DERMATOLOGY & VENEREOLOGY	13	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 1 2—I 3 4 *0*	1 0 2 3 4 *0*
ENDOCRINOLOGY, DIABETES & METABOLISM	15	1 2 2—I 3—I 4 *2*	1 2 2—I 3 4—I *1*	1 0 2 3 4 *3*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 2 2—I 3—I 4 *5*	1 0 2 3 4 *0*
GASTROENTEROLOGY & HEPATOLOGY	23	1—I 1 2 3 4 *4*	1 1 2—I 3 4 *1*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1—I 3 2 3—I 4—I *7*	1 0 2 3 4 *1*
GERIATRIC MEDICINE	15	1 0 2 3 4 *6*	1 0 2 3 4 *11*	1—I 1 2 3 4 *3*	1 0 2 3 4 *3*	1 0 2 3 4 *1*	1 2 2—II 3 4 *2*	1 0 2 3 4 *2*
HAEM/HAEM ONCOLOGY	6	1 1 2—I 3 4 *2*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1—I 3 2 3—II 4 *5*	1 0 2 3 4 *0*
IMMUNOLOGY & ALLERGY	0	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*
INFECTIOUS DISEASE	3	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *1*	1 0 2 3 4 *0*
INTERNAL MEDICINE	197	1—III 13 2—III 3—II 4—V *32*	1 6 2—IV 3—I 4—I *13*	1 0 2 3 4 *10*	1 0 2 3 4 *2*	1 2 2 3—I 4—I *5*	1—IV 27 2—X 3—VIII 4—V *46*	1 0 2 3 4 *9*
MEDICAL ONCOLOGY	4	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *2*	1 0 2 3 4 *0*
NEPHROLOGY	18	1—I 2 2 3 4—I *4*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 3 2—II 3—I 4 *5*	1 0 2 3 4 *3*
NEUROLOGY	17	1—I 1 2 3 4 *5*	1 1 2—I 3 4 *2*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1—I 3 2—I 3—I 4 *6*	1 0 2 3 4 *0*
PALLIATIVE MEDICINE	6	1 0 2 3 4 *0*	1—I 1 2 3 4 *2*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 1 2—I 3 4 *1*	1 1 2 3 4—I *0*	1 0 2 3 4 *0*
REHABILITATION	3	1 0 2 3 4 *0*	1 0 2 3 4 *2*	1—I 1 2 3 4 *4*	1 0 2 3 4 *1*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *5*
RESPIRATORY MEDICINE	15	1 1 2 3—I 4 *4*	1 0 2 3 4 *5*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *7*	1 1 2—I 3 4 *6*	1 0 2 3 4 *0*
RHEUMATOLOGY	12	1 1 2 3 4—I *2*	1 0 2 3 4 *1*	1 0 2 3 4 *1*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 1 2—I 3 4 *3*	1 0 2 3 4 *1*

		TRAINEES										
		KOWLOON CENTRAL CLUSTR		KOWLOON EAST CLUSTER			KOWLOON WEST CLUSTER					
SPECIALTY	TRAINEES TOTAL (PP/ DH/HA/ OTHERS)	KH	QEH	HOHH	TKOH	UCH	CMC	KWH	OLMH	PMH	WTSH	YCH
		YEAR		YEAR			YEAR					
CARDIOLOGY	24	1 0 2 3 4 ***0***	1—I 3 2—I 3 4—I ***9***	1 0 2 3 4 ***0***	1 2 2—I 3 4—I ***1***	1 2 2—I 3—I 4 ***4***	1 1 2 3—I 4 ***1***	1 2 2 3 4—II ***4***	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***7***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***
CLINICAL PHARMACOLOGY & THERAPEUTICS	1	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
CRITICAL CARE MEDICINE	7	1 0 2 3 4 ***0***	1 3 2—I 3—I 4—I ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 0 2 3 4 ***4***	1 0 2 3 4 ***3***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
DERMATOLOGY & VENEREOLOGY	13	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
ENDOCRINOLOGY, DIABETES & METABOLISM	15	1 0 2 3 4 ***0***	1 0 2 3 4 ***6***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***	1 0 2 3 4 ***3***	1 1 2—I 3 4 ***2***	1 0 2 3 4 ***3***	1 0 2 3 4 ***1***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***	1 2 2 3—II 4 ***1***
GASTROENTEROLOGY & HEPATOLOGY	23	1 0 2 3 4 ***0***	1—II 2 2 3 4 ***6***	1 0 2 3 4 ***0***	1 2 2—I 3 4—I ***1***	1 1 2—I 3 4 ***2***	1 1 2 3 4—I ***3***	1 2 2 3—I 4—I ***4***	1 0 2 3 4 ***1***	1 2 2 3—II 4 ***4***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***5***
GERIATRIC MEDICINE	15	1 1 2 3—I 4 ***4***	1 0 2 3 4 ***1***	1 0 2 3 4 ***4***	1 0 2 3 4 ***2***	1 1 2—I 3 4 ***8***	1 2 2 3—I 4—I ***7***	1 1 2 3—I 4 ***9***	1 0 2 3 4 ***1***	1 0 2 3 4 ***14***	1 0 2 3 4 ***4***	1—I 1 2 3 4 ***5***
HAEM/HAEM ONCOLOGY	6	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1—I 1 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
IMMUNOLOGY & ALLERGY	0	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
INFECTIOUS DISEASE	3	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 1 2 3—I 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
INTERNAL MEDICINE	197	1 1 2 3 4—I ***2***	1-V 22 2-VI 3-III 4-VIII ***49***	1 2 2—I 3 4—I ***4***	1 5 2—III 3 4—II ***14***	1—I 8 2—III 3—III 4—I ***33***	1 ***10*** 2—I 3—VI 4—III ***26***	1 ***7*** 2—I 3—I 4—V ***35***	1 0 2 3 4 ***3***	1—IV ***13*** 2—III 3—III 4—III ***45***	1 0 2 3 4 ***3***	1—I 8 2—I 3—V 4—I ***19***
MEDICAL ONCOLOGY	4	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
NEPHROLOGY	18	1 0 2 3 4 ***0***	1—I 3 2—I 3 4—I ***7***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***4***	1 1 2 3—I 4 ***2***	1 1 2—I 3 4 ***7***	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***6***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***
NEUROLOGY	17	1 0 2 3 4 ***0***	1—I 3 2—I 3—I 4 ***6***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***2***	1 0 2 3 4 ***2***	1 0 2 3 4 ***1***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***	1 2 2—I 3—I 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
PALLIATIVE MEDICINE	6	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 1 2 3—I 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
REHABILITATION	3	1 2 2 3—I 4—I ***7***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***
RESPIRATORY MEDICINE	15	1 0 2 3 4 ***5***	1 0 2 3 4 ***6***	1 1 2 3 4—I ***3***	1 0 2 3 4 ***2***	1 1 2 3—I 4 ***3***	1 1 2 3—I 4 ***2***	1 0 2 3 4 ***2***	1 0 2 3 4 ***0***	1—I 2 2—I 3 4 ***4***	1 0 2 3 4 ***2***	1 2 2 3—I 4—I ***1***
RHEUMATOLOGY	12	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 1 2 3—I 4 ***2***	1 0 2 3 4 ***1***	1 2 2 3—I 4—I ***2***	1 0 2 3 4 ***0***	1—I 2 2—I 3 4 ***2***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***1***

SPECIALTY	TRAINEES TOTAL (PP/DH/HA/ OTHERS)	TRAINEES						
		NEW TERRITORIES EAST CLUSTER					NEW TERRITORIES WEST CLUSTER	
		AHNH	NDH	PWH	SH	TPH	POH	TMH
		YEAR					YEAR	
CARDIOLOGY	24	1 1 2 3—I 4 ***2***	1 1 2 3 4—I ***3***	1—I 2 2 3 4—I ***8***	1 1 2 3—I 4 ***0***	1 1 2—I 3 4 ***0***	1 0 2 3 4 ***1***	1 3 2 3—II 4—I ***2***
CLINICAL PHARMACOLOGY & THERAPEUTICS	1	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
CRITICAL CARE MEDICINE	7	1 0 2 3 4 ***3***	1 0 2 3 4 ***3***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***3***
DERMATOLOGY & VENEREOLOGY	13	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
ENDOCRINOLOGY, DIABETES & METABOLISM	15	1 0 2 3 4 ***1***	1 1 2 3 4—I ***2***	1—I 3 2 3—II 4 ***8***	1 1 2—I 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***
GASTROENTEROLOGY & HEPATOLOGY	23	1 1 2 3—I 4 ***1***	1 0 2 3 4 ***2***	1 3 2—I 3—II 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2 3 4—I ***4***	1 2 2—I 3—I 4 ***3***
GERIATRIC MEDICINE	15	1 0 2 3 4 ***1***	1 0 2 3 4 ***1***	1 0 2 3 4 ***5***	1 1 2—I 3 4 ***6***	1 1 2—I 3 4 ***3***	1 1 2 3 4—I ***1***	1 3 2—II 3—I 4 ***7***
HAEM/HAEM ONCOLOGY	6	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2 3 4—I ***4***
IMMUNOLOGY & ALLERGY	0	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
INFECTIOUS DISEASE	3	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***2***
INTERNAL MEDICINE	197	1—I 5 2—I 3—III 4 ***10***	1 6 2—I 3 4—V ***13***	1—III 15 2—IV 3—IV 4—IV ***47***	1 5 2—II 3—I 4—II ***5***	1 6 2—III 3—I 4—II ***5***	1 3 2 3 4—III ***10***	1—IV 23 2—V 3—VI 4–VIII ***36***
MEDICAL ONCOLOGY	4	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 3 2—II 3—I 4 ***15***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
NEPHROLOGY	18	1—I 1 2 3 4 ***2***	1 0 2 3 4 ***1***	1—I 3 2—II 3 4 ***5***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***0***	1 0 2 3 4 ***0***	1—I 2 2 3 4—I ***5***
NEUROLOGY	17	1 1 2 3—I 4 ***1***	1 2 2—I 3 4—I ***1***	1 1 2—I 3 4 ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1—I 1 2 3 4 ***3***
PALLIATIVE MEDICINE	6	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***
REHABILITATION	3	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***
RESPIRATORY MEDICINE	15	1 0 2 3 4 ***3***	1 0 2 3 4 ***4***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***1***	1—I 4 2 3—I 4—II ***2***
RHEUMATOLOGY	12	1 0 2 3 4 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***2***	1 0 2 3 4 ***0***	1—I 2 2 3—I 4 ***3***

** Total No. of trainees is shown in upper right corner of each hospital*
*** No. of trainers is shown in italics & bold in lower right corner of each hospital*

SPECIALTY	TRAINEES TOTAL (PP/DH/HA/OTHERS)	TRAINEES	
		DH	
DERMATOLOGY & VENEREOLOGY	13	1—II 2—IV 3—II 4—IV	12 ***10***
INTERNAL MEDICINE	197	1 2—I 3—II 4	3 ***0***
INFECTIOUS DISEASE	3	1 2 3 4	0 ***2***
RESPIRATORY MEDICINE	15	1 2 3—I 4	1 ***10***

** Total No. of trainees is shown in upper right corner of each hospital*
*** No. of trainers is shown in italics & bold in lower right corner of each hospital*

Statistics on No. of Fellows in all Specialties
Updated in October 2012

		FELLOWS									
		HONG KONG EAST CLUSTER				HONG KONG WEST CLUSTER					HONG KONG EAST + WEST CLUSTER
SPECIALTY	FELLOWS TOTAL (PP/DH/HA/OTHERS)	PYNEH	RH	TWEH	Subtotal	FYKH	GH	QMH	TWH	Subtotal	
CARDIOLOGY	231	9	7	0	**16**	0	6	16	0	**22**	**38**
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	0	**0**	0	0	2	0	**2**	**2**
CRITICAL CARE MEDICINE	81	11	0	0	**11**	0	0	10	0	**10**	**21**
DERMATOLOGY & VENEREOLOGY	92	0	0	0	**0**	0	0	1	0	**1**	**1**
ENDOCRINOLOGY, DIABETES & METABOLISM	92	3	1	3	**7**	0	0	8	0	**8**	**15**
GASTROENTEROLOGY & HEPATOLOGY	163	6	2	1	**9**	0	0	10	1	**11**	**20**
GERIATRIC MEDICINE	176	6	12	3	**21**	3	2	4	2	**11**	**32**
HAEM/HAEM ONCOLOGY	53	3	0	0	**3**	0	0	9	0	**9**	**12**
IMMUNOLOGY & ALLERGY	6	0	0	0	**0**	0	0	0	0	**0**	**0**
INFECTIOUS DISEASE	39	3	0	0	**3**	0	0	2	0	**2**	**5**
INTERNAL MEDICINE	1164	49	22	13	**84**	4	12	88	11	**115**	**199**
MEDICAL ONCOLOGY	41	0	0	0	**0**	0	0	9	0	**9**	**9**
NEPHROLOGY	119	5	1	0	**6**	0	0	7	3	**10**	**16**
NEUROLOGY	101	6	3	0	**9**	0	0	9	2	**11**	**20**
PALLIATIVE MEDICINE	22	0	2	0	**2**	0	2	1	0	**3**	**5**
REHABILITATION	51	0	3	4	**7**	2	0	1	5	**8**	**15**
RESPIRATORY MEDICINE	172	11	6	2	**19**	0	8	10	0	**18**	**37**
RHEUMATOLOGY	68	4	2	1	**7**	1	0	7	1	**9**	**16**

		FELLOWS														
		KOWLOON CENTRAL CLUSTER			KOWLOON EAST CLUSTER				KOWLOON WEST CLUSTER							KOWLOON CENTRAL + EAST + WEST CLUSTER
SPECIALTY	FELLOWS TOTAL (PP/DH/HA/OTHERS)	KH	QEH	Subtotal	HOHH	TKOH	UCH	Subtotal	CMC	KWH	OLMH	PMH	WTSH	YCH	Subtotal	
CARDIOLOGY	231	0	16	**16**	0	1	7	**8**	2	6	1	10	0	5	**24**	**48**
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	**0**	0	0	0	**0**	0	0	0	0	0	0	**0**	**0**
CRITICAL CARE MEDICINE	81	0	6	**6**	0	4	6	**10**	4	6	0	7	0	0	**17**	**33**
DERMATOLOGY & VENEREOLOGY	92	0	0	**0**	0	0	0	**0**	0	0	0	0	0	0	**0**	**0**
ENDOCRINOLOGY, DIABETES & METABOLISM	92	0	8	**8**	0	4	5	**9**	2	4	2	6	0	1	**15**	**32**
GASTROENTEROLOGY & HEPATOLOGY	163	0	9	**9**	0	2	3	**5**	4	8	1	6	0	8	**27**	**41**
GERIATRIC MEDICINE	176	5	4	**9**	4	3	13	**20**	7	10	2	15	7	5	**46**	**75**
HAEM/HAEM ONCOLOGY	53	0	9	**9**	0	2	2	**4**	0	0	0	4	0	0	**4**	**17**
IMMUNOLOGY & ALLERGY	6	0	0	**0**	0	0	0	**0**	0	0	0	0	0	0	**0**	**0**
INFECTIOUS DISEASE	39	0	5	**5**	0	0	1	**1**	0	0	0	5	0	1	**6**	**12**
INTERNAL MEDICINE	1164	6	72	**78**	7	24	52	**83**	31	47	8	63	7	25	**181**	**342**
MEDICAL ONCOLOGY	41	0	2	**2**	0	0	1	**1**	0	0	0	1	0	0	**1**	**4**
NEPHROLOGY	119	0	8	**8**	2	2	4	**8**	2	9	0	9	0	2	**22**	**38**
NEUROLOGY	101	1	9	**10**	0	2	5	**7**	2	5	1	2	1	1	**12**	**29**
PALLIATIVE MEDICINE	22	0	0	**0**	4	0	2	**6**	4	0	1	0	1	0	**6**	**12**
REHABILITATION	51	9	0	**9**	2	0	2	**4**	1	1	0	2	5	0	**9**	**22**
RESPIRATORY MEDICINE	172	6	7	**13**	6	4	6	**16**	5	6	0	5	3	1	**20**	**49**
RHEUMATOLOGY	68	1	5	**6**	0	2	3	**5**	3	2	0	3	0	2	**10**	**21**

		FELLOWS									
		NEW TERRITORIES EAST CLUSTER						NEW TERRITORIES WEST CLUSTER			NEW TERRITORIES EAST + WEST CLUSTER
SPECIALTY	FELLOWS TOTAL (PP/DH/HA/OTHERS)	AHNH	NDH	PWH	SH	TPH	Subtotal	POH	TMH	Subtotal	
CARDIOLOGY	231	3	5	14	0	0	**22**	2	9	**11**	**33**
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	5	0	0	**5**	0	0	**0**	**5**
CRITICAL CARE MEDICINE	81	3	3	1	0	0	**7**	0	6	**6**	**13**
DERMATOLOGY & VENEREOLOGY	92	0	0	3	0	0	**3**	0	0	**0**	**3**
ENDOCRINOLOGY, DIABETES & METABOLISM	92	1	4	14	0	0	**19**	0	3	**3**	**22**
GASTROENTEROLOGY & HEPATOLOGY	163	1	5	7	0	0	**13**	5	9	**14**	**27**
GERIATRIC MEDICINE	176	1	2	7	8	3	**21**	1	10	**11**	**32**
HAEM/HAEM ONCOLOGY	53	0	0	5	0	0	**5**	0	6	**6**	**11**
IMMUNOLOGY & ALLERGY	6	0	0	0	0	0	**0**	0	0	**0**	**0**
INFECTIOUS DISEASE	39	2	1	3	0	0	**6**	0	3	**3**	**9**
INTERNAL MEDICINE	1164	19	27	76	9	6	**137**	14	60	**74**	**211**
MEDICAL ONCOLOGY	41	0	0	15	0	0	**15**	0	0	**0**	**15**
NEPHROLOGY	119	6	1	7	0	0	**14**	2	7	9	**23**
NEUROLOGY	101	1	1	9	2	0	**13**	1	5	**6**	**19**
PALLIATIVE MEDICINE	22	0	0	0	2	0	**2**	0	1	**1**	**3**
REHABILITATION	51	0	1	2	1	1	**5**	0	3	**3**	**8**
RESPIRATORY MEDICINE	172	3	8	8	0	3	**22**	3	6	**9**	**31**
RHEUMATOLOGY	68	3	0	5	0	2	**10**	2	3	**5**	**15**

Dr Neil G Dewhurst

BSc Hons, MD, PRCP Edin, FRCP Lond, FRCP Glas.

John MacKay

It is not often that one meets someone so enthusiastic about their job.

Dr. Neil Dewhurst is exceptional in that he is enthusiastic about all his jobs, and there are many.

He is a consultant Cardiologist and General Physician; Honorary Senior Lecturer at the University of Dundee; President of the Royal College of Physicians of Edinburgh; Chair of the Federation of the Royal Colleges of Physicians of United Kingdom; Chair of the Academy of Medical Royal Colleges Assessment Committee.

He was in Hong Kong in October on his first visit since 2008, to preside over the Signing of the Role for new Fellows of the Edinburgh College, and on the following evening to receive an Honorary Fellowship from the Hong Kong College of Physicians.

He was educated at Hutton Grammar School in Lancashire, whose history dates back to 1517 and the time of Henry VIII; and has nothing to do with that famous cricketer Len Hutton of Yorkshire. At school he enjoyed Biology and Human biology so concentrated on science subjects, which gained him a place to study medicine at Edinburgh University. There was no tradition in the family of Medicine as a career.

His University career was marked by three Medals including the one in medicine, and a First Class Honours BSc in Bacteriology. His postgraduate house job in Surgery was spent at Leith, the port city of Edinburgh and a rough neighbourhood. His Medical House Job was at the Royal infirmary wards 29 and 30 under Dr Alec Proudfoot who also ran the toxicology service. At this time paracetamol self poisoning was a problem. In the Poisons Unit they pioneered treatment with N-acetylcysteine, NAC, intravenously. Prior to this NAC had only been used as a mucolytic oral spray or cough linctus.

He was appointed to a Clinical Lectureship in the University Department of Medicine under Professor Jim Robson and Dr Sandy Muir, studying ventricular remodelling after myocardial infarction, the subject of his MD thesis. For his research on this subject he was awarded the Stelios Nicolaides prize by the RCPE and the Carey Coombs prize from the University of Bristol, (named after one of the pioneers in Cardiology),

On completion of his Lectureship Dr Dewhurst left the University but continued to work at the Edinburgh Royal Infirmary as a National Health Service doctor, rapidly rising to the position of Senior Registrar.

His first consultant appointment was as Consultant Cardiologist at South Devon Healthcare Trust. When he arrived he was the only cardiologist and it took some time to develop and finance a new and fully comprehensive invasive cardiological service. He is proud of the fact that he never had any infection of pacemakers, which he attributes to his insistence on using the ultra sterile facilities of an orthopaedic surgery theatre.

In 1997 Dr. Dewhurst moved to the Perth Royal Infirmary as consultant Cardiologist, and built up the service there as he had done in South Devon. He was appointed an Honorary Senior Lecturer, and in 2006 Perth Royal Infirmary was formally adopted as a teaching hospital for Dundee University.

He was the Registrar and Council Member of the RCPE in 2000-03. Subsequently, he was appointed Medical Director of the MRCP (UK) Examinations. Since this time he has developed strong links with the Hong Kong College of Physicians. He has been involved with the setting up of MRCP (UK) exams in Abu Dhabi, India and Pakistan. It is not a simple process, Fellows working in the country concerned have to be recruited and helped to prepare for the examination; a Mock Exam is held to test the systems; finally the MRCP (UK) is launched. So far all has gone according to plan; in Hong Kong particularly Dr Dewhurst finds the organisation of the exam is superb.

He is fascinated by the process of the MRCP examination, and counts its continuing development as one of the highlights of his career. He has written research papers on the methodology and reliability of the exam and gender and ethnic variation. Joint meetings with the counterparts, Specialist Board examiners in USA, have shown very similar thinking about the conduct of specialist assessment. The Part 1 written examination lasts six hours, and in order to make Part 2 more reliable it has been extended to nine hours over two days. The PACES Examination has replaced the traditional short and long cases; here again great attention to detail requires that the cases are relevant to the country concerned and that the idiomatic use of the English language is respected.

The Royal Colleges in UK are acutely aware of the strains on the health service as a whole and how this has a negative affect on the training of doctors. The Royal College of Physicians of London has recently published 'Hospitals on the Edge? The time for Action', which has brought home to doctors, the public and Government the seriousness of the situation. The ideal of hospitals, and General Practice, running a 24 hour a day 7 days a week service cannot be achieved simply by better organisation and use of resources, but will require more staff, more hospital beds and more money. This situation would seem to be similar to that of the Hospital Authority in Hong Kong.

The positions Dr Dewhurst holds as PRCPE and Chair of the Federation of Royal Colleges of Physicians of UK mean that he is deeply involved in assessing the needs, and advising government about the training of junior doctors.

DR NEIL DEWHURST, PRESIDENT OF THE RCP(EDINBURGH) RECEIVED THE HKCP PLATE AS A TOKEN OF LONGSTANDING FRIENDSHIP BETWEEN THE TWO COLLEGES

Since Dr Dewhurst began his first term as the President in March 2010, the RCPE has published on Health Priorities for Scotland and a Charter for Medical Training in the UK. The RCPE has also forged closer links internationally with Jordan, India and Myanmar and in addition, has extended web-streaming of symposia to Africa and Asia. Of the Fellows of the Edinburgh College half are from overseas, a quarter from England and the rest from Scotland so the College has to keep an international perspective.

In recognition of his many international contributions Dr Dewhurst has received honorary fellowships from the Colleges of Physicians of North America, Australasia, Ceylon, Ireland and Malaysia, and now Hong Kong.

Dr. Dewhurst will be finishing his clinical responsibilities in the spring of next year. He will continue as President of the Edinburgh College of Physicians for another year after his present term expires in March 2013, and will continue to contribute to training and examination matters thereafter.

He is looking forward to having more time to indulge his leisure interests, shared with his wife Elspeth, of music and promoting the College's adopted charity "Music in Hospitals". His other interests include gardening, and fishing for salmon and trout in the rivers and lochs of Scotland.

It was clear to those watching the Fellowship ceremony at the Run Run Shaw Hall of the Academy of Medicine Building that Dr. Dewhurst enjoyed the occasion and the opportunity to meet the newly appointed Fellows. There is no doubt that he will continue to take pleasure in these happy occasions and be remembered with affection in return.

(L TO R): PROF M NG, PROF P LI, MRS E DEWHURST, DR N DEWHURST, DR P LI