HONG KONG COLLEGE OF PHYSICIANS
香港內科醫學院

Sapientia et Humanitas

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Sapientia et Humanitas

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The Gerald Choa Memorial Lecture 2012

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

Professor KN Lai
Immediate Past President, HKCP

I thank the College for asking me to deliver this Lecture especially under the name of Professor Gerald Choa. I was probably one of the last medical graduates who had worked with Professor Choa and was most honoured to have served him at the Chinese University of Hong Kong at the inception of the medical school. Professor Choa was a unique person in the history of University of Hong Kong. First, he was the only graduate who neither took the entrance examination nor completed an exit examination before his graduation. He was so ill during his matriculation in 1930s that he could not sit for the examination. He was recommended by the Principal of the Wah Yan College to enrol in Medicine at the University of Hong Kong directly. His graduation examination was disrupted by the Japanese invasion of Hong Kong. Therefore he was the only HKU graduate who completed medical training without an entrance or an exit examination, an exceptional event never to be repeated again. After the war, he became a lecturer at the University of Hong Kong. This picture [Figure 1] with Dr Stephen Chang (left) taken in the early 1950s is from the archives of the Department of Medicine, University of Hong Kong. He became Consultant of Government Medical Unit when he returned from overseas training in the United Kingdom. He then became the Director of Medical and Health Department before being appointed as the Founding Dean of Medical Faculty of Chinese University of Hong Kong. He was also the Vice-President of our College.

Figure 1: PROFESSOR GERALD H. CHOA WITH DR. STEPHEN CHANG CIRCA 1950

I probably knew Professor Choa for 38 years. In those days, you would receive a certificate signed by the Dean stating that you had completed the one-year internship. The acting Dean then was Professor David Todd. At the same time, you applied for a licence to practise. This was my first practising licence and the name of the registrar was Professor Gerald Choa. My first personal encounter approximately 8 years later was when I returned from Australia. I was asked by my mentor Professor John Vallance-Owen to see Professor Choa. I saw Gerald for the first time when he offered me a lectureship. The best thing Gerald promised me was that "you can work at the Chinese University of Hong Kong but you will not be asked to stay at the university quarters". Therefore, I was probably the only person living outside the Chinese University of Hong Kong campus quarter during my years there. I served under him in this College when he was the Vice-President for many years. He was not only an excellent clinician, but also a good educator. In fact I must admit the curriculum at Chinese University of Hong Kong at that time was far more modern than the curriculum at the University of Hong Kong. In fact, when he was the Dean, he introduced the new curriculum and structure of different subspecialties which has become the main departments of medical schools nowadays.

I was asked to talk about medical education because Professor Choa was a great educator. I took the liberty to change the theme to humanity and medicine. The title of my topic is "Medical education: can we introduce humanity without a syllabus or a textbook". Medicine is always branded as a profession built on evidence-based scientific foundation coupled with an art of healing. Nevertheless, a humanistic touch is absolutely essential. The question is how we can introduce to our medical students issues of humanity which are embedded within the core of our medical curriculum and beyond the textbooks.

Normally, there are two ways to teach humanity in a medical school. The American system ensures doctors have an understanding of art, music, anthropology, mythology, archaeology, politics, culture, religion, history and sociology. In the United States or most medical schools in Australia, medicine is a second degree. Hence, there are compulsory core subjects including the above-mentioned topics as course requirements for the first basic degree. In the British system, the medical curriculum is a first degree which spans longer than the American or Australian system. However, the curriculum is so intense that it is difficult to incorporate exposure to the humanities. Recently, the Dean of University of Hong Kong had advocated the importance of humanity in Medicine. He started a Centre for the Humanities and Medicine at University of Hong Kong which conducts talks given by members from other faculties for medical students.

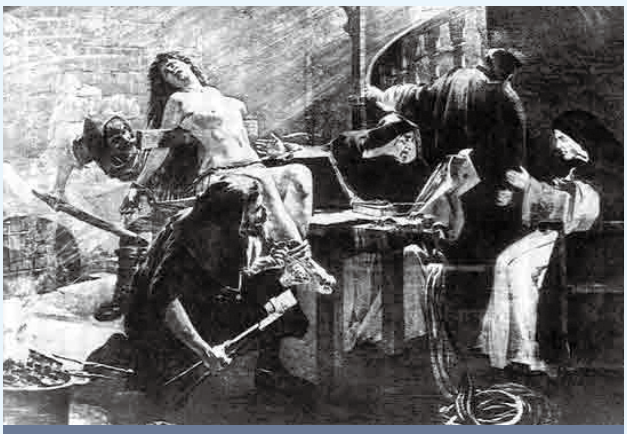

Figure 2: TORTURE OF A WOMAN WITH SUPERNUMERARY NIPPLES ALLEGED TO BE A WITCH BY THE ROMAN CATHOLIC INQUISITION

I always believe that teaching on any subject related to Medicine should be led by clinicians themselves, as non-medical academicians surely do not know Medicine as well. The problem lies in how we can teach or introduce Humanities in an interesting way so that it won't be delivered as a university course such as Fine Art 101, History 101 or Sociology 101. If offered as such a university course, it must be categorized as compulsory core curriculum or most students would not attend if it were voluntary.

In this lecture, I try to give examples how we can find interesting subjects in different aspects of Humanities within the realm of Medicine. I will illustrate how humanities influence Medicine and vice versa. This is proposed to honour Professor Gerald Choa's exceptional vision in medical education.

Firstly, I would like to illustrate how Medicine is affected by cultural perception with interesting examples from history. The first slide [Figure 2] shows a classical picture which may well have been taken straight out of Bailey & Love's surgical textbook. This woman has supernumerary nipples which are not uncommonly found. However, in old days, this was actually treated differently. In the western society, they were called "Devil's Marks and Witches' Marks". Even worse, ladies with such inborn anatomical anomalies were actually labelled as witches and were tortured by the Roman Catholic Inquisition. Every one of these "Devil's Marks" were stabbed with nasty looking knives called the "witch-prickers" conducted by a celibate, "chaste" priest. This was commonly practised until the 17th century. As described by Reverend John Bell (1705), minister of Gladsmuir in Scotland: "The witch marks is sometimes like a blew spot, or a little flat or red spots, like flea biting; sometimes also the flesh is sunk in, and hallow… I myself have seen it in the body of a confessing witch, like a little powder mark, of a blue colour, somewhat hard…". Sometimes, I wonder whether Reverend Bell was describing skin papules and macules. Hence, only 300 years ago, the western church still viewed an inborn anomaly as an evil expression. In China, attitudes towards supernumerary nipples is totally different. From 史記, it reads "文王昌，龍顏虎肩，身長十尺，胸有四乳。晏朝不食，以延四方之士。..... 文王嗣位五十年，即《周書》所謂「文王受命，享國五十年」是也". It was recorded the father of King Wu who established the Chow Dynasty about 12 century B.C. was born with four nipples and commanded respect from his people. In China, it is fine to have supernumerary nipples. In the western world until the early 18th century, you would be tortured

or condemned to death if you have supernumerary nipples.

Let's look at another anthropological item called "Duplex Pupils" [Figure 3]. Ancient Chinese believed that persons with two pupils are born with blessings from the heaven. Heaven bestows upon them talents and even immortality. In fact, this is a malformation of the pupil with adhesion of the sclera leading pupil from O to ∞ shape. The anomaly does not affect the visual image. It is similar to the compound eye in insects. From Chinese historic treatise, at least six persons had "Duplex Pupils". The first was倉頡who invented the Chinese characters in the Neolithic ages. The second was 虞舜 - an ancient Neolithic king. From then onwards, everyone believed person with "Duplex Pupils" was born as a holy person.

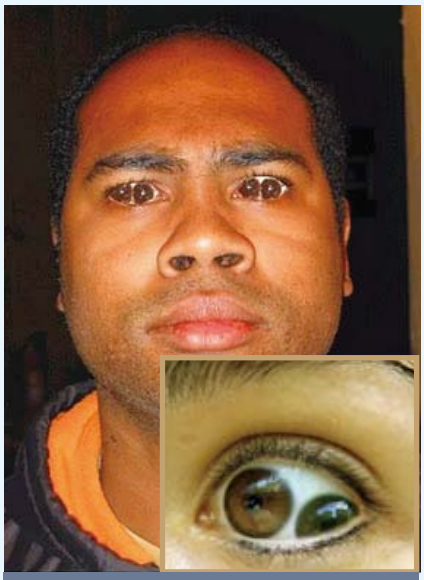

Figure 3: DUPLEX PUPILS

This happened in 項羽 (also born with Duplex Pupils) who overthrew the Qin Dynasty at 221 B.C. 項羽 at the time of his death still believed he had divine power from the heaven. Subsequently he died because of his failure to conquer 劉邦. Another two kings (呂光 and魚俱羅) in the fourth and fifth century A.D. were known to have "Duplex Pupils". The last one in the Chinese history was李煜. 李煜 was a famous poetic king in the kingdom of 南唐 around 950 A.D. He believed he would not lose his kingdom because he had "Duplex Pupils". In fact, he subsequently was captured by the Emperor of the Sung Dynasty and died in captivity. In contrast, "Duplex Pupils" is considered to be a token of "evil eyes" in the western world as stated in McDaniel's paper 1971 (McDaniel WB. Perspective in Biological Medicine 1971; 15: 72-79).

Another interesting person is 劉備 who was a famous king at the time of the Three Kingdoms (220-280 A.D.). From the historical archives,劉備 had many Marfanoid features. He could touch his knees in a standing posture. He had joint hypermobility and lived to almost 70 years. One may wonder 劉備looked like an orangutan or suffered from Marfan syndrome. In fact, there are features of 劉備 that had not been recognized. 劉備 had winged ears < 後漢書 • 呂布傳> 呂布大罵劉備：“大耳兒最叵信”, 身長七尺五寸 (tall), 垂手下膝 (long arm spam) 顧自見其耳 (hypermobility). He could see his ears because he had hypermobility. This condition is now recognized as joint hypermobility syndrome by the British Society of Rheumatology (Rheumatology 2001; 40: 559-562). The cardinal features of this syndrome are blue sclera, atypical ears (also called "winged ears"), abnormal nose, prominent chin, marfanoid habitus and hyperflexion of more than 90° of the joints in the hand. Therefore I believed劉備 had joint hypermobility syndrome.

Now I turn to Medicine and Politics with a few examples how Medicine affects Politics. The first example is Emperor Kangxi, the second emperor of the Qing Dynasty. Kangxi was the third son of his father Shunzhi. He had five brothers and was enthroned at the age of eight not because of his mother was a queen. Kangxi was probably half Manchurian and half Han as his mother was a consort from a military clan originally under the Han Chinese Plain Blue Banner. He was chosen to succeed the throne because he had smallpox at the age of five. He was the only child of Shunzhi who survived smallpox. Two of ten Qing emperors died of smallpox - one was Shunzhi and other one was Tongzhi (son of the Express Dowager Cixi). Due to Kangxi' immunity to smallpox, he was chosen to be successor of his father who died early of smallpox. In the Qing Dynasty, within the palace, they had a temple for Goddess called T'ou-Shen Niang Niang. She was supposed to spread flowers from heaven (天花) leading to smallpox infection. She headed four ministers who spread measles, chickenpox, scarlet fever and pockmarks. Kangxi was very keen in preventing smallpox. He encouraged a smallpox vaccination program called nasal insufflation. The Chinese practised the vaccination at least 200 years before Jenner introduced the smallpox vaccination in England. Mild smallpox cases were selected as donors in order to prevent serious attack. Scabs were ground into powder or mixed with a grain of musk and bound in cotton. Infected material was then packed into a pipe and puffed up the patient's nostril. The blowpipe used during the procedure was made of silver [Figure 4]. Variolated cases were treated as infectious as those who had acquired the disease naturally. Subsequently, this vaccination program was introduced to the West leading to the cowpox vaccination in 1796.

Another political figure in the West was Napoleon. In 1815, Napoleon was imprisoned and exiled to the Island of Saint Helena in the Atlantic Ocean, 1870 km from the west coast of Africa. Within three years his health deteriorated and he died at the age of 52 three years later. As he was in the British custody, naturally, it was believed throughout Europe that Napoleon was murdered. Napoleon's physician, François Carlo Antommarchi, led the autopsy. Antommarchi found the cause of death to be stomach cancer yet he did not sign the official report. The stomach ulcer was 4 cm in

Figure 4: SMALLPOX VACCINATION BY NASAL INSUFFLATION USING PUSTULES

diameter. Subsequently in 1955, from the diaries of Napoleon's valet led Dr. Sten Forshufvud to propose in a 1961 paper in Nature that deliberate arsenic poisoning as the cause of Napoleon's death. A study from Glasgow in 1960 stated the type of arsenic found in Napoleon's hair shafts was the most toxic mineral type leading to the conclusion by the toxicologist, Patrick Kintz, that Napoleon's death was murdered by British through chronic arsenic poisoning. Most intriguingly, researchers in 2008 analysed hair samples of Napoleon's collected at different ages comparing with samples from his family and other contemporaries. All samples had high levels of arsenic, approximately 100 times higher than the current average. According to these researchers, Napoleon's body was already heavily contaminated with arsenic since childhood. The high arsenic concentration in his hair was not caused by intentional poisoning; people were constantly exposed to arsenic from glues and dyes (Scheele's Green) throughout their lives as shown by hairs collected from Napoleon of different ages as well as from other contemporaries. Then, what was the cause of Napoleon's death? Truly, Napoleon died of stomach ulcer. In 2005, Swiss scientists were able to obtain 12 pairs of Napoleon's trousers; four pairs of trousers before his exile and eight in exile. They collated post-mortem information on the weights and waist measurement of patients who died of stomach cancer. They estimated Napoleon had lost 11 to 15 kg in the last 6 months of his life. They concluded that Napoleon died of stomach cancer. This settled the question of whether the British had poisoned Napoleon. Moreover, Napoleon's father and her sister, Pauline, also died of stomach cancer.

Now let us look at another piece of modern history. This is the blood pressure chart of a gentleman in July 1941. The systolic blood pressure fluctuated between 160 to 180 mmHg. This gentleman was Franklin D Roosevelt (FDR) who was serving his third term of the Presidency of the United States of America. Franklin D. Roosevelt was wheelchair-bound at that time following election to an unprecedented third term in 1940. It was during the end of this term that FDR's health began to decline. Exhausted from a summit in Teheran with Winston Churchill and Josef Stalin at the end of 1943, FDR's health deteriorated rapidly after his return. Months passed and the President did not bounce back. He lost weight, his face thinned, and he had shortness of breath. At first, FDR's personal physician, Vice Admiral Ross T. McIntire diagnosed the President's problem as the "flu" and bronchitis. Not satisfied with the diagnosis, FDR's family wanted a second opinion from Dr. Howard G. Bruenn in March 1944. Dr. Bruenn, Chief of Cardiology at the Bethesda Naval Hospital in Maryland, found that FDR was suffering from hypertension, left ventricular failure, and bronchitis. He recommended that FDR be given digitalis, put on a low salt diet, and have bed rest. Unfortunately, there were not many drugs that were effective to treat hypertension in early 1940. FDR's blood pressure again rose from 168/108 mmHg in March 1944 to 260/150 mmHg in January 1945. On the night of February 3, 1945, his blood pressure was 260/150 mmHg. FDR was sleepless, puffing and sitting up all night. He complained of headache and was unable to concentrate. He had a 9 a.m. meeting the next morning with Stalin and Churchill at the Yalta conference [Figure 5]. At this meeting, a reparation commission was set up to help repay Russia from German assets. Everyone realized that the conquest of eastern Europe by the Red Army guaranteed Soviet dominance, but efforts were made to get promises of fair elections. The promises were made but elections were never held. Roosevelt, despite his failing health, seemed to think that he and Stalin would personally iron out any difficulties after the war ended. In fact, he died two months after the meeting. Retrospectively, FDR's judgment might have been affected by his encephalopathy and cardiac failure. When I was an intern in 1975, we began to have a new "wonder" antihypertensive drug, propanolol. Nowadays, propanolol is rarely use as a single antihypertensive agent due to better efficacy of newer antihypertensive medications. The new generation of doctors will be surprised the pharmacologist, Sir James Whyte Black. who discovered propanolol, a beta-blocker and cimetidine, a histamine-2 blocker won the 1988 Nobel Prize in Medicine Looking back, these are major discoveries. One wonders if these antihypertensives were available 30 years earlier, the iron curtain or the cold war might be totally different if FDR's blood pressure was under control with no encephalopathy or cardiac failure.

Let's now consider Medicine and Mythology. All of us are interested in the story of the Amazon Warriors in Greek mythology. They were a nation of all-female warriors from Asia Minor or Ukraine. In Greek mythology, Amazons were fierce warriors with Queen Penthesilea and her sister Hippolyta participated in the Trojan War. Among classical Greek, Amazon was given a popular etymology as from a-mazos, "without breast", connected with an etiological tradition that Amazons had their right breast cut off or burnt out, so they would be able to use a bow more freely and throw spears without the physical limitation and obstruction. This is a picture from the 16th century

Figure 5: THE YALTA CONFERENCE, SOMETIMES CALLED THE CRIMEA CONFERENCE HELD IN 1945. WITH PRESIDENT FRANKLIN D ROOSEVELT .. IN THE CENTRE

showing Amazons capturing the Greeks [Figure 6]. From Greek pottery, the true Amazons carried a shield and a quiver and wore patterned cloth long trousers - not as romantic or sexy as Greek mythology depicted.

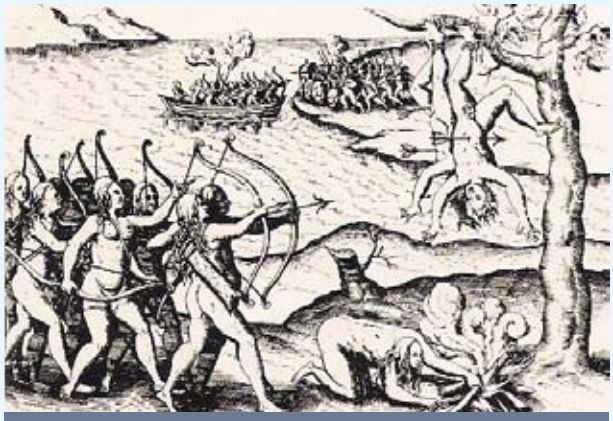

Figure 6: A 16TH CENTURY ENGRAVING DEPICTING THE WAR BETWEEN THE AMAZONS AND THE GREEKS

Next I move onto archeology before finishing my lecture with art and music.

This is a picture I took with my iPhone when I visited the Cairo museum despite photography was officially not permitted [Figure 7]. The picture showed an interesting stature with a man, his wife and his two children. The man was a wealthy Achondroplasia dwarf married to a normal looking wife. The children looked normal. Achondroplasia occurs as a sporadic mutation in approximately 75% of cases (associated with advanced paternal age) or may be inherited as an autosomal dominant genetic disorder (25%). The disorder itself is caused by a change in the DNA for fibroblast growth factor receptor 3 (FGFR3), which causes an abnormality of cartilage formation. The British liked to raise dogs of different physical appearance. In the 19th century, they successfully bred different species of dogs with short legs including of dachshunds, basset hound and bulldog. In 2009, a group led by Heidi Parker, a geneticist at the National Human Genome Research Institute in Maryland reported in Science they had identified the single evolutionary event that created the modern short-legged dog. It was an extra copy of a gene that was acquired by mutation at least 300 years ago, when modern dog breeding began. The extra gene is the reason for the stubby legs of these short-legged breeds. It caused the overproduction of a protein that disrupts their growth during fetal development. Interesting link between achondroplastic human and short-leg dogs lies in the fibroblast growth factor. A whole genome association studies in short-legged dogs revealed a strong association of this trait with a retro-gene coding for fibroblast growth factor 4 while defects in FGFR3 leads to achondroplasia in human.

Figure 7: A STATURE WITH AN ACHONDROPLASIA DWARF, HIS NORMAL WIFE AND HIS TWO NORMAL CHILDREN (PICTURE TAKEN IN THE CAIRO MUSEUM)

Let us talk about music. The first maestro was Ludwig van Beethoven. His cause of death was well known. He suffered from alcoholic cirrhosis, infectious hepatitis, sarcoidosis and Whipple's disease. He was deaf and he also had syphilis. He had lead poisoning because syphilis was treated with lead at that time. The second maestro is more mysterious. He was Wolfgang Mozart. Mozart's medical history included syphilis (treated with mercuric salts), rheumatic fever, renal impairment due to vasculitis and Trichinosis from eating undercooked pork chops. The summary of his illness in the last six months before his death was published in the Journal of the Royal Society of Medicine (1983; 76: 781). A recent study revealed Mozart died within a short time (Annals of Internal Medicine 2009; 151: 274-278). He had fever, skin rash, limb pain, dropsy, gross edema and renal failure. Lately, historians re-examined the archives in Vienna at the time of 1791 A.D. They discovered there were 5000 people (males and females) aged 40 or less died of similar clinical features in Vienna in six months. There was an epidemic streptococcal infection in middle Europe in the winter of 1791. Actually, Mozart died of acute nephritic syndrome due to post-streptococcal glomerulonephritis.

Another famous composer I like to mention is Johann Strauss I - maestro of the Waltz. There were three generations of Strauss. Johann Strauss I died in Vienna in 1849 A.D. from scarlet fever contracted from one of his illegitimate children. When he was critically sick, the attending physician gave three instructions: clean the room, put him in a side room and ask for a priest. Then he died. Nowadays we just give him a prescription of penicillin, ask him to stay at home, and he will give his concert in two weeks.

Figure 8: CAPTAIN GEORG RITTER VON TRAPP (1880- 1947), NAVAL OFFICER OF THE AUSTRIAN-HUNGARIAN EMPIRE

Let us continue our story of scarlet fever in the 20th century. In mid 19th century, there was an English engineer called Robert Whitehead who graduated from Manchester. He invented the first generation of self-propelled torpedo. He wanted to sell his torpedo to British Navy but the British Naval Office did not recognise its importance. He went to Europe to sell his invention. He established his factory in Flume within the Austrian-Hungarian Empire around 1870. At that time the Austrian-Hungarian Empire had a large navy with seaports in the Balkan peninsula. Whitehead left his fortune to his granddaughter Agathe Whitehead. In 1911, Agathe married Captain Georg Ludwig van Trapp, who used torpedoes as a submarine

commander in the First World War [Figure 8]. He sunk 12 cargo vessels, the French armoured cruiser *Leon Gambetta* (12,600 tons) and the Italian submarine *Nereide* (225 tons). Georg and Agathe had 7 children. Agathe died of Scarlet Fever at around 1920. Hence, Von Trapp had to find a teacher. This teacher was Maria Kutchera, a postulant from Salzburg intending to become a nun. She became their stepmother and matriarch of the Trapp Family Singers. Her story served as the inspiration for a 1956 German film that in turn inspired the Broadway musical and subsequently the 1965 blockbuster film "The Sound of Music". In the pre-antibiotic era, people succumbed to infection in their early life. In contrast, deadly infections like Scarlet Fever can now be treated with antibiotic as outpatient..

Finally, let us move to the last part of my talk - Medicine in Art. In the Renaissance period, one of the most influential painter was Rembrandt. His famous painting is called Night watch. Rembrandt painted *The Militia Company of Captain Frans Banning Cocq* between 1640 and 1642. This picture was called the *Nachtwacht* by the Dutch and the *Night Watch* by Sir Joshua Reynolds because by the 18th century the picture was so dimmed and defaced by time that it was almost indistinguishable and it looked quite like a night scene. After it was cleaned, it was discovered to represent broad day—a party of musketeers stepping from a gloomy courtyard into the blinding sunlight. Rembrandt actually painted pictures for free. He painted pictures for Council officers in Amsterdam. He also painted pictures for sales. One of his lovely painting series is called Toilet of Bathsheba depicting Queen Bathsheba having a bath. I show you two versions of the theme: one in the Metropolitan Museum of Art in New York and the other in the Louvre Museum in Paris. Rembrandt usually painted a series of pictures like these with variations such that he could sell to different people. For the painting in Louvre Museum, Bathsheba was holding a letter from King David. Oncologists always take these paintings as example of breast cancer in historic art. Medical researchers have observed a lumpy deformity in the left breast of the model for Bathsheba, and have offered various hypotheses including breast cancer, abscess due to tuberculosis and lactation mastitis following an unsuccessful pregnancy. Oncologists are attracted to the hypothesis that she had breast cancer. The model was Rembrandt's partner named Hendrickje Stoffels. She was at the age of 28 when these pictures were painted. Historians disputed against this idea of breast cancer because she lived for another 8 years before she died. She never had any surgery nor received any chemotherapy. It was thought that this lady had lactation mastitis which lasted about nine years before she died.

So much for the painting in the West. Now we come to the painting in the East. In early 19th century, there was a famous painter in Macau and Hong Kong from England called George Chinnery. Chinnery's paintings collected by the Hong Kong and Shanghai Bank and Jardine, Matheson & Co. are exhibited in the Hong Kong Museum of Art. Chinnery introduced a western style of landscape and portrait. Chinese pupils of Chinnery painted similar subjects but at a cheaper price. One of his famous pupil was 關喬昌, also known as Lam Qua. By 1840, Lam Qua became well-known and skilled in Western style of portraiture and developed a following among the international community resident in Canton. He was commissioned by Western surgeons practising in Canton to paint 114 medical illustrations of patients with different illnesses. Lam Qua painted the disturbing series of medical portraits that accompanied Reverend Peter Parker on his fund-gathering mission to medical schools and Protestant authorities in the West. These paintings had only been publicly exhibited once. Curators shied away from their grotesque, unflinching portrayal of cysts, tumours, growths and amputations juxtaposed with perplexingly serene human hosts: too unsettling, too "inappropriate for public view." Now medical artists recognise these paintings represent the largest unified body of extant works by the artist (86 pieces are in the collection of Yale Medical College Library; another smaller group is at Guy's Medical Hospital in London; and four paintings are housed at Cornell University). The following painting is a large pendulous neurofibroma now kept at the Gordon Museum in London. The second painting is a giant lipoma now housed in the Yale University Medical Museum. Unfortunately, not one of these 114 paintings stays in China or Hong Kong.

In conclusion, I have demonstrated how interesting humanity issues are found in Medicine. No better person than clinicians themselves can illustrate vividly to medical students the symbiosis of humanity and medicine. Humanity should be integrated into our medical curriculum by drawing examples as I have just shown. I like to dedicate my talk to the late Professor Gerald Choa for his contribution to the College and medical practice in Hong Kong. The last slide is a picture of a Chinese village in Anhui with seal at the right lower corner [Figure 9]. It reads"大夫春滿杏林". 大夫 is the Chinese term for doctors and 春滿杏林 originates from Confucius (孔子). He taught students in his peach garden and these outstanding students perpetuated his teaching thereafter. This is what Professor Choa had been doing in the last 50 years in Hong Kong. Thank you.

Figure 9: WORLD HERITAGE IN CHINA - 皖南古村落 — 宏村

Chronic Kidney Disease - Prevalence in China and around the world

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Overview

Chronic non-communicable diseases (CNCDs) present a global crisis, impeding development goals such as poverty reduction, health equity, economic stability, and human security. Among CNCDs, chronic kidney disease (CKD) imposes a significant burden for renal replacement therapy, on mortality and accelerated cardiovascular events.[1-3] Also, CKD is associated with increased risks of acute kidney injury, mineral and bone disease, adverse metabolic and nutritional consequences, infections, and reduced cognitive function. Consequently, the financial expenditure and medical resources consumed for the management of CKD patient are much higher than expected.

Prevalence of CKD

For the past decade, numerous cross-sectional surveys regarding the prevalence of CKD were published, mainly in the developed countries. In the United States, results of the National Health and Nutrition Examination Surveys (NHANES) 1999-2004 revealed a prevalence of 13.1%.[4] Recently, two national surveys of CKD among the developing countries, the Thai Screening and Early Evaluation of Kidney Disease (SEEK) study and the China National Survey of Chronic Kidney Disease,[5,6] indicate that CKD has become a leading public health problem worldwide.

The Chinese study used a multistage, stratified sampling method of people aged 18 years or older,[6] used eGFR calculated by the Chinese equation,[7] and albuminuria as defined by ACR of 30mg/g creatinine or higher, and reported a prevalence of CKD of

1.2 billion adults have CKD …… only 12·5% of them are aware of the condition.

10.8%[6]. Altogether 1.2 billion adults aged 18 years or older have CKD as defined in this study, and only 12·5% of them are aware of the condition. A lower prevalence of stage 3 and stage 4 CKD in China compared with developed countries was found. For example, the prevalence of stage 3 CKD was 1.6% in China, compared with 7.7% in the USA and 4.2% in Norway. One explanation might be that hypertension and diabetes have increased rapidly in the past 15–20 years in China, but it will take 10 years for these diseases to affect kidney. This lag provides a unique opportunity for measures to slow the increase of CKD prevalence caused by hypertension and diabetes in China.

Causes of CKD

Chronic glomerulonephritis is an important cause of CKD in China. According to a report from the Dialysis and Transplantation Registration Group of China in 1999,[8] about half the patients receiving chronic dialysis were diagnosed as chronic glomerulonephritis. IgA nephropathy predominates in China, Southeast Asia and the Pacific region. On contrast, focal segmental glomerulosclerosis is the most common type among the black populations of Africa, Saudi Arabia, India, and South America.[9]

Diabetic nephropathy is recognized as the most common cause of end-stage renal disease (ESRD) with an increasing tendency of diabetic nephropathy among incident hemodialysis patients, especially patients aged over 50.[10] Firstly, a rapid surge in diabetes has resulted in increasing the prevalence of diabetes in China from 1% in 1980[11] to 9.7% in 2008.[12] Secondly, underdiagnosed diabetes is common, as indicated in a national survey of diabetes in China where 59.7% of patients were diagnosed through screening.[12]

Hypertension is also one of important causes of CKD. Similar to diabetes, it has an escalating prevalence, low awareness rate, and sub-optimal treatment, especially in rural area. In 1991, the overall prevalence of hypertension in China was 13.6%.[13] Ten years later, the number was reported to be 23% in urban area and 18% in rural area.[14] Awareness rates and control rates of hypertension were 24% and 19%,[14] comparatively lower than in developed countries.

A significant proportion of the Chinese population depends on indigenous systems of medicine. A recent study[15] using a national-representative sample in China indicated that long-term intake of herbs containing aristolochic acid was independently associated with eGFR <60 mL/min/1.73 m^2 and albuminuria, with odd ratio of 1.83 (95% CI, 1.22-2.74) and 1.39 (95% CI, 1.03-1.87), respectively. Longitudinal studies from Taiwan revealed that use of aristolochic acid containing herbs, especially >60 g of Mu Tong or Fangchi from herbal supplements, is associated with increased risk of developing kidney failure.[16]

This lecture was delivered at the 18th Hong Kong Medical Forum held in Hong Kong on May 11-12, 2013 by Professor Haiyan Wang. The Department of Medicine, The University of Hong Kong has kindly granted permission for the article to be printed in Synapse.

The health and economic burden of CKD

One of the ultimate outcomes of chronic kidney disease is ESRD, which necessitates ever-growing dialysis and transplantation programs, and therefore places an unaffordable financial burden on developing countries. A recent national survey in China estimates the number of patients with CKD in China to be 1.2 billion.[6] If 1% progressed to ESRD, the total costs of dialysis would amount to twice the current health-care budget in China. The dialysis rate is low in China as compared to that in developed countries, being limited by affordability and accessibility of the treatment. Almost all hemodialysis centers are located in cities in China,[17] which are already believed to be running at capacity. Peritoneal dialysis which has grown at a rate of 30% per annum in China over the past two years,[18] are also located in cities.

The economic cost associated with milder forms of CKD was even higher. Cardiovascular disease (CVD) and CKD share common risk factors such as hypertension, diabetes, smoking, obesity, hyperlipidemia and aging as well as some non-traditional risk factors such as vitamin D deficiency, hyperphosphatemia, anemia, and albuminuria.[19] Even stage 1 or stage 2 CKD are associated with an increased risk of adverse overall, cardiovascular and renal outcomes.[3,20] A recent study from China[21] indicated that individuals with subtle decreased renal function seemed much more likely to have multiple cardiovascular risk factors and have higher prevalence of CVD than those without CKD. Furthermore, CKD is associated with high burden of other chronic conditions, including metabolic syndrome,[22] cognitive decline,[23] and ocular fundus pathology,[24] which contributed to the adverse patients' outcome and elevated health care costs.

Prevention and early detection of CKD

Although CKD shares common risk factors and/or coexists with other CNCDs, it has not received the same kind of attention in China.

Lifestyle intervention of common risk factors for CNCDs is important to the prevention of CKD. Several priority interventions were chosen to cope with the global CNCD crisis, including accelerated tobacco control, salt reduction, promotion of healthy diets and physical activity, and reduction of harmful alcohol consumption.[21] Those interventions are confirmed to be cost-effective in countries with a variety range of incomes.[25] Furthermore, optimal control of diabetes and hypertension is also crucial for the CKD prevention.[9]

Long-term intake of herbs containing aristolochic acid was independently associated with eGFR <60 mL/min/1.73 m^2 and albuminuria.

Screening for CKD among "high-risk" population (e.g., older than 60 years or with hypertension, diabetes) is proved to be cost-effective based on data from developed countries.[26] Initial screening using urinary dipstick test is useful for risk stratification,[3] considering the low awareness rate of CKD in China.[6] Along these lines, the Research and Prevention Committee of the International Society of Nephrology ISN-GO has developed a global early detection and intervention program for emerging countries that can be implemented according to local needs and facilities.[27]

Such programs to combat CKD, diabetes, hypertension, and CVD need to be closely integrated in China. Such integrated preventive measures could be performed by the medical assistants and nurses with low expenses. However, nephrologists need to be heavily involved to train primary care practitioners and to establish referral systems.

If 1% progressed to ESRD, the total costs of dialysis would amount to twice the current health-care budget in China.

Reference

1. Bello AK, Nwankwo E, El Nahas AM. Prevention of chronic kidney disease: a global challenge. Kidney Int Suppl. Sep 2005(98):S11-17.
2. Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. N.Engl.J.Med. 2004;351(13):1296-1305.
3. Matsushita K, van der Velde M, Astor BC, et al. Association of estimated glomerular filtration rate and albuminuria with all-cause and cardiovascular mortality in general population cohorts: a collaborative meta-analysis. Lancet. Jun 12 2010;375(9731):2073-2081.
4. Coresh J, Selvin E, Stevens LA, et al. Prevalence of chronic kidney disease in the United States. JAMA. 2007;298(17):2038-2047.
5. Ingsathit A, Thakkinstian A, Chaiprasert A, et al. Prevalence and risk factors of chronic kidney disease in the Thai adult population: Thai SEEK study. Nephrol Dial Transplant. May 2010;25(5):1567-1575.
6. Zhang L, Wang F, Wang L, et al. Prevalence of chronic kidney disease in China: a cross-sectional survey. Lancet. Mar 3 2012;379(9818):815-822.
7. Ma YC, Zuo L, Chen JH, et al. Modified glomerular filtration rate estimating equation for chinese patients with chronic kidney disease. J.Am.Soc.Nephrol. 2006;17(10):2937-2944.
8. Dialysis and Transplantation RegistrationGroup CSoN, Chinese Medical Association. . The report about the registration of dialysis and transplantation in China 1999. Chin J Nephrol. 2001;17:77-79.
9. Barsoum RS. Chronic kidney disease in the developing world. N Engl J Med. Mar 9 2006;354(10):997-999.
10. Beijing Hemodialysis Quality Control and Improvement Center. The Annual Data Report of Beijing Hemodialysis Quality Control and Improvement Center, 2012. Chinese Journal of Blood Purification. 2012;z1:42.
11. Zhong XL. Diabetes mellitus survey in China. Chin Med J (Engl). Jun 1982;95(6):423-430.
12. Yang W, Lu J, Weng J, et al. Prevalence of diabetes among men and women in China. N Engl J Med. Mar 25 2010;362(12):1090-1101.
13. Tao S, Wu X, Duan X, et al. Hypertension prevalence and status of awareness, treatment and control in China. Chin Med J (Engl). Jul 1995;108(7):483-489.
14. Wu Y, Huxley R, Li L, et al. Prevalence, awareness, treatment, and control of hypertension in China: data from the China National Nutrition and Health Survey 2002. Circulation. Dec 16 2008;118(25):2679-2686.
15. Zhang J, Zhang L, Wang W, Wang H. Association Between Aristolochic Acid and CKD: A Cross-sectional Survey in China. Am J Kidney Dis. Mar 2 2013.
16. Lai MN, Lai JN, Chen PC, Hsieh SC, Hu FC, Wang JD. Risks of kidney failure associated with consumption of herbal products containing Mu Tong or Fangchi: a population-based case-control study. Am J Kidney Dis. Mar 2010;55(3):507-518.
17. Zhang L, Wang H. Chronic kidney disease epidemic: cost and health care implications in China. Semin Nephrol. Sep 2009;29(5):483-486.
18. Xu R, Han QF, Zhu TY, et al. Impact of individual and environmental socioeconomic status on peritoneal dialysis outcomes: a retrospective multicenter cohort study. PLoS One. 2012;7(11):e50766.
19. Sarnak MJ, Levey AS, Schoolwerth AC, et al. Kidney disease as a risk factor for development of cardiovascular disease: a statement from the American Heart Association Councils on Kidney in Cardiovascular Disease, High Blood Pressure Research, Clinical Cardiology, and Epidemiology and Prevention. Circulation. Oct 28 2003;108(17):2154-2169.
20. Gansevoort RT, Matsushita K, van der Velde M, et al. Lower estimated GFR and higher albuminuria are associated with adverse kidney outcomes in both general and high-risk populations. A collaborative meta-analysis of general and high-risk population cohorts. Kidney Int. Feb 2 2011.
21. Beaglehole R, Bonita R, Horton R, et al. Priority actions for the non-communicable disease crisis. Lancet. Apr 23 2011;377(9775):1438-1447.
22. Zhang L, Zuo L, Wang F, et al. Metabolic syndrome and chronic kidney disease in a Chinese population aged 40 years and older. Mayo Clin.Proc. 2007;82(7):822-827.
23. Wang F, Zhang L, Liu L, Wang H. Level of kidney function correlates with cognitive decline. Am J Nephrol. 2010;32(2):117-121.
24. Gao B, Zhu L, Pan Y, Yang S, Zhang L, Wang H. Ocular fundus pathology and chronic kidney disease in a Chinese population. BMC Nephrol. 2011;12:62.
25. Gaziano TA, Galea G, Reddy KS. Scaling up interventions for chronic disease prevention: the evidence. Lancet. Dec 8 2007;370(9603):1939-1946.
26. Boulware LE, Jaar BG, Tarver-Carr ME, Brancati FL, Powe NR. Screening for proteinuria in US adults: a cost-effectiveness analysis. JAMA. 2003;290(23):3101-3114.
27. Perico N, Bravo RF, De Leon FR, Remuzzi G. Screening for chronic kidney disease in emerging countries: feasibility and hurdles. Nephrol Dial Transplant. May 2009;24(5):1355-1358.

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Newly Elected Fellows of The Royal College of Physicians, London

Elected in 2013

Dr Chan Kam Tim
Dr Cheng Yuk Lun
Dr Hung Cheung Tsui
Dr Hung Fan Ngai Ivan
Dr Kwan Ching Ha Bonnie
Dr Leung Yin Yan Jenny
Dr Lam Yat Yin
Dr Ronald Ma
Dr Mo Ka Keung Loar
Dr David Siu
Dr Janice Tsang
Dr Wong Mo Lin Maureen
Dr Wong Wai Leung
Dr Justin Wu

Newly Elected Fellows of The Royal College of Physicians, Edinburgh

Elected since March 2010

Dr Chan Hon Wai Felix
Dr Chan Chi Wai Kenny
Dr Chan Kit Yan Selina
Dr Chau Tai Nin
Dr Chow Siu Lun Eddie
Dr Fung Ka Shun Samuel
Dr Ho Wing Ming
Dr Hong Kam Fai Jeffrey
Dr Kwan Ching Ha Bonnie
Dr Kwan Yiu Keung
Dr Lai Kang Yiu
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Dr Lau Chi Pan Patrick
Dr Lau Yuk Kong
Dr Lee Chi Yan Conrad
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Dr Tam Chi Ming
Prof Tam Lai Shan
Dr Tam Li Wah
Dr Tam Kui Fu Stanley
Dr Tse Pak Yiu
Dr Tung Yau Man Stephen
Dr Wong Bun Lap Bernard
Dr Wong Lai Hung Grace
Dr Wong Sau Wai Grace
Dr Wong Ming Ho
Dr Wong Siu Yin
Dr Wong Wing Hang
Dr Yeung Shing Joseph
Dr Yeung Yiu Cheong
Dr Yip Man Lung
Dr Yong Sai Ping David
Dr Yuen Ka Hong

Announcement

The HKCP Annual Scientific Meeting will be held on the 19-20 October 2013 at the HKAM Jockey Club Building. The theme is “Novel Diagnostic Technologies in Internal Medicine”. The Annual General Meeting will be held at 6 pm on 19 October 2013 at the same venue. The flyer with details of the program will be sent out shortly.

Medical Oncology – Our Journey Continues...

Dr. Janice Tsang[1] & Professor Anthony Chan[2]

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[2] Li Shu Fan Medical Foundation Professor of Clinical Oncology, Sir YK Pao Centre for Cancer, Faculty of Medicine,The Chinese University of Hong Kong

The Birth of Medical Oncology

The origin of Medical Oncology lies in the haematological and paediatric malignancies. It began very much as a small research oriented specialty and till now clinical research remains an important feature of its activities. The emergence of cancer chemotherapy in the second half of the 1940s led to the principles of chemotherapy being laid out with only a few limited drugs at that time. Over a short period of time, advances in chemotherapy have led to cures in such diseases as childhood acute leukaemia and Hodgkin's disease as well as improved disease-free survival in breast cancer. Such achievements over the decades have paved the way and led to the birth of the specialty of Medical Oncology in 1972.

The dawn on chemotherapy in the 1940s began with the confirmed effect of nitrogen mustards in lymphomas. For the first time, the hope of curing human cancer with chemotherapy was a possibility. This was later followed by the leadership of Sir Austin Bradford Hill, who introduced the concept of controlled clinical trials by using individual random assignment. This innovative approach was adopted as an essential part of cancer medicine, as a tool for drug evaluation and revolutionized practice for investigating research questions and knowledge gaps. Since then, the unmet need for dedicated specialists in Medical Oncology became well recognized in the Western world during the 1970s and 1980s. Over the past 20 years, enormous developments have taken place in the medical management of cancer and particularly the development of orthodox therapies for common solid tumours. Today, Medical Oncology is a broad-based clinical Medical specialty with the responsibility to ensure that the state-of-the-art therapies of established efficacy for common cancers are delivered on a national basis, within a framework of care for the patient as an individual. Therefore, medical oncologists are trained to work as part of a multidisciplinary team, able to advise on all aspects of treatment including surgery and radiotherapy, as well as having the skills in training to deliver specialist medical therapy.

The History of Medical Oncology in Hong Kong

The history of Medical Oncology in Hong Kong traced back to the establishment of the Hong Kong Cancer Institute in 1990 at the Chinese University of Hong Kong under the Chairmanship of Professor Gerald Choa. The first comprehensive cancer centre in Hong Kong, the Sir Y.K. Pao Centre for Cancer at the Prince of Wales Hospital, was inaugurated in November, 1994 by HRH Prince Charles. The first Director of the Cancer Centre was Professor Philip Johnson who was a hepatologist and medical oncologist from King's College London. He was also appointed concurrently as the Professor and Chairman of Clinical Oncology at the Chinese University of Hong Kong. Medical Oncology was then recognized by the Hong Kong College of Physicians as a new specialty in 1996. At that time, the President of the College, Professor Tai-Kwong Chan, and the Chairman of the Education and Accreditation Committee, Professor Richard Yu, set up a Specialty Advisory Committee to develop training guidelines for Medical Oncology, the first version of the new guidelines being adopted in 1997. Medical Oncology was incorporated in the Joint Specialty Board of Haematology/Haematological Oncology/Medical Oncology which was co-chaired by Professor Tai-Kwong Chan and Professor Philip Johnson at that time. In January, 1998, Medical Oncology was formally recognized as a specialty by the Hong Kong Academy of Medicine. The first qualified fellows included prominent private medical oncologists such as Drs Diana Siu, Edmond Chiu, and Paul Cheng, and outstanding academic staff at both the Chinese University of Hong Kong such as Professor Anthony Chan, Professor Winnie Yeo, Professor Tony Mok, as well as The University of Hong Kong such as Professor Tai-Kwong Chan, Professor Wah-Kit Lam, and Professor Raymond Liang. Many of these senior pioneer medical oncologists had received their overseas specialty training in Australia, Canada and the United Kingdom.

In 2002, Professor Anthony Chan succeeded Professor Johnson as the Director of the Hong Kong Cancer Institute and the Sir Y.K. Pao Centre for Cancer. He also took over the chairmanship of Clinical Oncology department at the Chinese University of Hong Kong, and was appointed the Co-chairman of the Medical Oncology Specialty Board after Professor Johnson returned to the UK. In line with the Hong Kong College of Physicians, he recognized the increasing demands for medical oncologists due to an ageing population with rising numbers of cancer patients and cancer survivors, and advances in medical treatment such as molecular targeted therapy for patients with multiple co-morbidities. This formed the rationale for a broad-based training program providing comprehensive clinical experience for trainees to acquire competency and professionalism as a specialist in Medical Oncology. Training programmes included exchange opportunities between the two University teaching hospitals which later extended across clusters and departments of peripheral hospitals. This has led to the independent status of the Medical Oncology Specialty Board under the chairmanship of Professor Winnie Yeo in April, 2010. Since then, with the concerted efforts of all the Specialty Board members for Medical Oncology and the Programme Directors from all clusters, the Medical Oncology Specialty Board has been committed to ensure the highest standards of qualification of medical oncologists, with the highest standards of patient-centered training programmes dedicated for Medical Oncology trainees. These training programmes are further strengthened and enhanced with regular reviews and re-certification. Over the past decades, the concept of multidisciplinary team (MDT) model for cancer care has been introduced through the support of the medical oncologists, engaging other cancer care working partners, including clinical oncologists, surgical colleagues, radiologists, pathologists, nurses and para-medical colleagues. At the moment, the MDT model for cancer care is adopted with the organ-based approach in all major oncology centres or units in Hong Kong.

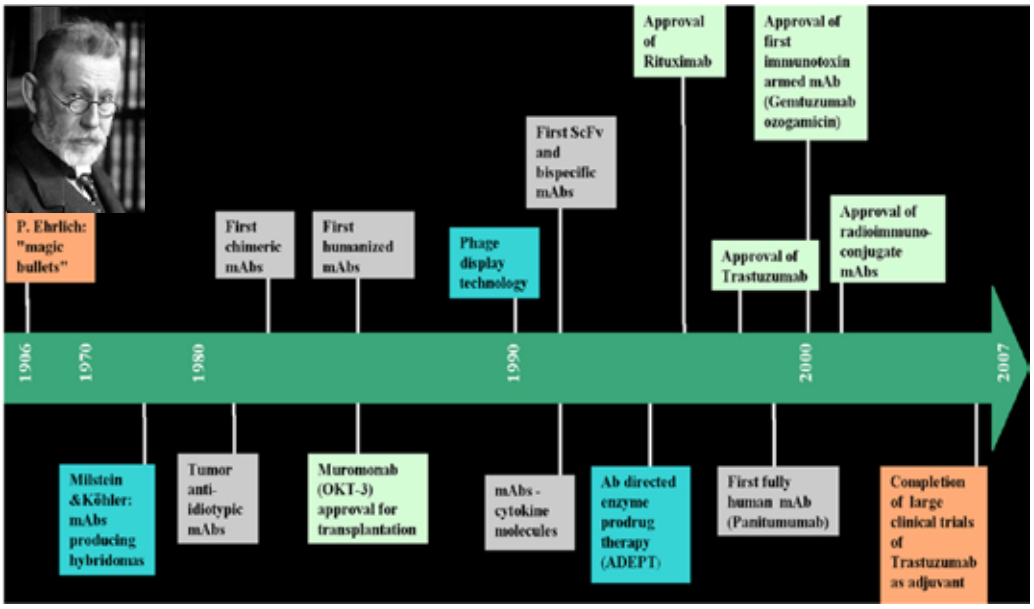
The Changing Landscape of Oncology – The Advent of Molecular Targeted Therapy & Personalized Medicine

Over the past decades, there has been a paradigm shift not only in the diagnosis, but also the treatment of solid tumours. Our understanding of tumour biology and the advances in molecular biology have led to the rapid development of personalized cancer management. In the old days, the main modalities of treatment for solid tumours include surgery, radiotherapy and medical treatment such as chemotherapy. Since the arrival of the millennium, with the advent of molecular targeted therapy such as the story of imatinib for chronic myeloid leukaemia targeting BCR-ABL, the constitutively activated tyrosine kinase fusion protein caused by the Philadelphia chromosome translocation; later followed by the story of trastuzumab giving superior overall survival benefits for the aggressive subset of breast cancer women having over-expression of the HER-2 receptor, there followed many more new drug articles and the expansion of our armamentarium of medical treatment including the ever-increasing list of molecular targeted therapies (Figure 1.). This has led to the era of personalized medicine where after the histological confirmation of diagnosis of the primary cancer, we often need to ascertain the particular characteristics or subtypes of the primary tumour in order for us to decide the best treatment plan for the patients. For example, the HER-2 positive breast cancer women will benefit from the use of anti-HER-2 targeted therapy; the EGFR mutation positive non-small cell lung cancer will benefit from the tyrosine kinase inhibitors; the K-ras wild type colorectal cancer patients will have added benefits of the epidermal growth factor receptor monoclonal antibodies in the metastatic setting. Progress in molecular biology and genomics is producing a revolution in cancer research and treatment. By uncovering the molecular heterogeneity of tumours, the "one-size-fits-all" approach is now over for we understand that different oncogenic drivers as well as mechanisms or resistance are being discovered in tumours that look pathologically the same. The conventional pathological features such as tumour size, nodal involvement still play a role in the cancer treatment decision process, but they are clearly not sufficient to establish or better predict the biological behaviour of the disease. These advances have further led to the changes in clinical trial designs and patient treatment decision making.

Furthermore, while an ever-growing list of new drug articles is being introduced into the market or tested in multi-centre randomised controlled trials, similarly exciting advances are occurring in the fields of surgical oncology and radiation oncology. Taking surgery as an example, where the definitive treatment for common cancers like breast and colon cancers has traditionally been the surgical removal of the primary tumour, there is now increasing data showing the added value of neoadjuvant therapy or induction therapy to down-size or down-stage the primary tumour pre-operatively, and is an opportunity to control occult metastasis for the patient. It also provides a new platform for both oncologists and scientists to study the pathological and treatment responses towards a particular medical treatment, leading to new opportunities and better understanding of the biology and molecular mechanisms of the tumour. On the other hand, there are also new developments in the field of radiotherapy with various kinds of new techniques emerging, leading to more focused and "targeted" approach of either definitive radical or palliative radiotherapy for the best locoregional control of the tumour.

Therefore, with all these advances in cancer management, currently the most optimal approach to treat a cancer patient requires the knowledge of who is the right patient/tumour to treat, which is the right drug to give, and when is the right time to offer radiotherapy, surgery or any medical intervention. This certainly requires a well-rounded approach with a team of experts working together towards common objectives of maximising treatment efficacy and patient outcomes, achieving the best cost-effective index, enhancing patient safety and minimising toxicity (Figure 2.).

The Revolution of Molecular Targeted Cancer Therapy



Timeline – Timeline of discoveries leading to the burgeoning clinical application of cancer therapeutic antibodies. Adapted from "Cancer therapeutic antibodies come of age: Targeting minimal residual disease," by Ben-Kasus et al., 2007, *Molecular Oncology*, 1(1), p. 43. Copyright 2007 by Elsevier B.V.

Figure 1

Personalized Management of Cancer

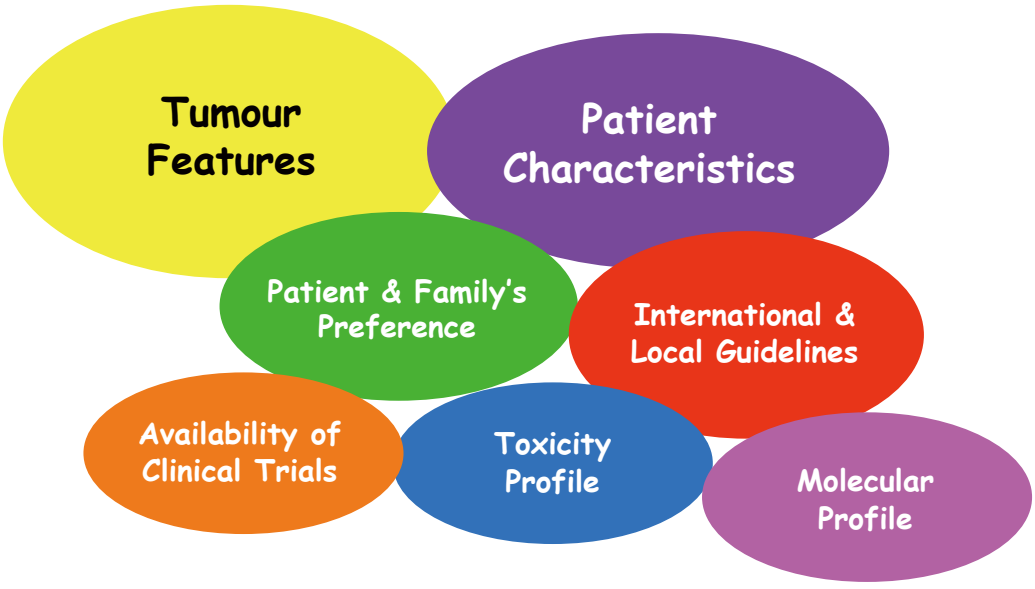


Figure 2

The Multidisciplinary Team (MDT) Model

Cancer care is now a complex issue not only with the more than one option for a particular patient with a particular type of primary disease, but also other challenges such as matching science with affordability, respecting patient and family's wishes and addressing psychosocial unmet needs.

The concept of the cancer Multidisciplinary Team (MDT) approach was formally introduced into the UK in the 1990s and endorsed as the principle way of managing cancer patients. A major impetus was the publication of the Calman-Hine report in 1995, and the consequent drive to ensure that all patients with cancer, no matter where they might live, and to whom they might have been referred, would have equal access to a high and uniform standard care. Since the 1990s, there have been at least 1500 cancer MDTs currently active in the UK with evidence of improvement in overall outcomes for patients with cancer. Nowadays, MDTs are also known as "tumour boards" or "multidisciplinary conferences" and have become an integral component of contemporary comprehensive cancer care.

In line with the overseas MDT approach, our Medical Oncology Specialty Board has introduced the concept of MDT for cancer care which has been adopted as part of the standard pathway among all the cancer centres in Hong Kong. The MDT now forms part of the daily work and a form of institutionalised communication in most hospitals providing cancer care services, in particular the 6 major cancer centres, where collaboration between clinical oncologists, surgeons, radiologists and pathologists have had a direct positive impact on the quality of patient care. Usually the MDT includes medical oncologists, clinical oncologists, relevant surgeons, radiologists, pathologists, nurse specialists, medical social workers, para-medics such as physiotherapists, occupational therapists and clinical psychologists (Figure 3.). A consensus decision based on a multidisciplinary discussion offers the benefits of combined expertise rather than just the sum of individual opinions. The last decades have witnessed the growth of many more organ-specific MDT meetings held at all the 6 cancer centres such as breast MDT meetings, colorectal MDT meetings, lung MDT meetings and head and neck MDTs etc.

There are multiple advantages for conducting MDT meetings to discuss new and complicated patients: 1) to enhance the communication among all experts from different specialties dedicated for the particular type of primary cancer; 2) to facilitate discussion for the best standards of care for each patient, especially in the public sector where there is no named-doctor system, so that both trainees or registrars can contribute to the team decision on the treatment plan and accountability is ensured; 3) to provide an additional platform for continued medical education and exchange of ideas among various specialists as well as widening training exposure for trainees and registrars.

The Multidisciplinary Team Model for Cancer Care (MDT Model)

Figure 3

The "Second Wave" of Personalized Medicine - Molecular Genomic Medicine

In parallel to continuous drug development and new advances in cancer care, medical oncologists are confronted with additional challenge of how to achieve the maximal selectivity towards the primary tumour while minimizing the toxic effects on the host, given that tumour cells continue to proliferate and develop drug resistance. Over the past decades, from our journey to the era of personalized medicine where we are now treating cancer patients with the MDT approach, identifying patients with specific oncogenic drivers with different tumour subtypes, our understanding of the tumour biology and the recent advances in molecular genomic profiling has further led us to a second wave of personalized medicine – predicting the benefits of a particular treatment for a particular patient or from our knowledge of the molecular genomic profiling of the tumour to see if we could spare some patients from unnecessary toxicities such as chemotherapy.

On the other hand, the degree of genetic heterogeneity within tumours from individual patients in both space and over time is increasingly well characterized. Recent whole-genome sequencing studies have reported tens of thousands of somatic mutations in different cancers. However, the evidence suggests that only a small minority of these are essential for cancer development ("driver mutations") with the majority having no significant biological impact ("passenger mutations"). At the same time, different cancer clones within a tumour are in constant competition, with the "fittest" clones proliferating at the expense of "less fit" clones. Key driver mutations are thought to provide a selective advantage on a cell to facilitate its clonal expansion.

Over the years, there have been increasing numbers of studies that have used the genomic molecular profiling technology with an attempt to identify new biomarkers for prognosis and treatment response for solid tumours.

One common example is the use of molecular genomic profiling to further stratify hormone positive HER-2 negative node negative breast cancer patients to low, intermediate or high risk of disease recurrence using a 21-gene molecular profiles for breast cancer, to facilitate further fine clinical decisions on treatment with adjuvant chemotherapy, thus sparing those low-risk patients (with a low recurrence score) from the unnecessary toxicities of chemotherapy. This has led to further professional continuous education and demand for improving communication skills for oncologists or physicians involved in cancer treatment.

Medical Oncology – Our Journey Continues

Cancer is indeed an important global and public health issue. It is one of the top killers in Hong Kong and yet it has also become a prevalent disease. With the ageing population as well as changes in lifestyle and eating habits, the incidence of various common cancers is increasing and even for patients with locally advanced or metastatic disease, their clinical courses vary and many of them live with the disease for years as a result of new advances in diagnosis and treatment. Thanks to these new advances for cancer medicine over the past decades, which include drug developments, medical technological breakthroughs and the promise of molecular genomic medicine, common cancers have now become a "chronic" illness, hence raising the challenge of matching science with affordability. There is much hope and promise for our cancer patients. For the future, the field of oncology faces challenges of a growing specialty and knowledge gaps for further research.

Over the years, members of the Medical Oncology Specialty Board with support from the Hong Kong College of Physicians have planned to address increasing unmet needs for cancer medicine – demands for medical oncologists to manage the growth in numbers of newly diagnosed cancer patients and the care of survivors; the explosion of an armamentarium of medical treatments and targeted anti-cancer agents; the needs of different subgroups of cancer patients such as geriatric oncology patients, young cancer patients and women cancer patients as well as male cancer patients. Currently, 31 Medical Oncology specialists are registered with the Hong Kong Medical Council and services provided by medical oncologists in collaborations with other key players of the MDT cancer care model are available in major hospitals. Over the last decade, there have been more than 20 doctors undergoing training locally and will become specialists in Medical Oncology. Besides the University teaching hospitals, we have medical oncologists serving in major regional hospitals, as well as in the private sector.

There are indeed many more challenges along our journey of combating against cancer, but with our firm belief of holistic care, our vision and mission to work together, we will certainly continue to see further improvements in the outcome of our cancer patients, while working even more closely together to triumph over this common disease. Our journey continues…

References

Awada A, Vandone A.M., Aftimos P. (2012). Personalized management of patients with solid cancers: moving from patient characteristics to tumor biology. Curr Opin Oncol, 24(3): 297-304.

Baird R, Caldas C. (2013). Genetic heterogeneity in breast cancer: the road to personalized medicine. BMC Med, 11:151. doi: 10.1186/1741-7015-11-151.

Band P.R. (2010). The birth of the subspecialty of medical oncology and examples of its early scientific foundations. J Clin Oncol, 28(22):3653-3658.

Ben-Kasus T., Schechter B., Sela M., Yarden Y. (2007). Cancer therapeutic antibodies come of age: targeting minimal residual disease. Mol Oncol, 1(1):42-54.

Chan A.T.C. (2011). Medical Oncology. Sapientia et Humanitas –A history of Medicine in Hong Kong. Hong Kong College of Physicians, 157-161.

de Boer R.H., Baker C, Speaker D, Chao C. Y., Yoshizawa C. Mann G.B. (2013). The impact of a genomic assay (Oncotype Dx) on adjuvant treatment recommendations in early breast cancer. Med J Aust, 199(3):205-8.

Dutcher J.P., Novik Y., O'Boyle Kevin, Marcoullis G., Secco C., Wiernik P.H. (2000). 20th-century advances in drug therapy in oncology – part I. J Clin Pharmacol., 40(9):1007-1024.

Fleissing A., Jenkins V., Catt S., Fallowfield L. (2006). Multidisciplinary teams in Cancer care: are they effective in the UK. Lancet Oncol, 7(11):935-943.

European Society of Medical Oncology (ESMO) / American Society of Clinical Oncology (ASCO) Recommendations for a Global Curriculum in Medical Oncology Update (2010)

Gerlinger M., Rowan A.J., Horswell S., Larkin J., Endesfelder D., Gronroos E et al. (2012). Intratumor heterogeneity and branched evolution revealed by multiregion sequencing. N Engl J Med, 366:883-892.

Guidelines on Postgraduate Training in Internal Medicine, Hong Kong College of Physicians (Fifth Edition, July, 2011)

Haward R.A. (2006). The Calman-Hine report: a personal retrospective on the UK's first comprehensive policy on cancer services. Lancet Oncol., 7(4):336-346.

Lamb B., Payne H., Vincent C., Sevdalis N., Green J.S.A. (2011). The role of Oncologists in multidisciplinary cancer teams in the UK: an untapped resource for team leadership. J Eval Clin Pract, 17(6):1200-1206.

Munror A.J., Swartzman S. (2013). What is a virtual multidisciplinary team (vMDT). Br J Cancer, 108(12):2433-41.

Pleasance E.D., Cheetham R.K., Stephens P.J., McBirdie D.J., Humphray S.J., Greenman C.D. et al. (2010). A comprehensive catalogue of somatic mutations from a human cancer genome. Nature, 463: 191-196.

Rouzier R., Pronzato P., Chereau E., Carlson J., Hunt B., Valentine W.J. (2013). Multigene assays and molecular markers in breast cancer: systematic review of Health economic analyses. Breast Cancer Res Treat, 139(3):621-637.

Ruhstaller T., Roe H., Thurlimann B., Nicoll J.J. (2006). The multidisciplinary meeting: an indispensable aid to communication between different specialties. Eur J Cancer, 42(15):2459-2462.

Specialty Training Curriculum for Medical Oncology endorsed by the Joint Royal Colleges of Physicians Training Board (August, 2010)

Examination Dates

Joint HKCPIE/MRCP(UK) Part I Examination 2014

14 January 2014	9 September 2014

Joint HKCPIE/MRCP(UK) Part II (Written) Examination 2014

9 -10 April 2014	10 -11 December 2014

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%)
10 and 11 December 2013	60	52 (87%)

Pass Rates for the Joint HKCPIE/ MRPC (UK) Part II PACES Examination

October 2001	36/72 = 50%
February 2002	34/74 = 46%
October 2002	29/72 = 40%
February 2003	30/69 = 43%
October 2003	27/59 = 46%
March 2004	39/64 = 61%
October 2004	26/69 = 38%
March 2005	35/75 = 47%
October 2005	28/75 = 37%
March 2006	36/75 = 48%
October 2006	16/73 = 22%
March 2007	44/74 = 59%
June 2007	44/74 = 59%
October 2007	36/55 = 65%
March 2008	36/74 = 49%
October 2008	29/65 = 45%
February 2009	39/75 = 52%
October 2009	24/72 = 33%
March 2010	33/75 = 44%
October 2010	40/74 = 54%
February 2011	23/66 = 35%
October 2011	34/70 = 49%
February 2012	32/74 = 43%
October 2012	32/74 = 43%
March 2013	28/75 = 37% (for HK Local candidates)

Pass List for the Joint HKCPIE/MRCP (UK) Part II PACES Examination March 2013

Cheng Hei Shun
Cheng Ka Chun
Ho Hung Kwong
Ho Ka Shing
Ho Wing Bing Lydia
Hui Wai Shan
Ip Chun Tak
Kwan Hon Hang
Lam Sai Tim
Li Man Lung
Lo Ying Fung
Lui Abdul Rashid Nok Shun
Luk Yee Andrew
Ng Hoi Yee Ivy
Ng Yu Chuen
Shum Chui Yin
Suen Gee Kwang Victoria
Sung Jonathan Gabriel
Sze Shu Yue
To Ki Wai Heather
Wong Ting Ting Angel
Wong Kin Ho
Wong Tin Chiu
Yeung Ka Pik Vivian
Yeung Wan Yin
Yiu Ka Ling
Yu Kit Yu
Yu Wen Ming

Important Notice to Basic Physician Trainees
A Case of Deferment of Higher Physician Training Commencement Date

Doctor A completed his Basic Physician Training (BPT) in November 2012. The Basic Physician Board issued a letter of Completion of Basic Physician Training to him in January 2013, stipulating that he should apply for College Membership within three months in accordance with College requirements. However, the College Secretariat only received Doctor A's Membership application in May 2013, after which he was admitted as College Member in the same month.

At its 249th Meting of 28 June 2013, the Council had decided that all Basic Physician Trainees who had completed 36 months' accredited training and passed the PACES examination must apply for College Membership within three months after being informed by the Basic Physician Board that the Council had approved the Dates of Completion of their Basic Physician Training (BPT) in order to apply the day after BPT Completion as their Dates of Commencement of Higher Physician Training. In addition, Trainees who had submitted insufficient or inaccurate information in their application forms must submit clarification within one month of enquiry from the Secretariat. Failure to apply for College Membership or respond to the Secretariat's enquiry within the time frames specified will result in automatic postponement of the Trainee's Date of Commencement of Higher Physician Training till College Membership is confirmed, which will impact on the subsequent progress of training. This rule has been promulgated to all Specialty Boards and Chiefs of Service in July 2012, and published in the September 2012 issue of Synapse.

Since Doctor A did not apply for College Membership within three months from the date of his letter on Completion of BPT, his Higher Physician Training commencement date has been deferred to the date of admission as College Member.

Notice to Basic Physician Trainees and Higher Physician Trainees in Broad-based Specialties (AIM and Geriatric Medicine)
New Regulations for Completion of Self Learning Tool (SLT)

Loretta Yam, Chairman
Education and Accreditation Committee

At its 260th Council Meeting of 30 May 2013, the Council has promulgated the following regulations regarding SLT for Basic Physician Trainees (BPT) and Higher Physician Trainees (HPT) in broad-based specialties (ie, Advanced Internal Medicine and Geriatric Medicine), for which SLT completion is an ongoing requirement of training:

(1) BPT and HPT Trainees who have not completed SLT must complete a remedial exercise, which may be held during office hours at the College Chamber. A Registration Fee of HK$500.00 will be imposed per Trainee.

(2) All BPT Trainees who fail to complete the remedial exercise will have their admission to College Membership deferred for a period of three months and must in addition complete SLT remedial exercise;

(3) All HPT Trainees who fail to complete the remedial exercise will have to postpone their Interim Assessments in AIM or Geriatric Medicine for a period of six months and must in addition complete the SLT remedial exercise. HPT Trainees who have passed the Interim Assessment will have to postpone their Exit Assessments in AIM or Geriatric Medicine for a period of six months and must in addition complete the SLT remedial exercise.

The above rules have already been disseminated to Trainees via the respective Boards and Chiefs of Service in June 2013, and will take effect from the next SLT cycle for BPT and AIM respectively in July – October 2013 and October 2013 – March 2014.

Statistics on No. of Trainees in all Specialties

Updated in July 2013

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	15	1 0 2 3 4 *5*	1 1 2 3 4—I *2*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 0 2 3 4 *5*	1 1 2—I 3 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 3—I 4 *1*	1 0 2 3 4 *0*
CRITICAL CARE MEDICINE	5	1 1 2 3—I 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 2 2—I 3—I 4 *4*	1 0 2 3 4 *0*
DERMATOLOGY & VENEREOLOGY	8	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 3—I 4 *0*	1 0 2 3 4 *0*
ENDOCRINOLOGY, DIABETES & METABOLISM	9	1 2 2 3—I 4—I *2*	1 0 2 3 4 *1*	1 0 2 3 4 *3*	1 0 2 3 4 *0*	1 0 2 3 4 *0*	1 1 2 3—I 4 *5*	1 0 2 3 4 *0*
GASTROENTEROLOGY & HEPATOLOGY	17	1 1 2—I 3 4 *4*	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 1 2—I 3 4 *7*	1 0 2 3 4 *1*
GERIATRIC MEDICINE	18	1 0 2 3 4 *6*	1 1 2 3—I 4 *11*	1 1 2—I 3 4 *3*	1 0 2 3 4 *3*	1 0 2 3 4 *1*	1 2 2 3—II 4 *2*	1 0 2 3 4 *2*
HAEM/HAEM ONCOLOGY	3	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 1 2—I 3 4 *6*	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	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 *1*	1 0 2 3 4 *0*
INTERNAL MEDICINE	141	1 8 2—III 3—III 4—II *34*	1 5 2 3—IV 4—I *15*	1 0 2 3 4 *9*	1 0 2 3 4 *3*	1 1 2 3 4—I *6*	1 23 2—V 3—X 4—VIII *47*	1 0 2 3 4 *9*
MEDICAL ONCOLOGY	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 *2*	1 0 2 3 4 *0*
NEPHROLOGY	17	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 0 2 3 4 *0*	1 2 2 3—II 4 *5*	1 0 2 3 4 *3*
NEUROLOGY	15	1 1 2—I 3 4 *5*	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 3 2—I 3—I 4—I *5*	1 0 2 3 4 *0*
PALLIATIVE MEDICINE	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 1 2 3 4—I *1*	1 0 2 3 4 *0*	1 0 2 3 4 *0*
REHABILITATION	2	1 0 2 3 4 *0*	1 0 2 3 4 *2*	1 1 2—I 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 *5*
RESPIRATORY MEDICINE	11	1 0 2 3 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 3—I 4 *6*	1 0 2 3 4 *0*
RHEUMATOLOGY	9	1 0 2 3 4 *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 3—I 4 *4*	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	15	1 0 2 3 4 ***0***	1 2 2—I 3—I 4 ***10***	1 0 2 3 4 ***0***	1 2 2—I 3—I 4 ***1***	1 2 2 3—I 4—I ***4***	1 1 2 3 4—I ***1***	1 1 2—I 3 4 ***3***	1 1 2 3—I 4 ***0***	1 1 2—I 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	5	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***	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	8	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	9	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 ***4***	1 1 2 3—I 4 ***2***	1 0 2 3 4 ***4***	1 0 2 3 4 ***1***	1 0 2 3 4 ***4***	1 0 2 3 4 ***0***	1 2 2 3 4—II ***1***	
GASTROENTEROLOGY & HEPATOLOGY	17	1 0 2 3 4 ***0***	1 2 2—II 3 4 ***5***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***1***	1 1 2 3—I 4 ***2***	1 1 2—I 3 4 ***3***	1 1 2 3 4—I ***6***	1 0 2 3 4 ***1***	1 2 2 3 4—II ***4***	1 0 2 3 4 ***0***	1 1 2 3 4—I ***5***	
GERIATRIC MEDICINE	18	1 1 2 3 4—I ***4***	1 1 2—I 3 4 ***1***	1 1 2—I 3 4 ***3***	1 1 2—I 3 4 ***2***	1 1 2 3—I 4 ***8***	1 1 2 3 4—I ***7***	1 1 2 3 4—I ***9***	1 0 2 3 4 ***1***	1 1 2—I 3 4 ***14***	1 0 2 3 4 ***3***	1 1 2—I 3 4 ***5***	
HAEM/HAEM ONCOLOGY	3	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 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 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	1	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 ***1***	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***	1 0 2 3 4 ***0***	
INTERNAL MEDICINE	141	1 0 2 3 4 ***3***	1 ***15*** 2-VI 3-VI 4-III ***47***	1 1 2 3—I 4 ***3***	1 4 2—I 3—III 4 ***18***	1 ***9*** 2—III 3—III 4—III ***35***	1 ***8*** 2—I 3—I 4—VI ***25***	1 ***3*** 2—I 3—I 4—I ***38***	1 1 2 3—I 4 ***3***	1 ***11*** 2—V 3—III 4—III ***42***	1 0 2 3 4 ***2***	1 ***7*** 2—I 3—I 4—V ***19***	
MEDICAL ONCOLOGY	2	1 0 2 3 4 ***0***	1 0 2 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 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	17	1 0 2 3 4 ***0***	1 2 2—I 3—I 4 ***7***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 1 2—I 3 4 ***2***	1 1 2 3 4—I ***2***	1 1 2 3—I 4 ***6***	1 0 2 3 4 ***0***	1 1 2—I 3 4 ***5***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	
NEUROLOGY	15	1 0 2 3 4 ***0***	1 2 2—I 3—I 4 ***6***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***2***	1 2 2—II 3 4 ***2***	1 0 2 3 4 ***1***	1 0 2 3 4 ***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***	
PALLIATIVE MEDICINE	4	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***3***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***	1 1 2 3 4—I ***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	2	1 1 2 3 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 ***2***	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	11	1 0 2 3 4 ***5***	1 0 2 3 4 ***6***	1 0 2 3 4 ***3***	1 1 2—I 3 4 ***2***	1 1 2 3 4—I ***4***	1 1 2 3 4—I ***2***	1 0 2 3 4 ***2***	1 0 2 3 4 ***0***	1 2 2—I 3—I 4 ***4***	1 0 2 3 4 ***2***	1 1 2 3 4—I ***1***	
RHEUMATOLOGY	9	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***2***	1 0 2 3 4 ***0***	1 0 2 3 4 ***2***	1 1 2 3 4—I ***3***	1 0 2 3 4 ***1***	1 1 2 3 4—I ***2***	1 0 2 3 4 ***0***	1 2 2—I 3—I 4 ***2***	1 0 2 3 4 ***0***	1 1 2 3—I 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	15	1 1 2 3—I 4 ***2***	1 0 2 3 4 ***3***	1 1 2—I 3 4 ***8***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***0***	1 0 2 3 4 ***1***	1 0 2 3 4 ***3***
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	5	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 3—I 4 ***4***
DERMATOLOGY & VENEREOLOGY	8	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	9	1 0 2 3 4 ***1***	1 0 2 3 4 ***3***	1 2 2—I 3 4—I ***8***	1 1 2 3—I 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***1***
GASTROENTEROLOGY & HEPATOLOGY	17	1 1 2 3 4—I ***1***	1 1 2 3—I 4 ***2***	1 1 2 3—I 4 ***4***	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***	1 2 2 3—I 4—I ***4***
GERIATRIC MEDICINE	18	1 0 2 3 4 ***1***	1 0 2 3 4 ***1***	1 0 2 3 4 ***4***	1 1 2 3—I 4 ***7***	1 1 2 3—I 4 ***3***	1 1 2 3 4—I ***2***	1 2 2 3—II 4 ***7***
HAEM/HAEM ONCOLOGY	3	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 0 2 3 4 ***3***
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	1	1 0 2 3 4 ***2***	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 ***2***
INTERNAL MEDICINE	141	1 5 2—I 3—II 4—II ***10***	1 2 2 3—II 4 ***15***	1 11 2—III 3—IV 4—IV ***50***	1 3 2 3—II 4—I ***6***	1 4 2 3—III 4—I ***6***	1 2 2 3 4—II ***12***	1 15 2—V 3—V 4—V ***38***
MEDICAL ONCOLOGY	2	1 0 2 3 4 ***0***	1 0 2 3 4 ***0***	1 2 2 3—II 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	17	1 1 2—I 3 4 ***1***	1 0 2 3 4 ***1***	1 3 2—I 3—II 4 ***5***	1 0 2 3 4 ***0***	1 1 2 3—I 4 ***1***	1 0 2 3 4 ***0***	1 3 2—II 3—I 4 ***5***
NEUROLOGY	15	1 1 2 3 4—I ***1***	1 1 2 3—I 4 ***1***	1 1 2 3—I 4 ***5***	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***
PALLIATIVE MEDICINE	4	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	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 ***1***	1 0 2 3 4 ***0***	1 0 2 3 4 ***3***
RESPIRATORY MEDICINE	11	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 3 2—I 3 4—II ***2***
RHEUMATOLOGY	9	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 1 2 3 4—I ***2***	1 0 2 3 4 ***0***	1 1 2—I 3 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	8	1 6 2—I 3—IV 4—I ***9***
INTERNAL MEDICINE	141	1 2 2 3—I 4—I ***0***
INFECTIOUS DISEASE	1	1 0 2 3 4 ***2***
RESPIRATORY MEDICINE	11	1 0 2 3 4 ***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 July 2013

		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	236	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	84	12	1	0	**13**	0	0	10	0	**10**	**23**
DERMATOLOGY & VENEREOLOGY	96	0	0	0	**0**	0	0	1	0	**1**	**1**
ENDOCRINOLOGY, DIABETES & METABOLISM	96	4	2	3	**9**	0	0	9	0	**9**	**18**
GASTROENTEROLOGY & HEPATOLOGY	167	6	2	1	**9**	0	0	11	1	**12**	**21**
GERIATRIC MEDICINE	177	6	12	3	**21**	3	2	4	2	**11**	**32**
HAEM/HAEM ONCOLOGY	56	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	1223	54	25	12	**91**	4	13	94	11	**122**	**213**
MEDICAL ONCOLOGY	41	0	0	0	**0**	0	0	9	0	**9**	**9**
NEPHROLOGY	121	6	1	0	**7**	0	0	7	3	**10**	**17**
NEUROLOGY	102	6	3	0	**9**	1	0	7	2	**10**	**19**
PALLIATIVE MEDICINE	22	0	2	0	**2**	0	2	1	0	**3**	**5**
REHABILITATION	53	0	3	3	**6**	2	0	1	5	**8**	**14**
RESPIRATORY MEDICINE	177	11	6	2	**19**	0	8	11	0	**19**	**38**
RHEUMATOLOGY	70	4	2	1	**7**	8	0	0	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	236	0	17	**17**	0	2	7	**9**	2	6	1	10	0	6	**25**	**51**
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	**0**	0	0	0	**0**	0	0	0	0	0	0	**0**	**0**
CRITICAL CARE MEDICINE	84	0	6	**6**	0	4	6	**10**	5	5	0	7	0	0	**17**	**33**
DERMATOLOGY & VENEREOLOGY	96	0	0	**0**	0	0	0	**0**	0	0	0	0	0	0	**0**	**0**
ENDOCRINOLOGY, DIABETES & METABOLISM	96	0	8	**8**	0	4	5	**9**	2	5	2	5	0	1	**15**	**32**
GASTROENTEROLOGY & HEPATOLOGY	167	0	8	**8**	0	3	3	**6**	4	9	1	7	0	8	**29**	**43**
GERIATRIC MEDICINE	177	5	4	**9**	3	3	13	**19**	7	10	2	15	5	5	**44**	**72**
HAEM/HAEM ONCOLOGY	56	0	8	**8**	0	2	2	**4**	0	0	0	5	0	0	**5**	**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	1223	8	74	**82**	6	25	51	**82**	34	50	8	66	6	26	**190**	**354**
MEDICAL ONCOLOGY	41	0	2	**2**	0	0	1	**1**	0	0	0	1	0	0	**1**	**4**
NEPHROLOGY	121	0	8	**8**	2	2	3	**7**	2	8	0	9	0	2	**21**	**36**
NEUROLOGY	102	1	9	**10**	0	2	4	**6**	2	5	1	2	1	2	**13**	**29**
PALLIATIVE MEDICINE	22	0	0	**0**	4	0	2	**6**	4	0	1	0	1	0	**6**	**12**
REHABILITATION	53	9	0	**9**	2	0	3	**5**	1	1	1	2	4	0	**9**	**23**
RESPIRATORY MEDICINE	177	6	7	**13**	6	4	7	**17**	7	6	0	5	3	1	**22**	**52**
RHEUMATOLOGY	70	2	4	**6**	0	2	3	**5**	3	3	0	3	0	2	**11**	**22**

		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	236	3	5	15	0	0	**23**	3	8	**11**	**34**
CLINICAL PHARMACOLOGY & THERAPEUTICS	8	0	0	5	0	0	**5**	0	0	**0**	**5**
CRITICAL CARE MEDICINE	84	3	4	1	0	0	**8**	0	6	**6**	**14**
DERMATOLOGY & VENEREOLOGY	96	0	0	3	0	0	**3**	0	0	**0**	**3**
ENDOCRINOLOGY, DIABETES & METABOLISM	96	1	5	14	0	0	**20**	0	3	**3**	**23**
GASTROENTEROLOGY & HEPATOLOGY	167	1	5	7	0	0	**13**	4	10	**14**	**27**
GERIATRIC MEDICINE	177	1	2	6	9	3	**21**	4	10	**14**	**35**
HAEM/HAEM ONCOLOGY	56	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	1223	20	29	79	12	9	**149**	18	64	**82**	**231**
MEDICAL ONCOLOGY	41	0	0	15	0	0	**15**	0	0	**0**	**15**
NEPHROLOGY	121	5	1	8	0	1	**15**	2	7	9	**24**
NEUROLOGY	102	1	1	10	2	0	**14**	1	5	**6**	**20**
PALLIATIVE MEDICINE	22	0	0	0	2	0	**2**	0	1	**1**	**3**
REHABILITATION	53	0	1	2	1	1	**5**	0	3	**3**	**8**
RESPIRATORY MEDICINE	177	3	8	8	0	3	**22**	4	6	**10**	**32**
RHEUMATOLOGY	70	3	0	5	0	2	**10**	2	3	**5**	**15**

Professor Vivian Nap Yee CHAN

John MacKay

It is particularly appropriate that this Profile of Professor Vivian Chan should take place this year, sixty years after the discovery by Francis Crick and James D Watson of the structure of DNA in 1953, and ten years after the completion of the Human Genome Project.

Vivian Chan was born in Hong Kong to a non-medical family, her father a businessman and her mother a teacher. She went to school at the prestigious Sacred Heart Canossian College for girls, until she left for England to prepare for the GCSE examination at the famously academic St Saviour & St Olave Grammar School. Her elder brother and younger sister were also at school in England. Life was not all study; in the winters she enjoyed playing squash, and in the summers swimming; year round she enjoyed horse riding.

Her first degree was a B.Sc. Hons. earned at London University in 1968, followed by M.Sc. and DIC at Imperial College, London a year later. She then joined the Chemical Pathology Department of St. Bartholomew's (Barts) Hospital, London as a Clinical Biochemist, whilst working for her Ph.D., which she obtained in 1973 based on a thesis on in-vitro thyroid function tests. Her final year at St. Bartholomew's was as a postdoctoral researcher on a Medical Research Council Award. During these years she authored or co-authored 15 papers in peer-reviewed journals on Thyroid function and physiology.

In 2000 Professor Todd wrote of Dr Chan's early years: "Her initial research in endocrinology with Professors J Landon and GM Besser in London, and Professors Rosie TT Young and Christina Wang in Hong Kong, has contributed significantly to the management of patients with thyroid and pituitary disorders."

In 1974 Dr Chan made the decision to return to Hong Kong. It was not an easy one. She had been approached by Dr Chan Woon Cheung, then Reader in the Dept of Pathology, HKU, and later by Professor Rosie Young when she came for a visit to the Dept of Chemical Pathology at Barts, and told her of the advertisement for a non-clinical biochemist in the Department of Medicine at Hong Kong University.

On the one hand she was doing well with her research at Barts, making presentations at international and national meetings every month, and had been offered a promotion to Senior Grade Biochemist. Her brother and sister were still in UK doing accountancy exams. On the other hand her parents were still living in Hong Kong, although they never pressed her to return. Also, Hong Kong University was keen to have her. "I think I felt I have been away for a while already and wished to explore living in Hong Kong as an adult."

In the end she went for an interview with two officials at the Commonwealth Office, and was interviewed again by Professor A J S McFadzean while she was on holiday in Hong Kong. Her appointment was confirmed on expat terms, but by the time she started work as a lecturer in non-clinical biochemistry in Sept 1974, Professor McFadzean had retired and Professor David Todd had become the head of in the Department of Medicine at Hong Kong University.

It took her 18 months to feel part of the community in her department, most of her colleagues having graduated from Hong Kong University. "However, after a couple of years, Hong Kong sort of gets under one's skin, it was then difficult to leave and return to UK."

In 1978 she was appointed a Senior Lecturer and received a China Medical Board Fellowship to study for a year at the National Institute of Child Health and Development, Endocrinology and Reproductive Research Branch, under Dr Kevin J Catt (a world renowned endocrinologist) in Bethesda, USA. On her return to Hong Kong she focussed more on haematology: Genetic disease, diagnostic

techniques, microarrays for diagnosis of thalassemia, Hepatitis B, and even lung cancer by blood test.

In 1982 she received a Wu Chung Travelling Fellowship to study in California with Prof. Y W Kan (the Hong Kong University graduate who was the discoverer in 1975 that a severe form of thalassaemia, was due to the lack of the gene encoding the alpha chain of the haemoglobin molecule. This was the first time a gene deletion was recognized as a cause of human disease). She counted herself fortunate to have learnt DNA technology from one of the pioneers. On her return she established the DNA-based Prenatal Diagnosis Programme for common genetic diseases in Hong Kong (a first in the Asian Pacific region). She was appointed as Reader in the Department of Medicine. Since then she has returned to Professor Kan's laboratory for further short periods of research.

In 1988 there were two noteable events in her life, she became a Fellow of the American College of Biochemists, and more importantly, she married Professor T K Chan.

She was appointed in 1993 a Full Professor of Medicine, with the Personal Chair of Molecular Medicine. In 1995 she became Deputy Director, and latterly, Acting Director of the

Institute of Molecular Biology at the University of Hong Kong. She enjoyed teaching, but would like to have seen more medical undergraduate interest in scientific research. She feels that students are too focussed on studying purely to get through their exams.

In 1995 she was awarded a Fellowship (by Publication) from the Royal College of Pathologists in UK.

For "exceptional contribution to the practice of Medicine ", she was elected Honorary Fellow of the Royal College of Physicians of London in 2001. This being the highest honour that the Royal College can bestow on a non-medically qualified person. Later that same year she was awarded an Honorary Fellowship of the Hong Kong College of Physicians. Part of the citation read, "Since its establishment in 1982, her unit, (the University DNA Diagnostic Service), remains undoubtedly the first and best prenatal diagnosis programme for the common genetic diseases in this region." and also," She has effectively taught Molecular Medicine to medical undergraduates, and trained medical doctors and scientists in DNA technology in China, South-East Asia as well as Hong Kong."

More recently the unit has been working on osteoporosis, hepatitis B virus (HBV) and lung cancer. The development of a comprehensive diagnostic array for mutation detection in HBV will have a great impact towards disease management (in allowing clinicians to make timely switch to second and third generation antiviral therapy). Likewise, the EGFR Array allows the detection of gene mutations in plasma DNA for monitoring lung cancer patients' response to therapy, obviating the need of repeated lung biopsy.

She was the first appointee, in 2008, to the endowed Chui Fook-Chuen Chair of Molecular Medicine.

Upon her retirement, she was Honorary Professor of the Dept of Medicine as well as the Department of Obstetrics and Gynaecology at Hong Kong University between 2010 -2012. Currently, she is Honorary Professor of the Dept of Obstetrics & Gynaecology, and Honorary Consultant Scientist, Wu Chung Prenatal Diagnosis Laboratory, Tsan Yuk Hospital, Hong Kong, a post she has held since 1983.

During her career Prof Chan has been a prolific publisher of 275 academic papers, a contributor to seven books, and has been an invited speaker at 49 international meetings and 32 local meetings. She has been a member of 19 University Committees, and 17 Professional Society Committees, and Reviewer for 14 Scientific Journals.

Professor Chan was a member of the Global Burden of Disease, in the Hemoglobinopathies Expert Group, a project jointly organised by the World Health Organisation and Harvard University; member of the Asian Thalassemia Network and member of Faculty for Haemophilia Control, World Federation of Haemophilia.

She was a member of the Senate of the University from 1993 to 2005.

During that time she was Project Chairman from 1995 to 2002, of the Committee on New Medical Complex Development, Faculty of Medicine. She is justly proud of her contribution in building the New Medical Complex, a state of the art facility, to benefit many generations of medical undergraduates.

Professor Chan remarks, "Looking back, I have always worked closely with clinicians, from my postgraduate days onwards. It meant that I do mostly translational, rather than basic research. I enjoyed seeing my efforts being applied readily to the bedside, rather than waiting for years before my findings can be linked to or used for further understanding of disease."

Professor Richard Y H Yu in 2005 gave a dissertation to the 10th Hong Kong Medical forum on the career of Professor A J S McFadzean. In this he said," In the mission objective of academic research, one of McFadzean's most far-reaching decisions was to recruit a scientist – a non-clinical lecturer – to organize and initiate research. Dr. (now Professor) Vivian Chan was interviewed by him in 1974. The Division of Molecular Medicine was thus born, laying a timely and solid foundation for the development of medical research and service in Hong Kong."

Professor Chan joined a department where research on Haematology was already a focus, thanks to the work of Professor Sir David Todd and others. She carried this on, keeping Hong Kong University at the forefront of world research and the application of that research to clinical uses. In this work she has had important co-workers, none more so than her husband Professor T K Chan.

Her son Derek looks as if he may follow in his parent's footsteps. He is on the MB/PhD programme at Cambridge University in England. He is now in the 2nd year of his PhD, upon completion of which he will return to the final two years of the MB course. He was only three when he accompanied his parents to his first international conference. Perhaps he will be a delegate again, to listen to his mother making her next presentation later this year at 13th International Conference on Thalassemia and Haemoglobinopathies in Abu Dhabi.