SCIENCE FOCUS 科言

Issue 006, 2015

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Itching to Know:
Why Do We Itch?
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Interviews with Nobel Laureates
Prof. Venkatraman Ramakrishnan
& Prof. William E. Moerner
諾貝爾得獎者
文卡特拉曼·拉馬克裡斯南教授及
威廉·莫爾拿教授 專訪

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SCIENCE AND TECHNOLOGY

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Message from the Editor-in-Chief 主編話語

Dear Readers,

Welcome back to another issue of Science Focus. We have scouted a variety of activities for you in this issue, eight more exciting science articles awaiting your perusal, and two profiles of world-renowned scientists and Nobel Laureates Prof. Venkatraman Ramakrishnan and Prof. William E. Moerner. I'm particularly excited about this issue as we ask important global questions, such as "Is the ozone layer recovering?" and "Can extinct animals be resurrected?" Find out the answers to these questions on pages 4 and 6.

In addition, I'd like to congratulate our second Science Focus Article Submission Competition winner, Miss Lee Lok Sze (Diocesan Girls' School) for her excellent and highly relevant article, "Why Do We Itch?" You can read her article on page 20. This time round, we have received various interesting articles on all sorts of different science topics, and enjoyed reading all of them. The competition is year round and ongoing. If you would like to participate, please send us your article to our website today!

Yours faithfully,

Prof. Yung Hou Wong
Editor-in-Chief

親愛的讀者：

歡迎瀏覽第六期「科言」。今期我們為你搜羅了更多活動資訊和八篇精彩有趣的科普文章，還有兩篇人物特寫，介紹世界著名科學家及諾貝爾獎得主：文卡特拉曼．拉馬克裡斯南教授及威廉．莫爾拿教授。今期內容特別令人振奮，報導了全球關注的問題，例如「臭氧層是否正在恢復？」、「滅絕的動物能否復活？」。若你也想知道答案，快看第 4 頁和 6 頁。

此外，我想借此機會恭喜第二屆「科言徵文比賽」的得獎者拔萃女書院李樂思同學，其得獎作品「我們為什麼會痕癢？」刊於第 20 頁。這屆比賽我們收到許多涵蓋不同科學主題的文章，趣味盎然，讓我們樂在其中。本年度徵文比賽現已接受投稿，歡迎你踴躍參與，立刻上載稿件到我們的網站。

祝你閱讀愉快！

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WHAT'S HAPPENING IN HONG KONG?

SCIENCE

香港科技活動

蘇韋霖
By Wai Lam Raphaella So

Hello students, and welcome to the new school year! As always, there are many fun and exciting events waiting for you around the corner.

InnoCarnival

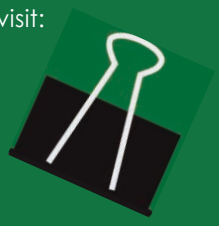
InnoCarnival is an event organised by the Hong Kong government and the Hong Kong Science Park in November. It consists of a wide range of events to showcase local scientific innovation to the general public, involving exhibitions, seminars, workshops, games, guided tours to different Science Park facilities, as well as science competitions and prize presentations. Major exhibitors include local universities, research bodies, award-winning student inventors, and the government. Admissions to all InnoCarnival activities are free, though some events require participants to pre-register online. To view the activities calendar and to register online, please visit: *http://www.itm.gov.hk/en/activityinnoc.php*.

The Jockey Club Museum of Climate Change

If you care about the environment and about the changing climate, a trip to the Museum of Climate Change at the Chinese University of Hong Kong is a must. There, you can enjoy both themed and permanent exhibitions, join eco tours of relevant facilities on campus, as well as attend workshops to learn about the natural environment and sustainability. Permanent exhibitions in the museum include the Polar Gallery, Remote Sensing and Environmental Monitoring, Research and Innovation at CUHK, and the Hong Kong Jockey Club Green Gallery. Themed exhibitions feature new discoveries and developments regarding different environment-related topics. The museum is open to the public on Mondays, Tuesdays, Thursdays to Saturdays, except for public and university holidays. Admission is free. For more information, please visit: *http://www.gaia.cuhk.edu.hk/*.

Hong Kong Youth Science and Technology Innovation Competition

Organised by the Hong Kong New Generation Cultural Association, The Hong Kong Youth Science and Technology Innovation Competition aims to arouse students' interests in science, technology, engineering and mathematics (STEM). Categories of the competition that are relevant to high school students are "Research and Invention", as well as "Student Organised Science and Technology Activity." Students may either submit a research or invention in any of the five subjects: Mathematics, Physics, and Engineering; Chemistry and Materials; Biology and Health; Energy and Environmental Science; and Computer Science and Information Technology for "Research and Invention". For the Student Organised Science and Technology Activity category, participants are expected to hold an educational activity that promotes science or technology to the public. The deadline for online applications is January 30, 2016, and the deadline for project submission is February 17, 2016. For more details, please visit: *http://stic.newgen.org.hk/*.



同學們好！新學年開始了，與往常一樣，有很多樂趣和精彩的活動在等著你。

創新科技嘉年華

「創新科技嘉年華」是由香港政府、香港青年協會與香港科學園在十一月舉辦的活動，透過豐富的節目向廣大市民展示本地的創新科技，內容包括展覽、講座、工作坊、遊戲、參觀科學園不同設施的導賞團、以及科技競賽及頒獎典禮。主要的參展機構包括本地大學、科研機構、獲獎學生發明家、以及政府部門等。「創新科技嘉年華」全部活動免費入場，部分節目要求參與者在網上預先登記。請到下列網址查看活動時間及報名參加：*http://www.itm.gov.hk/zh/activityinnoc.php*。

賽馬會氣候變化博物館

如果你關心環境以及氣候變化，絕對要逛一趟香港中文大學的賽馬會氣候變化博物館。在這裏，你可以觀看常設展覽和定期主辦的專題展覽，參加生態行遊覽校園相關設施，以及介紹自然環境和可持續性發展的工作坊。博物館的常設展覽包括極地廊、衛星遙感和環境監測、香港中文大學的創新研究、和香港賽馬會環保天地。專題展覽定期展出與環境相關的最新資訊和發展。除了公眾期及大學假期，博物館逢週一、二、四至六向公眾免費開放。欲知詳情，請瀏覽：*http://www.gaia.cuhk.edu.hk/*。

香港青少年科技創新大賽

香港新一代文化協會希望透過舉辦「香港青少年科技創新大賽」來培養同學對科學、科技、工程及數學(STEM)的興趣。適合高中生參加的項目包括「研究及發明」和「學會科技實踐活動」。同學若是參加「研究及發明」比賽項目，可以按以下組別遞交研究論文或發明品：數理工程、化學及材料、生物及健康、能源及環境科學、和電腦及資訊科技。「學會科技實踐活動」則要求參賽者舉辦向公眾推廣科技的教育性活動。網上報名將於2016年1月30日截止，同學必須在2016年2月17日或以前遞交參賽作品。欲知詳情，請瀏覽：*http://stic.newgen.org.hk/*。

GUIDE TO UNIVERSITY APPLICATIONS:

德國和法國留學指南

By Wing Chau 鄒穎妍

Educating over 170 Nobel Prize winners in a variety of disciplines, Germany and France have been unsurprisingly popular destinations for students to pursue tertiary educations.

HIGHER EDUCATION SYSTEM

The higher education systems in France and Germany mainly comprise of universities and vocational institutions. Universities typically offer bachelor degree programmes of 3 to 4 year durations under the European Credit Transfer System (ECTS). On the other hand, specialist vocational schools provide a broad range of subjects and theoretical teachings on the sciences and liberal arts, allowing students to obtain an all-rounded education.

APPLICATION REQUIREMENTS

Many German institutions offer international degree programmes that do not require German as a prerequisite, but most require students to show their secondary school-leaving certificates to qualify for undergraduate studies. Local students in Hong Kong would be required to apply with their HKDSE or IB Diploma academic results. Although there is no central admission system for German universities, applicants may still obtain information from the German Academic

曾經培育超過170位不同領域的諾貝爾獎得主，德國和法國一直是極為受歡迎的教育搖籃。

高等教育體系

法國和德國的高等教育系統主要是包括大學和專業學校(specialist vocational schools)。在歐洲學分轉換系統(European Credit Transfer System)下，大學一般提供三至四年學士學位課程。有別於傳統大學，專科學院則提供了廣泛文理科目的理論教學，致力讓學生學以致用。

申請要求

大部分德國大學提供國際學位課程，並不須申請人具備一定的德語能力，但會要求申請人展示其中學畢業證書，證明具備大學入學資格。本地學生須要以公開試成績報考，例如：香港中學文憑考試(HKDSE)或國際文憑大學預科課程(IB Diploma Programme)的成績。雖然德國大學並沒有統一收生制度，申請者仍可透過德國學術交流總署(DAAD)的入學資格數據庫或Anabin網站確定其本土大學入學資格在德國的認受性。如果申請人的入學資格不被認可，亦可接受約一年的基礎課程並通過大學入學評估獲取資格。

Exchange Service (DAAD) entrance qualification database to determine whether one's university entrance qualification is recognised in Germany or the specific university. Alternatively, a one-year foundation course accompanied with a university entrance assessment can be taken if other requirements are not met.

Applicants interested in French universities should apply through a French national education agency called Campus France. Users can create an account on the central online application system (CEF) which allows direct application and progress tracking of their Preliminary Admission (DAP) as well as their visa application.

VISA AND RESIDENCE PERMIT

Applicants are required to submit proof of financial resources, certificates of past academic work and achievements, certificate of health insurance, French or German language proficiency (as required by the institution), and proof of admission from the university.

Validity is typically limited to 90 days. Thus, if the applicant is not a citizen of an EU country, he/she must apply for a residence permit during his/her stay in France or Germany.

TUITION AND LIVING EXPENSES

On average, the tuition fees of universities in France vary from 150 EUR (1,300 HKD) to 900 EUR (7,900 HKD) per semester. German universities generally waive university tuition fees under the belief that everyone deserves an education regardless of financial capability. However, students will be asked to pay semester contributions, which command an average of 250 EUR (2,200 HKD) and cover a list of social expenses such as accommodation, school administrative support and computer facilities.

Average monthly living expenses for students studying in France and Germany total to approximately 800 EUR (7,000 HKD) for both countries, but varies depending on the city. Higher expenditure would be expected in larger cities such as Paris and Munich for accommodation.

另一方面，法國國家教育機構Campus France則為申請人提供一個平台，讓申請人可在網上報名系統CEF申請不同的大學和簽證，以及跟進申請進度。

簽證和居留許可

法國和德國的學生簽證申請者須提交財務證明、過往學術記錄和成就、醫療保險證明、(按機構要求)法語或德語語言水準證書、和大學錄取證明書。

學生簽證的有效期一般為九十天，倘若申請人並非任何歐盟國家的公民，並計劃留學超過三個月，就必須在德國或法國申請居留許可。

學費和生活費

法國大學每學期的學費平均由每學期150歐元(約1,300港元)至900歐元(約7,900港元)等。德國大學認為每個人擁有獲得教育的權利，通常會豁免所有大學生的學費，但學生每學期須繳付約250歐元(約2,200港元)(semester contribution)，以資助住宿、大學行政、電腦設施等方面的費用。

法國和德國平均每月的生活費約為800歐元(約7,000港元)，按城市而有所不同。在巴黎和慕尼克等較大的城市居住，將有更大支出。

For more detailed information, please visit:
欲了解更多資訊，請瀏覽：

Campus France (Hong Kong):
http://www.hongkong.campusfrance.org/en/

German Consulate General Hong Kong
德國駐香港總領事館：
http://www.hongkong.diplo.de/

Study in Germany
留學德國：
https://www.study-in.de/en/

The late 20th century saw an alarming increase in the size of the ozone hole, with its diameter spanning the entirety of Antarctica and beyond. In addition to the health risks for humans associated with an increase in ultraviolet ray penetration, an expanding ozone hole has significant ecological and meteorological impacts. In nature, ozone, oxygen gas, and free oxygen atoms are constantly being combined, broken apart, and recombined in the stratosphere. This process absorbs radiative energy from UV. Ozone depleting substances such as chlorofluorocarbons (CFCs), hydrochlorofluorocarbons (HCFCs), and hydrofluorocarbons act as catalysts speeding up the breakup of ozone to oxygen gas. This lack of recycling of ozone is the main cause of ozone depletion.

The ozone hole above Antarctica is particularly large due to the extremely cold temperatures. As a result, thin layers of ice crystals aggregate into polar stratospheric clouds, which act as catalysts exacerbating the effects of existing ozone-depleting compounds in the atmosphere, such as chlorine and bromine. An accumulation of these gases rapidly depletes the ozone layer in these coldest areas of the stratosphere.

CFCs have been slowly phased out since the beginning of the 80s under the global environmental treaty known as the Montreal Protocol. As a result, the stratospheric ozone layer may be making a slow recovery. In 2014, a group of geophysicists hypothesised that the ozone layer has the ability to mend itself, provided that the depletion rate is at a level much lower than the rate of natural stratospheric ozone production. Scientists forecast that the Antarctic ozone hole is expected

Did you know 你知道嗎?

The ozone is distributed among two layers of the atmosphere. 10% of atmospheric ozone lies within the troposphere, measuring approximately 15 km above sea level, and the remaining 90% resides in the stratosphere. The ozone layer acts as the primary UV radiation shield, reducing health risks and photo-oxidative damage brought to both terrestrial and aquatic ecosystems.

臭氧被分佈在對流層及平流層。對流層位於海拔約15公里，約10%的臭氧處於此。而90%臭氧分佈在平流層的底部。臭氧層是紫外線輻射屏蔽，有效減低對人類健康的影響及保護陸地和水生生態系統。

to reduce from 24.1 million km^2 today to less than 20 million km^2 in size by 2040 (the size of Antarctica is 14 million km^2 in comparison) [1], but it will not be until after the mid-21st century for the ozone layer to reach the state that it was in before the early 1980s [2].

Unfortunately for us, even this estimate may be too optimistic. In fact the trend of the ozone layer has not been a smooth sailing downward decline over the past decade. While it does seem that the hole is getting smaller, the largest sizes were recorded in 2006 and 2011. Scientists at NASA argue that climate change is to be blamed for the fluctuating patterns [3]. To further complicate matters, although the Montreal Protocol did its job in curbing ozone depleting chemicals, substantial amounts of these chemicals are still being detected during the past decade, indicating that illegal use still exists.

If left unattended, ozone depletion will likely cause higher rates of skin cancer, harm ecosystems, and threaten marine food chains. The good news is that the ozone hole does not seem to be getting any bigger, largely thanks to a concerted global effort. Only time will tell, if it will return to its pre-80s glory. For now, we should continue to strive as a global force to curb the use of ozone depleting chemicals.

Recovering the Ozone Layer

恢復臭氧層之路

By Thomas Lee 李浩賢

在二十世紀末，南極臭氧洞規模飆升，直徑橫跨整個南極。臭氧損耗不但增加紫外線輻射強度，造成人類健康風險、對生態和氣象的影響亦不容忽視。在自然界中，平流層的臭氧、氧氣和遊離氧原子不斷合併重組，吸收紫外線的輻射能量。破壞臭氧層的物質如氯氟烴 (CFC)、氫氯氟烴 (HCFC) 和氫氟等，加速臭氧解體為氧氣，減少可循環的臭氧，成為臭氧損耗的主要原因。

南極上空的臭氧洞極大，主要是因為極度寒冷的溫度。薄層冰晶因而聚集形成極地平流雲，加劇氯、溴等臭氧消耗化合物所造成的惡果。這些氣體的積累迅速消耗在平流層中的最冷區域的臭氧層。

自80年代開始氯氟烴已受全球環境條約「蒙特利爾議定書」約束而逐漸被淘汰。結果，平流層的臭氣層有緩慢恢復的跡象。在二零一四年，有地球物理學家假設，只要臭氧消耗率比天然平流層臭氧生產率低，那麼臭氧層便能自我修補。科學家預測，南極臭氧洞有望從現在的2,410萬平方公里，到二零四零年縮小至低於2,000萬平方公里 (南極洲面積約為1,400萬平方公里) [1]。科學家估計直到二十一世紀中後葉，南極臭氧洞規模才會回復至上世紀八十年代的大小 [2]。

可是，我們是否過於樂觀？過去十年，臭氧洞規模一直沒有平穩的降幅，甚至在二零零六和二零一一年，錄得最大的臭氧洞面積！美國航空航天局認為，最簡單的解釋是氣候變化 [3]。事實上，即使有「蒙特利爾議定書」規管，在過去十年仍能檢驗到大量破壞臭氧層的化合物，顯示有非法使用的情況。

如果漠視不理，強烈紫外光會持續穿透薄弱的臭氧層，增加人類患上皮膚癌的可能，亦會破壞生態系統和威脅食物鏈。幸好通過全球協力，南極臭氧洞沒有增大的趨勢。時間會告訴大家，臭氧層能否回復至八十年代前的狀況。目前我們應繼續遏止使用消耗臭氧層的化學物質。

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Resurrection Biology: Project De-Extinction

By Wing In Chau 鄒穎妍

The passenger pigeon once thrived in North America as the most abundant bird species until the early 20th century. Persistent poaching and destruction of their natural habitat via deforestation led to their eventual extinction, with the last of its known species to die in captivity by 1914. But almost a hundred years later, the passenger pigeon may be making a comeback.

Believe it or not, the concept of bringing the extinct back to life in science fiction movies such as Jurassic Park is not as far-fetched as one might think. Resurrecting dinosaurs is undoubtedly out of the question, but scientists may be able to revive animals that have not been extinct for that long, thanks to cutting-edge technology in genetic engineering.

Scientists at the University of California, Santa Cruz, have obtained genetic data from stuffed passenger pigeons and its closest relative, the band-tailed pigeon, the latter of which will serve as a DNA blueprint to the incomplete and contaminated DNA of a bird that died more than a century ago. Modifying the DNA of the genetic cousin to match that of the passenger pigeon, injecting the cell into another pigeon's egg and then hoping this cell will develop into a young pigeon is probably their best bet. The most difficult and time-consuming part of this endeavour is genome mapping – while the two species are closely related, their genomes still differ at possibly millions of locations and these need to be determined [1]. If all goes according to plan though, we may be seeing a chimeric version of the passenger pigeon once again in the near future.

In an even more ambitious project, some scientists have been working on bringing perhaps the most iconic extinct animal back to life – the woolly mammoth – four thousand years after they disappeared off the face of the Earth. Also using genome sequencing, scientists have been able to figure out what makes a mammoth a mammoth, as opposed to an elephant for instance. It is with hope that using this information, alleles in the elephant DNA can be replaced by those that express mammoth-like features, such as their large tusks or characteristic furry bodies, hence bringing a woolly mammoth back from the dead.

If this were possible, a whole host of ethical questions accompany the challenges of de-extinction. If de-extinction succeeded and a mammoth were to be created through a surrogate elephant mother, it would most likely be brought up in captivity, a non-ideal way to bring up a social animal (if they are anything like elephants), while being the only mammoth alive. In

復活生物：逆滅絕計劃

De-extinction – not to be confused with cloning

Cloning is a process that requires cells to be transferred from a living specimen. De-extinction, on the other hand, involves editing the DNA of existing animals that are genetically similar to the extinct animal, which results in a 'chimera'.

不要將「逆滅絕」(de-extinction)與「克隆」(cloning)混為一談

「克隆」過程中會從仍存活的實驗體抽取細胞；而「逆滅絕」是通過改造與滅絕物種相近的現存物種的基因，產生嵌合物種。

在二十世紀初，旅鴿曾經是北美地區最繁盛的鳥類。由於人類大肆捕獵，加上大規模伐林，令其喪失棲息地，使其數目持續減少。直到1914年，最後一隻旅鴿死於籠檻中，旅鴿正式宣告滅絕。但百年後的今日，旅鴿或許能夠重現於世。

addition, it could be argued that elephants themselves are already somewhat endangered and should not be subjected to scientific projects that may or may not succeed.

On the other hand, scientists like Dr. Sergey Zimov argue that there are ecological benefits to re-introducing animals such as mammoths into the environment. For instance, the tundra used to be home to mammoths and an abundance of other grazing creatures thousands of years ago, rich with grassy steppes instead of covered with icy moss. Dr. Zimov insists that these animals were responsible for maintaining the landscape through grazing and fertilisation. The grasslands could potentially protect the inevitable melting of the tundra's permafrost, helping to slow down the release of centuries-trapped greenhouse gases, such as carbon dioxide, that rests beneath the surface.

Regardless of the ethical implications of de-extinction, what is undeniable is that de-extinction is on the forefront of technology and an exciting concept. Will we see passenger pigeons once again repopulating the skies or mammoths grazing in Siberia in the near future? Given the difficulties scientists face both technically and ethically, it will no doubt take some time - but who knows what the future has in store.

信不信由你，讓滅絕物種重現未必只是科幻電影的情節。當然，如電影侏羅紀公園(Jurassic Park)中復活恐龍的構思仍是不可能的，但隨著基因工程(genetic engineering)的急速發展，科學家們也許能夠重現滅絕不久的動物。

美國加州聖克魯斯大學(University of California, Santa Cruz)的科學家，從旅鴿的標本和牠的近親—帶尾鴿(band-tailed pigeon)，抽取基因組織。帶尾鴿的基因可作為脫氧核糖核酸(deoxyribonucleic acid, DNA)藍圖，以補足百多年前死去的旅鴿所留下的不完整和受污染的DNA。科學家打算按旅鴿DNA序列修改帶尾鴿的基因，然後將細胞注射到另一個鴿子蛋，希望細胞可以成長為鴿子。雖然兩個物種十分相近，牠們的基因圖譜(genomes)可能有幾百萬不同處，有待鑒定。所以製作基因組圖譜(genome mapping)是這項目中最困難和最耗時的[1]。如果一切按計劃完成，我們或許在不久的將來可以再次看見嵌合旅鴿。

一些科學家有一個更大膽的計劃，將四千年前滅絕的品種—長毛象帶回世間。透過基因組測序(genome sequencing)，科學家能夠找出當中令長毛象成為長毛象而非大象的基因。他們期望利用這些結果，以帶有長毛象特徵的對偶基因(alleles)取代大象的對偶基因，使其展現巨齒和毛茸茸的身體等。

逆滅絕計劃的成功將會帶來一籃子的道德問題。倘若科學家能以大象代母孕育出一隻長毛象，終其一生牠都會在籠檻中生活。作為獨一無二的長毛象，形單隻影，這是對象這類群體動物(social animals)而言，最殘酷的折磨。加上，大象亦是瀕危物種，無論這項科研能否成功，都不應將其牽扯入其中。

另一邊廂，部份科學家如謝爾蓋茲莫夫博士(Dr. Sergey Zimov)則認為讓已滅絕的動物復活，可改善現時的生態環境。幾千年前，苔原曾經是長毛象和許多其它食草動物的家園，芳草茂盛，而不是覆蓋著冰冷的青苔地衣。茲莫夫博士堅信，這些動物通過吃草和施肥保持了草原風貌，可能間接揭制平原的永久凍土層的融化，有助於減緩釋放困於地下多年的溫室氣體，如二氧化碳等。

儘管逆滅絕充滿著道德爭議，它仍然是令人振奮和前衛的科技。人類可否再次看見大群旅鴿在空中飛翔，或長毛象群在西伯利亞行走？鑑於現時科學家遇上的道德爭議和技術困難，肯定還要再等待一段時間，答案才會揭曉。

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Real-life INVISIBILITY Cloak

By Marco Wong 黃俊銘

This article may be useful as supplementary reading for physics classes, based on the DSE syllabus. 根據物理科文憑試課程剛要，本文或可作為有用的補充讀物。

If only being invisible was as easy as hiding under a nifty cloaking device bequeathed from your father who happened to dabble in wizarding mischief in his younger days. Unfortunately, us 'muggles' are just not blessed with the magical gene, but the good news is that we can strive to understand and invent technology inches away from the supernatural, thanks to a little thing called science. In fact, invisibility has been researched for quite some time now, with several inventions coming incredibly close to fruition.

What we currently understand is that an object appears to be 'invisible' when light is able to pass through the space that the object in question occupies, at least in the perspective of the observer. However, most objects are opaque, which block light from penetrating through. To circumvent this, one way would be to bend light with the aid of 'smart' metamaterials, which are able to marginally alter the path of light.

In 2006, Duke University researchers successfully hid an object from microwave detection by using a material that can bend electrons. Composed of copper and fibre glass, the material has a unique interaction against the planar electromagnetic field. The device was specifically tuned to minimise distortion of microwaves caused by the object, creating interference patterns which cancel each other out [1]. Fast forwarding six years, Korean researchers at Yonsei University created a 10 mm thick elastic cloak with the claim that any haphazardly shaped object can be hidden from broadband microwave detection, bringing the device closer to practical application. These inventions are the initial step to concealing objects in the visible light spectrum, as visible light has a much shorter wavelength in comparison, thus exhibiting a smaller degree of refraction [2].

To cloak an object under visible light, refraction is still utilised but on a much larger scale. In 2014, researchers at the University of Rochester constructed a device made of four lenses, the first of which can boast successful cloaking of small objects in three-dimensional space. The technology is surprisingly straightforward - two diverging lenses are arranged between two converging lenses, whereby light is concentrated as a thin beam between the diverging lenses, creating a doughnut shaped region of invisibility. Sounds fine and dandy, but the biggest limitation of this device is that it only works with small angles [3].

Researchers worldwide are now more focused on creating solid state metamaterials for the purpose of cloaking. In 2014, a research team from Karlsruhe Institute of Technology (KIT) in Germany constructed a diffusive optical cloaking device using common materials that hides objects placed within it [4]. The idea behind it is to allow light to enter through the material and guide it around the object, and then ultimately re-establishing the original path of the light before disturbance. Instead of using refraction, this 'cloak' adopts light scattering. With the use of a medium, the propagation of light is slowed down until it reaches a cylinder containing the object to be hidden. Coated with titanium dioxide doped polydimethylsiloxane, the material is then able to scatter light and speed

現實中的隱形斗篷

up the propagation to compensate for the discrepancy in distance. Normally, an object shone with light would cast a shadow in the backdrop, but the device demonstrated that no shadow was cast; showing that light had successfully been guided 'through' the object.

While these devices are still a long way away from hiding everyday objects, or people for that matter, the concept of invisibility has advanced from hiding microscopic objects in very specific conditions to hiding normal objects (provided that they fit) outside of laboratory settings. The KIT team has hopes that their device can be developed to blur out burglar sensors and enhance home security. All of this with no spells involved!

使人隱去身影的隱形斗篷，是魔幻故事中最受歡迎的道具。雖然現實生活中沒有讓人隱身的咒語，更沒有隱形魔法斗篷，我們仍可以運用科學達到這目的。隱形技術的研究其實已進行多年，而且已有接近成功的發明。

要達到隱形的效果，光線就要通過物件所佔用的空間。不過不透光的物件比比皆是，於是科學家需要改變光線的路徑，造成光線穿過了物件的假象。其中一個方法是運用超常材料(metamaterial)，局部改變光的路徑。

於2006年，杜克大學(Duke University)的研究人員利用可以改變電子路徑的物料，讓測試物成功避過微波(microwave)探測。該物料是由銅和纖維玻璃組成，可與平面電磁場(electromagnetic field)發生獨特的相互作用。他們的設計將測試物對微波的干擾降至最低，產生相互抵消的干涉圖案[1]。六年之後，韓國延世大學(Yonsei University)以類近原理製作了10毫米厚的彈性斗篷，聲稱可以使任何形狀的物件不被寬頻微波偵測發現。然而，這些發明只能視作初步成果。可見光波長極短，相對於微波較難被折射[2]。

於是，有人造出了更大型的折射裝置。在2014年，美國羅賈斯特大學(University of Rochester)的研究人員使用四面鏡片製造了一個光學隱形裝置，首次成功讓小物件在三維空間中隱形。這設備極為簡單，研究人員於兩個凸透鏡之間設置兩個凹透鏡，讓兩個凹透鏡之間的光線集中為細光束，形成一個無光線範圍，即不可見區域。但此設備的有效角度十分狹窄，並不能提供全方位隱形效果[3]。

現在科學家們比較鍾情開發固態超常材料。2014年7月，德國卡爾斯魯厄理工學院(Karlsruhe Institute of Technology)研發了漫射性(diffusive)光學隱形裝置，可隱去置於其中的任何物件[4]。其背後理念是讓光在物料中被引導繞過目標物，最終還原至被引導前的路徑。概念相似，只是漫射取代了折射。這漫射性的介質會減慢光線速度，直至光線到達存放目標物的金屬管。該管塗有摻雜聚二甲基矽氧烷的二氧化鈦，使光的擴散比在介質中更快，因此光線會圍繞金屬管，而且「加速」彌補了繞道的時間差。物件在漫射媒介中被照射，正常情況必定會留下影子，不過在這個裝置中的物件卻沒有，因此證明光線已被成功引導"穿越"目標物。

雖然要很久之後才能以這類裝置收藏一般物件或人，隱形的概念已經從在特定的條件下隱藏微小物件，進展到可以在實驗室以外隱藏一般(符合體積之限制)物件。KIT研究人員希望這項發明能夠用於掩護防盜感應器，加強家居安全。順帶一提，以上一切都沒有使用魔法。

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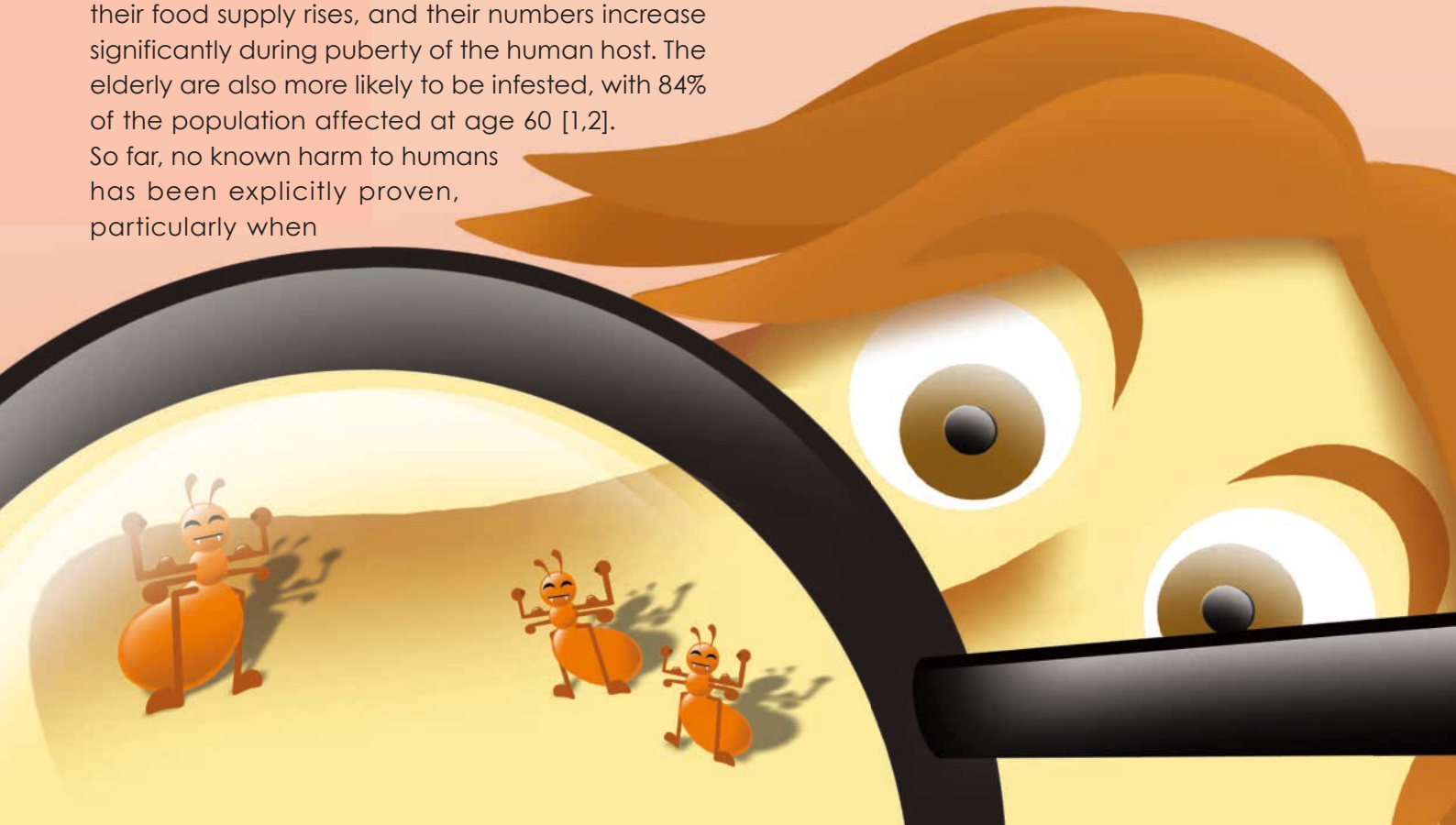
When faced with the fact that there are ten times more microorganisms than human cells in your body, "bacteria" is perhaps the first thing that comes to mind. However, we are also hosts to a wide variety of microscopic mites. Mites of the genus *Demodex* (Demodecidae) were first discovered in 1842 in human earwax; but we now know that there are two species of mites that dwell on our faces: *Demodex folliculorum* and *D. brevis*, with an average population density of around two mites per eyelash. In 2014, it was found in a study that every single human subject studied tested positive for *Demodex* DNA. That's right. They are likely universal human associates.

Scientists have yet to determine what role these mites play on the microbiota associated with our face. Whether they consume dead skin cells or facial sebum is part of the mystery as well, and examining the mite's guts might provide insights to their diet. More disturbingly, however, is the fact that these mites have no way of expelling waste. Instead, they 'hold it in' throughout their lifespan of 18-24 days. Upon death, their bodies dry out on the face, leaving behind the built-up waste, and an expulsion of bacteria.

Before you freak out and make a beeline for a chemical peel, rest assured that these mites are very common among humans and can appear shortly after birth. Mite population increases as their food supply rises, and their numbers increase significantly during puberty of the human host. The elderly are also more likely to be infested, with 84% of the population affected at age 60 [1,2]. So far, no known harm to humans has been explicitly proven, particularly when mite populations are in check. Nonetheless, people who suffer from the skin condition rosacea have reportedly ten times more *Demodex* mites on their skin than an average person. Additionally, an infestation of *Demodex* is associated with blepharitis, an unsightly inflammation of the eyelids [2]. However, the direct causation of these afflictions is difficult to prove.

These mites may have been acquired long ago, starting from our hominid ancestors. In one study, mites collected from Chinese populations were found to have distinct genes from those collected from North and South American populations [3]. Understanding these differences can provide clues to the migration patterns of our ancestors, revealing early interactions between human populations. Due to its lengthy history in association with humans, *Demodex* may shed light on the evolution of the human immune system, which helps us shape our response to diseases.

If you are still disgusted by these mites despite the potential benefits of studying them, you might be glad to know that there are therapies to get rid of *Demodex*. Unfortunately, the sad news is that you can never fully be rid of them as they will eventually recolonise your face after about six weeks. They are easily picked up from other people in close contact or from sheets, pillows and towels. It looks like face mites are here to stay!



居住在臉上的蠕蟲

Face Dwelling Mites

By Jacqueline Aw 歐婷梅

如果告訴你身體上的微生物比細胞還要多十倍，你也許首先會想到細菌。很少人知道，我們其實還是一些微型蠕蟲的宿主。微型蠕屬蠕形蠕 *Demodex* (Demodecidae) 是在1842年首次被發現在人類的耳垢中。現時知道生活在我們臉上的蠕蟲有兩個品種: *Demodex foliculorum* 和 *D. brevix*，平均密度約為每睫毛兩隻。在2014年進行的測試，每位參與者都被驗出帶有蠕形蠕 DNA，顯示它們已成為人類的夥伴。

科學家仍試圖確定這些蠕在微生態系統，也就是我們的臉上的角色。這些蠕是否消耗死皮或是皮脂，仍屬未知；科學家嘗試從這些蠕類腸道尋找線索。另一個令人略會不安的事實是，由於這些蠕蟲無法排出廢物，在牠們的一生18至24天內都會‘憋著’。牠們死了之後，身體就會在你臉上乾掉，將其一生所積累下來的廢物全部留下，更不用提釋放出來的細菌。

在你抓狂地大力洗臉之前，請放心，這些蠕蟲在人體內很常見，出生後即很快出現。蠕蟲蟲群會隨著食物供應上升而擴大，在人類青春期更是顯著增加。年齡60歲以上的長者更有84%受蠕蟲的影響[1,2]。到目前為止，並未有證據顯示蠕蟲會對人體造成危害，特別是當蠕蟲蟲群受控。儘管如此，酒槽鼻患者皮膚上的蠕形蠕數目據報比平均多10倍；此外，蠕蟲亦與不堪入目的眼臉緣炎有關[2]。不過，很難證明蠕蟲與這些疾患的直接因果關係。

科學家推測，在很久以前，我們的原始人祖先已與蠕蟲為伍。一項研究發現，在中國人和南、北美洲人的樣本中找到的蠕有顯著的基因差異[3]。研究這些差異可以為探索我們始祖的遷徙模式提供線索，從而揭示現代人口之間早期的關聯。在與人類共處的悠長歷史中，蠕形蠕可能也參與在人類免疫系統的進化中，從而影響我們對疾病的反應。

如果這些蠕的研究價值未能動搖你想擺脫牠們的決心，那麼好消息是有療法能殺死蠕形蠕。然而，不幸的消息是你不能永遠完全擺脫牠們。我們很容易地從親密接觸的人、床單、枕頭、毛巾等帶走牠們，然後只消六個星期，牠們又再次鋪滿你的臉。

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This article may be useful as supplementary reading for biology classes, based on the DSE syallbus. 根據生物科文憑試課程剛要，本文或可作為有用的補充讀物。

蘇韋霖
By Raphaella So

Can Plants develop cancer? This is an easy question to ask, but the answer is more convoluted than one might imagine. In the 90s, two related articles were published in close succession: "Why don't plants get cancer?" and "Plants can get cancer". The first article argued that plants could tolerate abnormal cell division (which is what cancer is) by incorporating the extra cells into other development to prevent these cells from forming tumours [1]. The second article conveys a conflicting opinion. It proposed the notion that plants that develop cancer may appear otherwise normal but are harbouring cancer internally. However, "plant cancer" may not refer to the same thing as cancers in mammalian animals, because the symptoms of plant cancer are manifested differently [2]. Years later, scientists continue to contribute to this debate [3].

In animal cells, mutations in two types of genes can cause the formation of tumours: proto-oncogenes and tumour suppressor genes. The former facilitates normal cell division by coding for proteins that regulate the process. Certain mutations, however, transform the gene into an oncogene and cause the cell to synthesise a lot more protein products than required. An overabundance of cell division promoting proteins in turn leads to continuous cell proliferation and results in tumour formation. During normal cell division, another group of genes, the tumour suppressors, act as guards at different checkpoints and prevent abnormal or uncontrolled cell division. At the DNA damage checkpoint, for example, relevant tumour suppressors would not allow the cell to enter mitosis until all the damaged DNA has been repaired. Mutations can render the tumor suppressor genes ineffective, allowing damaged DNA to be passed onto the daughter cells. This process results in the accumulation of even more mutations, causing uncontrolled cell division. Moreover, if the genes that help anchor the cells to their normal locations are mutated, the tumour can then spread to other parts of the body by metastasis.

Like in animal cells, mutations in proto-oncogenes and tumour suppressors can occur in plants. However, except in certain hybrid species, these mutations are rarely oncogenic. Interestingly, the most frequent cause of plant tumours is due to pathogenic infection [3]. Certain species of bacteria can transfer DNA into the plant cell nucleus and alter the signalling of plant growth hormones. Fungal pathogens can also promote cell proliferation by affecting these hormones. However, they transfer proteins instead of DNA into the plant cell. While some viruses can also act through

hormones, geminiviruses bypass the endocrine system and directly inhibit tumour suppressors, encouraging the cells to replicate.

Nonetheless, despite various ways of tumour formation in plants, its occurrence is much less frequent than in animals. One possible explanation is that the checks and balances against abnormal cell division in plants appear to be more effective in plants than in animals. For instance, plant stem cells are hypersensitive to DNA damage and undergo programmed cell death in response to abnormalities. Thus, it is rare that plant cells will accumulate mutations that lead to cancer.

The structure and organisation of plant cells also serve to protect plants from tumour formation or metastasis. Unlike animal cells, plant cells possess rigid cell walls that maintain proper cell structure and prevent disorganised cell growth. Cells are also attached to each other and are immobile. Furthermore, plants do not rely on cellular circulatory systems such as blood or lymph vessels. Instead, they rely on an acellular vascular system that consists of the xylem and phloem. This system significantly limits the locomotion of tumour cells. Hence, a collective deregulation of cell division must arise in order for a tumour to form. In short, yes – plants can develop tumours, or "cancer", but they occur a lot less often than in animals and are certainly not as lethal.

植物也會患癌症嗎？這問題看似容易，但是答案卻比想像中複雜。90年代，科學家們前後發表了兩篇論文：「為什麼植物沒有癌症？」和「植物可以得癌症」。第一篇指出，就算植物有異常的細胞分裂（也就是癌症），也會把多餘的細胞融入自己正常的成長過程。因此，植物細胞不會形成腫瘤[1]。可是，第二篇論文卻反駁了這個論點提出植物即使沒有病徵，它還是會患癌。只是這種癌症和動物患的癌不太相同[2]。許多年後，科學家們還是繼續參與這場辯論[3]。

動物細胞裏有兩類基因的突變可以形成腫瘤，分別是：原癌基因（proto-oncogenes）和腫瘤抑制基因（tumour suppressor genes）。原癌基因製造出來的蛋白質調控細胞分裂，本是有助正常分裂過程。但當原癌基因突變成癌基因時，細胞會異常地大量生產那種蛋白質，使細胞不停分裂，形成腫瘤。正常情況下，腫瘤抑制基因，會防止失控和異常的細胞分裂過程。例如在DNA損傷檢查點，腫瘤抑制因子在所有的受損的DNA獲修復前，不會允許細胞進入有絲分裂。可是，由於基因突變令腫瘤抑制基因失效，容許受損的DNA進入子細胞，導致更多的基因突變，釀成細胞分裂失控。更甚者如果幫助錨定細胞在正常位置的基因發生突變，腫瘤就轉移擴散到身體的其他部位。

跟動物細胞一樣，植物細胞也會發生原癌基因和腫瘤抑制基因的突變。然而，除了部份雜交品種之外，這些基因突變很少會形成腫瘤。因此，最常見的植物致癌原因是感染[3]。有些細菌會把脫氧核醣核酸（DNA）轉移進植物細胞核，影響植物生長激素的訊號傳遞。另外，真菌也會把蛋白導入植物細胞，影響這些激素以促進細胞增殖。有些病毒也會透過激素來加快細胞繁殖，但其中雙生病毒（Geminiviruses）卻能繞過內分泌系統，直接抑制腫瘤抑制蛋白，從而鼓勵細胞分裂。

儘管在植物中有諸多途徑產生腫瘤，植物患癌的機率大大低於動物。一個可能的原因是，植物比動物更能有效制衡異常的細胞分裂。例如，每當植物的幹細胞有DNA受損便會立刻進入細胞凋亡，令突變細胞不能積累，無法形成腫瘤。

植物的組織和細胞結構也會防止腫瘤的形成和擴散。植物細胞擁有堅硬的細胞壁，可防止細胞異常地生長。加上植物細胞互相緊靠，不能隨意移動。此外，植物亦沒有血液和淋巴等細胞循環系統（circulatory system），只有由木質部（xylem）和韌皮部（phloem）組成的無細胞維管系統（vascular system），癌細胞沒法移動和擴散。因此，必須要集體細胞分裂失控才能形成腫瘤。簡而言之，是的——植物可以形成腫瘤並患上「癌症」，但不及動物腫瘤普遍，而且絕對沒那麼致命。

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Why is it so Difficult to Swat a Fly?

Perhaps we are all too familiar with the irritating experience of unwelcome winged house guests buzzing obnoxiously above our food, trying to share a bite of our meal. Flies feed freely on both food and fecal matter, carrying the devastating risk of transmitting enteric, eye or skin infections. However, successfully murdering these pesky little disease incubators is no mean feat, as they seem to possess an arsenal of abilities to evade the swing of death.

Flies are evolutionarily equipped for escape. With a pair of compound eyes that provide an almost 360° field of vision, they have undisputedly one of the fastest visual response times in the animal kingdom and are able to track movements five times more quickly than do human eyes. A study conducted in 2012 suggested that their rapid response time is partially attributed to a physical contraction of photoreceptors - specialised cells found on the retina. These photoreceptors respond to light, which in turn generate electrical responses that are directed almost instantaneously to the brain. In contrast, the typical mechanism of using chemical messengers for transmission is much slower [1].

In addition to their keen spatial awareness, they are also equipped with three pairs of athletic legs, with the ability to adjust position faster than you can blink. Their tiny brains are able to process an incoming threat and prepare their escape by aligning their legs to the optimal position to hop in the opposite direction of said threat, all within 100 thousandths of a second. For instance, if the direction of the threat is positioned in front of the fly, it moves its middle legs forward, leans back and extends its legs, thrusting itself backward. They are keenly aware of looming danger and take into account their body position whether they are grooming, feeding, walking or courting. Thus, they are required to integrate the visual and the mechanosensory information to adjust their pre-flight pose [2].

These insects have the ability to turn flight muscles on and off incredibly fast, contributing to their rate of successful escapes. In order to save time needed for electrical responses to travel from the brain to the muscle, muscles that move the wings bypass the brain completely to undergo stretch activation – an automatic contraction of a set of muscles when they are stretched as a result of the contraction in opposing muscles. Decades ago, scientists have reported that flies "freeze" mid-flight or mid-walk when they sense air stream in the vicinity. Their antennae contain two groups of neurons – one of which responds to flowing air particles, and the other responds to sound [3].

Gaining an understanding of the flight and reaction mechanics of flies can provide insight into developing airborne robots that are able to evade obstacles. As for successfully swatting a fly? It appears that your best bet is to clap the area directly above them or in front of them to anticipate their flight trajectory. After all, flies have impressive reaction time to danger, but don't have much for brains.

為什麼撲殺蒼蠅這麼困難？

By Jacqueline Aw 歐婷梅

或許你對蒼蠅不太陌生。它們常常衝著佳餚而來，試圖分去我們的一點美食。它們也會以糞便或變質的有機物為主食。因此，它們不但會污染食物，也是腸胃、眼或皮膚病菌的載體。想打死這些討厭鬼是否那麼容易？不！他們似乎能迅速逃離死神之手。

經過漫長歲月，蒼蠅演化出逃生絕活。它們的複眼提供全方位視野。蒼蠅的視覺反應在動物世界中堪稱數一數二，追蹤移動物件的速度比人類的眼睛快五倍。二零一二年，一項研究指出視網膜感光細胞的物理收縮是影響蒼蠅反應的因素之一。接收光子後，感光細胞會產生電流，並瞬間傳輸到大腦。相比之下，依靠化學訊息傳遞的典型機制，傳送速度就慢得多[1]。

除了擁有敏銳的空間感，那三對節肢可讓蒼蠅快速調整位置。當遇到外來威脅，腦部在十萬分之一秒內，調整好節肢最佳位置，預備向威脅的相反方向逃脫。例如，如果威脅是在蒼蠅的前方，它會把中間節肢伸向前，身體向後傾斜，接著雙腿一伸，向後施力。不論在任何地方、任何時間，它們對週遭的危險極為敏銳，並會計算身體的位置。它們常常會整合視覺和應力感覺資訊來調整飛行前姿勢[2]。

蒼蠅能夠極速啟動或關閉飛行肌，增加逃跑的成功率。為了節省電流傳遞時間，連接雙翼的肌肉組因拮抗肌收縮而被牽張時，就會自動收縮，直接啟動，不須經由腦部控制。數十年前，科學家報告當蒼蠅感覺到空氣流動，它們會"凍結"飛行或其他活動。它們的觸角包含兩組神經元，一組偵測流動的空氣粒子，而另一組偵測聲音[3]。

了解蒼蠅的飛行方式和反應機制，有助開發能夠迴避障礙物的空中機器人。至於如何成功撲殺蒼蠅？大概最好的辦法是預見它的飛行軌跡，拍打它的上方或面前的區域。蒼蠅雖然可以對危險作出迅速反應，但畢竟不及你聰明。

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THE MONTY HALL Probabili

This article may be useful as supplementary reading for mathematics classes, based on the DSE syllabus.

Imagine you are on a game show and the host displays three large boxes, one of which contains the grand prize of a car and the other two contain goats. You will be awarded the contents of the box that you choose at random, but the contents will not be immediately revealed. After you make your choice known, the host opens one of the other two boxes and reveals a box with a goat. The host then asks if you would like to switch your chosen box for the remaining unopened box.

The scenario is from a notable statistical game known as the Monty Hall Problem. This mathematical problem is simple enough to understand, but the answer is somewhat counterintuitive. What would you do in this scenario? Would you stick with your original choice or would you swap? Which would make more sense in giving you a higher chance of winning the car? When this problem was released in a magazine back in the 70s, most believed that it made absolutely no difference whether you swapped or stuck to your choice, and that the chance to win the grand prize stayed at 50% either way. Intuitively sensible – but statistically incorrect.

The mathematics reveals that swapping nearly doubles the chance of winning the grand prize. However, it should be noted that this is only true when the following 3 conditions are met [1]:

(1) The host does not reveal the original choice;

(2) The host always opens a box containing a goat (we are assuming that he knows what each box contains);

(3) The host makes a random choice of boxes to open, when your initial choice was correct.

When you are asked to pick a box at the very beginning, the probability of selecting the grand prize is one out of three (1/3). The probability of picking a goat is 2/3. If you do not decide

ty PROBLEM 反直覺的統計學

根據數學課文憑試課程剛要，本文或可作為有用的補充讀物。

By Thomas Lee 李浩賢

試想像在遊戲節目中，主持人給你三個箱子。當中一個藏有名貴房車，另外兩個裝著兩頭羊。你隨機選擇一個箱子，可以得到內藏的獎品，不過主持人不會即時告訴你箱子內是什麼，而是打開另外兩個箱子中的一個，露出了一頭羊。主持人繼續問：「你會否用你選擇的箱子換取未開的箱子？」

這個情景改編自著名的數學遊戲：三門遊戲（或蒙提霍爾問題）。故事不難理解，但答案卻是有點違反直覺。你可能會糾纏在交換還是堅持原來的選擇，同時，你會考慮哪個決定有較大勝數。在七十年代當一本雜誌發表三門遊戲時，大多數讀者認為交換及堅持沒有任何分別，因為兩者贏取大獎的機會都是一半。儘管從直觀上看似合理，在統計角度而言卻不正確。

數學告訴我們如果換掉當初的選擇，你有雙倍機會贏得大獎，但必須滿足以下三個條件[1]：

（1）主持人不會打開你最初的選擇；

（2）主持人打開的箱子必定裝著羊（我們假設主持人原本便知道箱子內藏著什麼）；

（3）若你最初的選擇是大獎，主持人便會隨機打開其中一個未開的箱子。

開始時你被要求挑選一個箱子。在這個情況下你獲得房車的概率是1/3，選擇羊的概率為2/3。若你堅持最初的選擇，獲得大獎的機率仍然是1/3。但是，如果你決定換掉原本的選擇，獲勝的概率是2/3，你有雙重機會獲得房車！

不相信？圖1顯示有三種選擇：A、B和C。假設你選擇A而主持人打開C，大獎在A的概率相對於大獎在B的概率是甚麼？

to change your original choice, the chance of winning the grand prize remains at 1/3 – regardless of which box the host opens. However, if you decide to switch from your original choice to the remaining unopened box, you end up increasing your chances of obtaining the car.

From **diagram 1**, there are three choices: A, B and C. Suppose you choose box A and the host opens up box C. What is the probability that the grand prize is in box A **given** that the host opened box C, versus the probability that the grand prize is in box B **given** that the host opened box C?

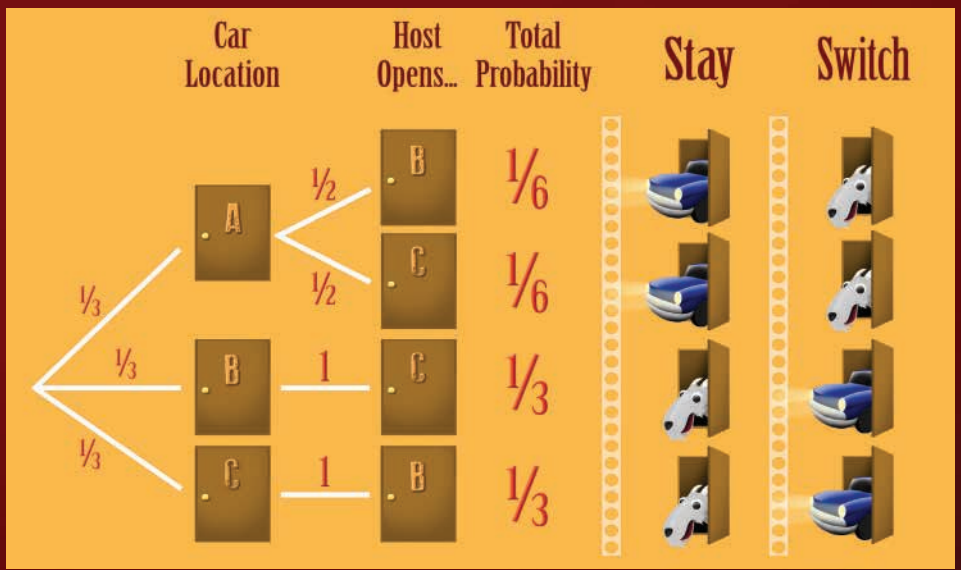

Diagram 1 圖1

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Let P (A|C) be the probability that the prize is in box A given that the host opens box C. Using Bayes Theorem,

$$P(A|C) = \frac{P(C|A) \times P(A)}{P(C)}$$

we get,

$$P(A|C) = \frac{\frac{1}{2} \times \frac{1}{3}}{\frac{1}{2}} = \frac{1}{3}$$

If the grand prize is in box A, the probability that it is in box A given that the host opens box C is 1/3. To calculate the probability that the prize is in box B, we use Bayes theorem again to give:

$$P(B|C) = \frac{P(C|B) \times P(B)}{P(C)}$$

P(B) = 1/3 and P(C) = 1/2. If the grand prize is in box C, then the host can only open box B, giving us P(C|B) = 1. Substituting these values we obtain:

$$P(B|C) = \frac{1 \times \frac{1}{3}}{\frac{1}{2}} = \frac{2}{3}$$

Thus, swapping gives us a 2/3 chance of selecting the grand prize, doubling our chances. Still unconvinced? Let's look at an example where there are 100 boxes. The probability of picking a grand prize is 0.01 (1%) and the probability of picking a goat is 0.99 (99%). Thus, the only time swapping is a bad idea is if you had already picked the winning box, provided that the host shows you 98 other boxes with goats.

It was shown that the simple pigeon adapted to the Monty Hall Problem surprisingly well [3]. In an animal experiment, pigeons were shown three computer-controlled lit keys, one of which contained food. On the first pigeon peck, the computer would switch off all three keys but would then switch two back on shortly afterward, one of which was the pigeon's original choice. The pigeon was then awarded food if it selected the correct key in the remaining lit keys. At the beginning, only one third of the pigeons switched to a different key. However, after one month, all six pigeons involved in the study switched their choice consistently to obtain the highest chance of selecting food, indicating that switching became a learned behaviour from reinforcement.

In a similar set up, students were also given three lit keys and instructed to obtain the highest points. Again, over one month, they were to guess the right keys, limited to 200 tries. Their results unfortunately did not measure up to the pigeons. Equally likely to switch or stick to their original choices in the beginning, there was little sign of improvement toward the end of the month.

While this experiment does not indicate that pigeons are smarter than humans, it does show us that we tend to do a lot of overthinking, perhaps leading to false reasoning. Interestingly enough, it was the youngest students who fared best in this experiment, and the older students were more likely to overthink. Not too surprising, considering it was claimed that Nobel physicists routinely gave the wrong answer to the Monty Hall Problem!

反直覺的統計學

THE MONTY HALL Probability PROBLEM

設P(A|C)為獎品在A，而主持人打開C的概率。運用貝氏定理，

$$P(A|C) = \frac{P(C|A) \times P(A)}{P(C)}$$

我們得出：

$$P(A|C) = \frac{\frac{1}{2} \times \frac{1}{3}}{\frac{1}{2}} = \frac{1}{3}$$

所以，如果大獎藏在A箱，那麼大獎在A而主持人打開C的概率為1/3。現在，讓我們計算大獎在B的概率。運用貝氏定理得出:

$$P(B|C) = \frac{P(C|B) \times P(B)}{P(C)}$$

我們知道P(B) = 1/3和P(C)=1/2。而唯一區別是，如果大獎在C，那麼主持人只能打開B，那概率為P(C | B) =1。運算為：

$$P(B|C) = \frac{1 \times \frac{1}{3}}{\frac{1}{2}} = \frac{2}{3}$$

因此，換掉原來的選擇帶給我們2/3機會贏得大獎，即是有雙倍機會。如果以上的答案無法說服你，那我們看看另一個例子：由100個箱子中選擇大獎。最初，獲得大獎的概率是 0.01 (1%)，而有0.99 (99%) 機會獲得安慰獎。只有在你已選中了大獎，而主持人亦指出其餘98個箱子都藏著山羊，交換才會不利。

有實驗指出鴿子在三門遊戲的表現是出奇的好！在一個動物實驗中，人員展示三個按鈕給鴿子，按鈕由電腦控制，而且可以發光，當中只有一個選擇會讓鴿子獲得食物。在第一次選擇後，電腦先關上三個按鈕。接著，兩個重新發光，其中一個是鴿子原本的選擇。如果鴿子能夠正確選擇發光的按鈕，就會獲得食物。在實驗初期，約三分之一的鴿子切換到不同的按鈕。一個月後，六隻鴿子全都不停切換按鈕，爭取最大機會拿到食物，由此證明「改變選擇」是可以透過獎勵強化而學習的行為。

在類似的實驗中，學生須要操作三個發光按鈕以爭取最高分數。同樣在一個月內，他們有二百次機會，猜測正確的發光按鈕。遺憾的是他們的表現比鴿子遜色。實驗初期，他們切換和保留原本選擇的概率大致相同，到月底仍未有明顯進步[3]。

這個實驗並非證明鴿子比人類更聰明，只是告訴我們，人類很容易想得太多，導致錯誤推理。有趣的是在實驗中，低年級的學生表現最好，高年級的學生易傾向於過多思慮。正因為這樣，有人認為諾貝爾獎物理學家往往會錯誤回答蒙提霍爾問題！

Winning article of the *Science Focus* Article Submission Competition.
「科言」徵文比賽得獎文章。

Itching to Know: Why Do We Itch?

我們爲什麼會痕癢？

By Lee Lok Sze
李樂思
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Itching is an irritating sensation that most people experience on a daily basis. Typically, itches go as swiftly as they come, but a stubborn itch can last for far too long that does not seem to abate despite continuous scratching, or for the more determined - willfully ignoring. What exactly is the mechanism that causes our skin to itch?

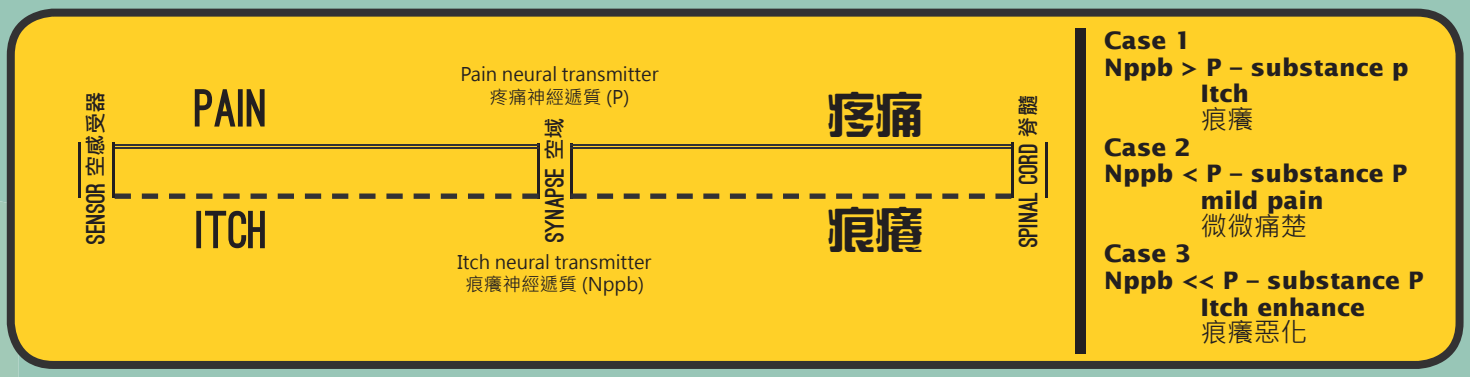
Clinically known as pruritus, itching can be triggered by various irritants. Itching occurs when an external stimulus, such as a feather or dust, comes into contact with the skin. As the stimulus brushes against skin, it fires receptors situated at the surface of the skin which then generate nerve impulses [1], an electrical signal that passes through a neuron. The journey of a nerve impulse begins from one neuron to the next, stopping at the synapse which causes the ends of the axons – a neuron's nerve fiber – to release a chemical known as the neurotransmitter. This then diffuses across the synapse before attaching to the membrane of the destination neuron, which then generates another impulse in a relay-like fashion. Repeating this action from one neuron to the next passes the itch signal onto the spinal cord and eventually, the brain registers it as an itch.

For a long time, scientists believed that the sensation of itching was the result of pain related neurotransmitters and receptors, and that itching was simply considered as a milder form of pain [2]. This was questioned in a paper published in 2013, which reported that the neurotransmitter — natriuretic polypeptide B (Nppb), and the sensory receptor — natriuretic peptide receptor A (Npra), are the primary vehicles that carry the itching sensation [3]. Neurons containing Npra detect Nppb from sensory neurons and then continue to transmit the itching sensation to the brain. What is important to note, however, is that Nppb and Npra are specific to the itch sensation pathway, and thus is a sensation distinct from pain.

How then, does our body relieve itself from this sensation? The body's natural response to an itch is the urge to scratch. Scratching produces a mild form of pain that overrides the transmission of the sensation of itching into the spinal cord [4], resulting in a temporary pleasurable feeling that relieves the itch. What is not desired, however, is when the brain releases a pain-controlling neurotransmitter called serotonin in response to the pain generated by scratching. While serotonin acts as a pain suppressor, it can also affect itch-sensing neurons by increasing the intensity of the itch sensation [5]. This is why scratching sometimes simply makes things worse and leads to a vicious itch-scratch cycle.

Having discovered the mechanisms involved in itching, scientists are now looking for a solution to relieve this nagging sensation. The challenge lies in the fact that Nppb is also responsible for regulating blood pressure, while serotonin is involved in other bodily processes. Therefore, simply suppressing either of these chemicals would not be a practical option. On the bright side, there are other factors that govern the sensation of itching which have yet to be discovered, and scientists are investigating whether another type of neuron exist, which may be part of the reason why most itches go away after scratching [6]. Although this is still underway, it is no doubt an interesting problem to think about the next time you feel an itch!

This article was edited to fit the needs of the Science Focus publication. 本文以「科言」所需而被更改。



我們幾乎每天都會感受到痕癢這種惱人的感覺。通常，這種感覺過了片刻就會消失。不過有時候，不管我們怎麼抓它、搔它，甚至嘗試不去理會它，它仍會繼續頑固地困擾著我們。究竟是甚麼原因導致我們的皮膚痕癢呢？痕癢的機制又是怎樣的？

痕癢或稱「風瘙癢」，是一種由不同刺激物所引致的感覺。當羽毛、塵埃等外來刺激跟皮膚接觸，皮膚表面的感受器就會受到刺激並產生神經脈衝[1]，即在神經元中穿過的電信號，並形成痕癢的感覺。當神經脈衝由第一個神經元走到第二個的時候，它其實不會真正的移動過去。相反，它會停在第一個神經元的突觸(即兩個神經元之間的空域) 前，導致軸突 (即神經元細胞本體伸展出來的部分)的末端釋放一種名為神經遞質的化學物質。接著，這個神經遞質通過突觸擴散到第二個神經元的細胞膜並與之連接，令那個神經元也產生另一個神經脈衝。如此類推，痕癢的神經脈衝便可由皮膚的感受器傳到脊髓，直達大腦。

一直以來，科學家以為痕癢的感覺源於疼痛相關的神經遞質和感受器，因此痕癢不過是一種較弱、低程度的疼痛[2]。可是，在2013年，科學家Mishra和Hoon[3]發現神經遞質B型利鈉肽(Nppb)以及感受器A型利鈉肽受體(Npra)是傳送痕癢資訊的主要工具。含有Npra的神經元會接收感受器傳來的Nppb，然後繼續把痕癢信息傳送到大腦。因為Nppb和Npra只針對痕癢資訊發揮作用，由此可見痕癢和疼痛是截然不同的。

那麼我們怎樣才能止癢呢？我們身體的自然反應是去搔刮它，當中的原因是搔刮會產生微微痛楚，中斷脊髓裏痕癢信息的傳送[4]，令痕癢得以舒緩。不過，大腦同時也會釋放控制疼痛的神經遞質—血清素—來對抗搔刮所製造的痛楚。血清素不但會壓抑神經元中的痛楚感覺，而且還可以轉移到其他覺得痕癢的神經元，增加痕癢的強度[5]。所以，有時候搔刮痕癢處只會令其惡化，形成一個越搔越癢的惡性循環。

發現痕癢機制後，科學家現正尋找止癢的方法，可是目前還是挑戰重重。上述的Nppb能幫助調節血壓，而血清素也是身體其他機能不可或缺的，因此抑制這兩種化學物質以達到止癢的目的是不切實際的。可幸的是尚有其他影響痕癢的因素還未被發現，科學家也正在研究另一種可能與搔刮止癢有關的神經元[6]。雖然可能需要一段時間才能取得成果，但這確實是一個相當有趣的課題，大家不妨在下一次痕癢的時候思考思考！

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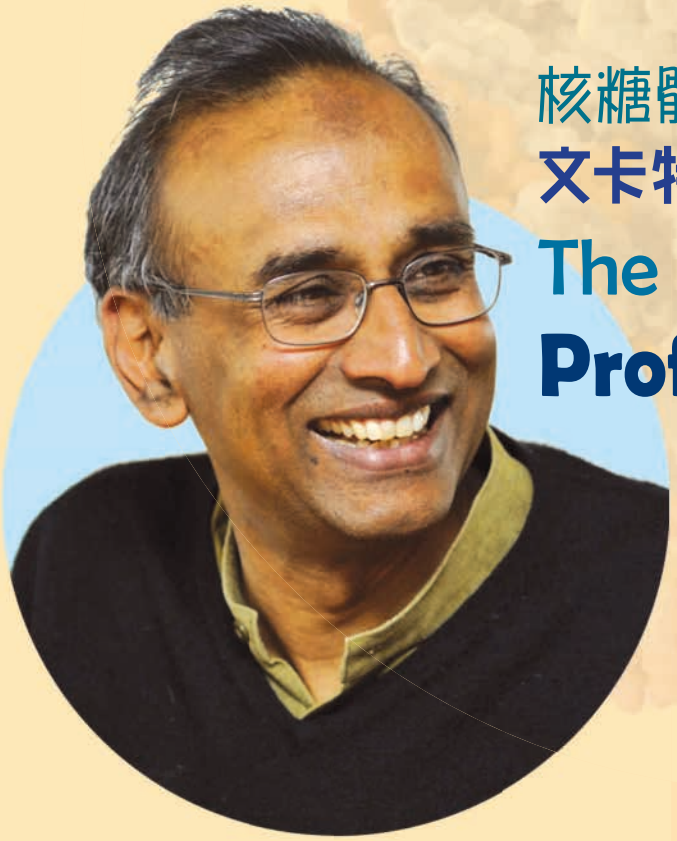


核糖體——文卡特拉曼·拉馬克裡斯南教授

The Ribosome with Prof. Venkatraman Ramakrishnan

By Cherry Chow 周卓瑩

Perhaps being accepted into graduate school for physics at the tender age of 19 was a clear indication that Prof. Venkatraman Ramakrishnan was destined for greatness. Convinced that he had chosen the wrong field half way through his Ph.D. and unable to see what the future in physics held in store for him, he made the difficult decision to switch to biology after already obtaining his Ph.D. in physics — a field that seemed to mimic the exciting phase that physics was in, in the first part of the 20th century. He returned to graduate school to learn biology from the basics, leaving behind all shreds of the arrogance of a physicist but retaining the translatable knowledge he had gathered as an applied physicist to his new field.

After his switch to biological studies, Prof. Ramakrishnan familiarised himself with the ribosome. The ribosome was first observed under an electron microscope in the 1950s by cell biologist George Emil Palade. However, its cellular role was not well understood at the time. A cell organelle universal to bacteria, eukaryotes and mitochondria, the ribosome has the fundamental role in deciphering translation of genetic information to create proteins. In addition to reading RNA, ribosomes also have the vital task of linking amino acids together – what was previously thought by some biologists to be the result of proteins linking themselves. Merely a speculation for decades, the result of solving the structure of the ribosome confirms its essential role in protein synthesis. In Prof. Ramakrishnan's own words, it is the building block essential to all of life; and everything made in a cell is either made by the ribosome or by enzymes that themselves were made by the ribosome.

The road to deciphering the ribosome was not one without tribulations. Ribosomal structure consists of a large and small subunit that fit together in operation. Prof. Ramakrishnan and his team were able to obtain crystals of the ribosome subunit but not of the small subunit. To complicate matters, the crystals were prone to radiation damage from their methods, and larger than anything that they had solved previously. In addition, trapping the entire ribosome in its precise functional state was an arduous and time-consuming process. However, their perseverance paid off.

2009 marked the year that Prof. Ramakrishnan was jointly awarded the Nobel Prize in Chemistry with Thomas A. Steitz and Ada E. Yonath for their work in mapping the structure and function of the ribosome, and decoding the processes at the atomic level. Their critical role in the synthesis of proteins is essential to life. Not only are they important nutrients that help build muscle, but they are the molecules responsible for almost every molecular action in the human body. From haemoglobin that circulates in blood, to insulin that regulates the body's glucose levels. The

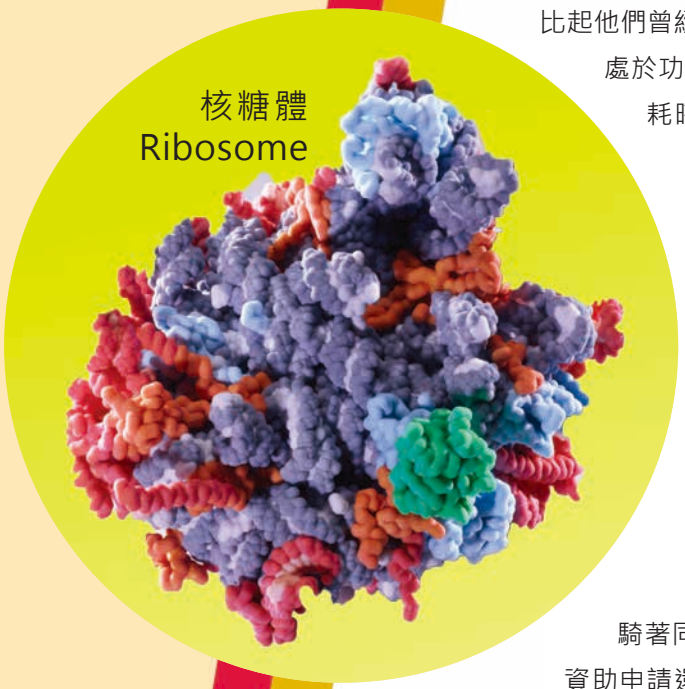
understanding of the inner-workings of the ribosomes will help scientists to develop new antibiotics that are more efficient and more effective.

Life after the Nobel Prize has remained virtually the same for Prof. Ramakrishnan. He still rides the same bicycle and lives in the same house and joked that papers or grants are just as difficult to get accepted or awarded. For now, he continues to work on the ribosome. Its mysteries are far from being fully deciphered and with such complex biological functions, each advance opens even more questions. "It is unlikely that ribosomes and the regulation of translation will stop being interesting during my working lifetime."

文卡特拉曼•拉馬克裡斯南教授以19歲稚齡獲研究院錄取修讀物理學，這或許可以顯示他註定是要成就大事。在攻讀博士課程時，他看不清自己在物理領域的未來前景，認為選擇錯誤，於是在取得博士學位後，作出了艱難的決定，轉投生物學 — 那個正像20世紀初的物理學那樣蓬勃發展的範疇。他回到研究院從頭學習生物學，放下物理學家的身段，將應用物理學家的知識轉化運用在新領域中。

拉馬克裡斯南教授改投生物學後，即著手研究核糖體(ribosome)。在20世紀50年代，細胞生物學家喬治•艾米爾•帕拉德運用電子顯微鏡，首次觀察到核糖體。然而，當時還未十分清楚其細胞作用。核糖體是細胞器的一種，普遍存在於細菌(bacteria)、真核生物(eukaryotes)和線粒體(mitochondria)，在轉譯遺傳信息製造蛋白質的過程中起基礎性作用。除了讀取核糖核酸(RNA)的遺傳資訊，核糖體還負責連接氨基酸(amino acids)。在此之前，有些生物學家以為蛋白質是自行連接的。數十年來只能憑臆測推斷核糖體在蛋白質合成中的重要作用，最後因核糖體的結構研究而得到證實。引用拉馬克裡斯南教授所說，核糖體是所有生命的基本組件，細胞內的一切皆由核糖體，或是由核糖體所製成的酶(enzyme)所構成。

解析核糖體的過程並非一帆風順。核糖體是由在運作時相互嵌合的大，小亞基構成。拉馬克裡斯南教授和他的團隊起初得到的核糖體亞基晶體並非小亞基。小亞基晶體容易因當時使用的方法遭輻射損壞，而且結構要比起他們曾經處理的要大得多，讓研究更具複雜性。此外，要精確地捕捉處於功能狀態(functional states)的整個核糖體是一項艱鉅而耗時的過程。最後，他們的堅持得到了回報。



2009年，拉馬克裡斯南教授聯同托馬斯•施泰茨和愛達•約納芙，獲頒諾貝爾化學獎，以表彰他們對核糖體的結構和功能研究，成功地在分子水平上描繪這些過程。核糖體在蛋白質合成過程中有關鍵作用，是生命必須，不僅是有助塑造肌肉。從血液中的血紅蛋白(haemoglobin)，到調節葡萄糖水平的胰島素(insulin)，核糖體負責人體內幾乎每一個分子作用的運作。深入了解核糖體的運作，將有助科學家開發更為高效和有效的新一代抗生素。

拉馬克裡斯南教授獲獎後，生活並沒有太大改變。他還是騎著同一輛自行車，住在同一所房子。他更開玩笑說，論文投稿和資助申請還是同樣地難獲接納或批准。現時，他繼續進行核糖體的研究工作，還有許多奧秘未被完全破解。核糖體具有如此複雜的生物學功能，每解開一個謎題，就會衍生更多的問題。「我相信，至少在我的研究生涯中，核糖體與翻譯功能調控將會是趣味不減的課題。」

Breaking Boundaries:

By Cherry Chow 周卓瑩

Since the development of the modern optical microscope, we have been limited to viewing microscopic objects at a resolution of roughly 250 nm. Even with a perfect microscope – that is, one not limited by lens imperfections or alignment issues – for a while it was thought that we cannot bypass this diffraction limit, which is half the wavelength of light. The diffraction limit is enough to examine most biological cells but smaller biological components such as viruses or proteins lie outside of this range. Attempts to circumvent the diffraction limit have involved the use of shorter wavelengths such as ultraviolet or X-ray but these rays can damage cellular matter and thus are not entirely suitable for biological use. The development of super-resolved fluorescence microscopy, however, has taken microscopy to the next level.

In 2014, Prof. William E. Moerner was recognised for his work in the development of super-resolved fluorescence microscopy with a joint Nobel Prize in Chemistry with Prof. Eric Betzig and Prof. Stefan W. Hell. Currently serving as the Harry S. Mosher Professor in Chemistry and Professor of Applied Physics by Courtesy at Stanford University, Prof. Moerner holds, impressively, three bachelor's degrees and a Ph.D. in physics.

The development of super-resolution involving single molecules began in the 80's. Working together with his postdoc, Lothar Kador, at IBM Research in San Jose, Prof. Moerner was the first person to optically detect a single molecule, through the use of laser frequency modulation spectroscopy. In 1997, they observed green fluorescent proteins (GFP), which exhibit bright green fluorescence if exposed to the lower wavelengths of the visible light range and subsequently discovered that GFPs can blink and be optically switched.

His award-winning work has revolutionised the visualisation of objects on the nanoscopic scale. Optical study of single molecules and molecular mechanisms of living cells have become possible due to the development of super-resolved fluorescence microscopy, expanding microscopy to nanoscopy. In our interview, he explained that the structure of interest is first labelled with fluorescent dyes. Then these single-molecule emitters are viewed under conditions where only a few are emitting at a single time via photoactivation, for example. The positions of the emitters are pinpointed, which continue over some time, where the single points of light allow sampling of many positions of the structure, until the full structure is constructed. The end product is a computational image formed with tiny points of light.

In the past, scientists were only able to study many molecules simultaneously and take an average of all those results. Super-resolved fluorescence technology has enabled the study and image single molecules, allowing for much more accurate and detailed information. "If we can watch them one by one, then we can learn much, much more". Prof. Moerner's current work

Further reading

http://www.nobelprize.org/nobel_prizes/chemistry/laureates/2014/popular-chemistryprize2014.pdf

http://jcb.rupress.org/content/190/2/165.full.pdf+html

focuses on exactly that. They attempt to extract more and more information from a single molecule, track its activities and image them into many new avenues of science.

Since winning the Nobel Prize, life has been more or less the same for Prof. Moerner. However, his monthly trips have increased to 3-5 times a month, with invitations to speak both domestically and internationally. He manages these new engagements and continues his research simultaneously, having to be very cognizant of time management.

縱使現代光學顯微技術發展一日千里，一直以來，觀察對象的大小都只限於250納米或以上。我們曾經以為即使採用完美的顯微鏡，沒有透鏡缺陷或對焦問題，也無法突破衍射限制（diffraction limit）（即光的波長的一半）。衍射限制一般無礙觀察大多數的生物細胞，但對檢視更小的生物體（如病毒、蛋白質）則影響很大。有人試圖使用較短的波長，例如紫外線和X射線，以克服衍射限制。可惜這類光線會損害細胞，並不太適合用於生物學研究。直至超解析度螢光顯微技術（Super-resolved Fluorescence Microscopy）的誕生，成功帶領顯微技術邁進新時代。

威廉•莫爾拿教授憑著研發超解析度螢光顯微技術，與艾力克•貝齊格教授及斯特凡•荷爾教授共同獲得2014年的諾貝爾化學獎。莫爾拿教授現為史丹福大學哈裡•莫舍化學系教授，以及應用物理學系教授。不得不提的是，莫爾拿教授擁有三個學士學位及物理博士學位。

牽涉單個分子的超解析度顯微技術，發展始於80年代。在聖何塞的IBM研究院（IBM Research），莫爾拿教授與他的博士後研究員洛薩•卡多爾協力研究，成為通過鐳射調頻光譜分析（laser frequency modulation spectroscopy）檢視到單個分子的第一人。1997年，透過觀察綠色螢光蛋白（GFP），也就是一種在波長較短的可見光線激發下會發出綠色螢光的蛋白，他們發現GFP可以閃爍的特性，其光線的開關亦可被控制。

此項獲獎研究，無疑為納米級別的觀測帶來了革命性的改變。超解析度螢光顯微技術的出現，使針對單個分子及活細胞分子機制的光學研究變得可能，更將普通顯微拓展至納米顯微。在我們的訪問中，莫爾拿教授解釋了這技術的使用方法和過程。首先使用螢光染料標記想研究的指定結構，接著在特定條件下進行檢測，每次只讓少數單分子的放射體同時發光。放射體被精確定位，如此重複經過一段時間，採集了大量的放射體位置後，電腦會計算出該結構的完整形狀，最後以小光點呈現出來。

科學家過去只能同時研究多個分子，再把數據取平均值。超分辨螢光技術卻可直接圖像化單個分子，從而取得更準確、更詳細的資訊，有利研究。「若果我們可以逐一觀察它們，我們可以學習到更多更多。」莫爾拿教授目前的研究重點正正在此。他們嘗試提取越來越多有關單分子的資訊，跟蹤其活動，並造就許多科學上新的可能性。

贏得諾貝爾獎後，莫爾拿教授的生活大致依舊。不過由於要到國內外多地進行演講，他每月出差的次數增加3-5次。現在他既要安排這些新工作，同時又繼續研究，令他更加發覺到時間管理的重要性。

Prof. William E. Moerner

打破界限：威廉•莫爾拿教授

Quiz 測一測

1. Which type of birds are the only birds that can distinguish the colour blue?
   以下哪種鳥類可以分辨藍色？
   a. Owls 貓頭鷹
   b. Blue Jays 藍松鴉
   c. Sparrows 麻雀
   d. Hawks 鷹
2. How long does it take for light to travel around the Earth?
   光環繞地球轉一圈要多久？
   a. 1.02 seconds 秒
   b. 2.11 seconds 秒
   c. 0.52 seconds 秒
   d. 0.13 seconds 秒
3. How many bones are humans born with?
   人體天生有多少根骨頭？
   a. 199
   b. 206
   c. 254
   d. 300
4. True or False: the second most lightest material on Earth is a metal.
   是非題：地球上第二最輕的物質是一種金屬。
   a. True 是
   b. False 非
5. A ship anchored in a port has a ladder which hangs over the side. The length of the ladder is 200cm, the distance between each rung is 20cm and the bottom rung touches the water. The tide rises at a rate of 10 cm an hour. How long will it take for the water to reach the fifth rung?
   在港口停泊了一艘船，船側掛著梯子，長200厘米，梯級之間的距離是20厘米，最底層梯級接觸水面。潮汐每小時上升10厘米。須要多長時間，第五層梯級就會被水淹沒？
6. Car 1 started from Point A towards point B. At the same time Car 2 started from point B towards A. They crossed each other at point C. After which, Car 1 reached point B in another 9 hrs, and Car 2 reached point A in another 4 hrs. If both cars maintained a constant speed throughout the journey, and the speed of Car 1 was 36 miles/hr(mph), then what was the speed of Car 2?
   1號車從A點到B點。同時，2號車由B點到A點。兩車在C點相遇。之後，1號車用了9個小時到達B點，2號車用了4個小時到達A點。如果兩架車在路上的速度保持不變，而1號車的速度是每小時36 英里，請問2號車的速度是多少？

Answers 答案 : a, d, d, a,

Never. The tide raises both the water and the boat so the water will never reach the fifth rung.
永不。船和水面同時隨潮汐升起，梯子不會被淹沒。

54 miles/hr 每小時54英里

For detailed answers and explanations, please visit our website.
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