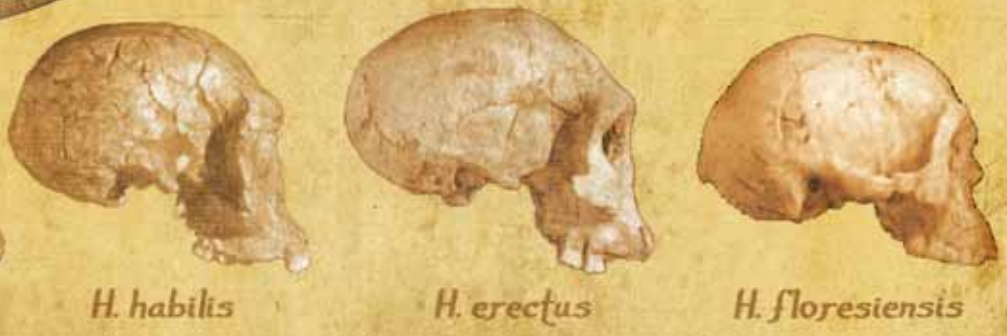
SCIENCE FOCUS 科言

Issue 011, 2017



School of 理學院 Science

香港科技大學
THE HONG KONG UNIVERSITY OF SCIENCE AND TECHNOLOGY

Contents

Science Focus Issue 011, 2017

Message from the Editor-in-Chief 主編話語

Dear Readers,

I hope everyone is getting some well-deserved vacation time this summer after a year of studying and examinations. This issue of *Science Focus* has particular emphasis on the roots of humanity and the history and science of our ancestors. Find out how our species fare in space and read about the science behind unboiling eggs.

I would also like to use this platform to congratulate our newest Science Focus Article Submission Competition winner, Natalie Si Yeung Yu, of King George V School (Year 12), for her fascinating article on the science behind human finger nails. You can read her winning article on page 21. If you are interested in science and writing, send us your submission to our next Science Focus Article Competition for the potential to have your article published in our magazine and website, and an opportunity to win an Apple iPad Air. Visit our website for more details at *http://sciencefocus.ust.hk*.

Enjoy the rest of the summer and don't forget to bring your copy of *Science Focus* to the beach!

Yours faithfully,
Prof. Yung Hou Wong
Editor-in-Chief

親愛的讀者：

希望各位同學在一年的辛勤學習和考試後，在這個夏天享受了應得的假期。這期「科言」聚焦在人類的根源，以及我們祖先的歷史和科學。我們還給各位同學準備了趣聞，談談人類在太空怎樣度過，還有逆轉熟蛋的科學。

我想借此機會祝賀英皇佐治五世學校 (12 年級) 的俞思扬同學 (Natalie Yu)，成為「科言」徵文比賽最新的得獎者。得獎作品講述人類指甲的科學，刊登在第 21 頁。若果你對科學和寫作有興趣，請投稿參加新一輪的「科言」徵文比賽，一經選中便會刊登在本雜誌及網站，並有機會贏得蘋果 iPad Air 乙部。若想了解更詳細資訊，請瀏覽「科言」官方網站：*http://sciencefocus.ust.hk*。

希望各位享受餘下的暑假，別忘記帶著你的「科言」到海灘閱讀！

主編 王殷厚教授
敬上

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Homepage: **http://sciencefocus.ust.hk**

WHAT'S HAPPENING IN HONG KONG ?

Zürich Meets Hong Kong – A Festival of Two Cities

This October, you have the opportunity to partake in *Zürich Meets Hong Kong*, where the two world-class cities join together to bring a week's worth of events and activities to different venues in Hong Kong.

蘇黎世與香港的約會

這個十月，你將有機會參加為期一週的「蘇黎世與香港的約會」，兩個國際都會攜手合作，在多個場地舉行不同活動。

A Virtual and Augmented Experience

You know those virtual reality headsets that are all the rage recently? Well, now you have a chance to experience the virtual reality of flying, animate your favourite cartoon characters, create art like a professional painter and more. Learn the science behind how virtual reality works and become part of the next generation of technology.

Location: Hong Kong Science Museum
Date: Wed 25 October 2017
Time: 7:00pm – 9:00pm

來自蘇黎世的虛擬及擴增體驗

你知道近期火熱的虛擬實境顯示器嗎？可曾夢想有機會像鳥一樣飛翔、賦予生命給你最喜愛的卡通人物、或像專業畫家一樣創作？來看看虛擬實境是如何運作，又是怎樣成為新一代的技術。

地點：香港科學館
日期：2017 年 10 月 25 日
時間：7:00 PM 至 9:00 PM

Augmented Reality, Creativity and the Arts

Onto more augmented reality! Technology and art combined creates something beautiful. Join Bryan Chung Wai Ching, Assistant Professor at Hong Kong Baptist University, and Robert Sumner, Associate Director of Disney Research Zürich to experience technology and art together.

Location: AAB201 Lecture Theatre, Hong Kong Baptist University
Date: Mon 23 October 2017
Time: 5:00pm – 7:00pm

擴增實境、創意和藝術

還有擴增實境！技術和藝術結合就能創造出美麗的東西。來與香港浸會大學助理教授鍾緯正，以及和迪士尼蘇黎世研究部的副總監Robert Sumner 一起體驗技術和藝術的互動。

地點：香港浸會大學曾陳式如會堂AAB201
日期：2017 年 10 月 23 日
時間：5:00 PM 至 7:00 PM

Save Orangutans at Hong Kong Ocean Park

Hong Kong Ocean Park and Zoo Zürich Switzerland will work together to save our beloved and endangered orangutans. But how does conservation work? In a guided tour, find out the "behind-the-scenes" of keeping endangered animals in modern zoos.

Location: Hong Kong Ocean Park
Date: Tue 24 October 2017
Time: 10:00am – 12:00pm

保育紅毛猩猩和海洋

香港海洋公園與瑞士蘇黎世動物園將會共同合作，拯救我們心愛的瀕危紅毛猩猩。想知道更多內幕，可以參加導賞團，了解現代動物園怎樣保護瀕危動物。

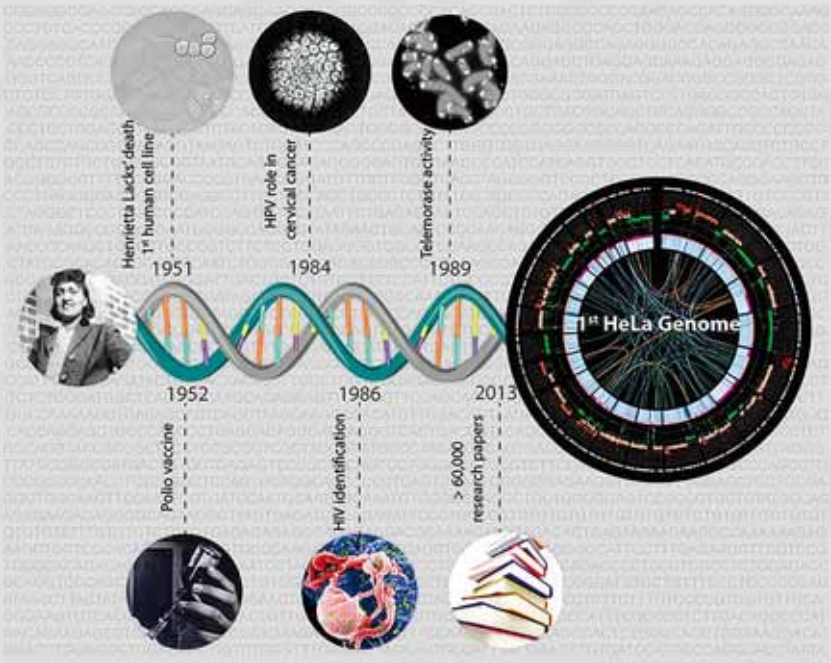
地點：香港海洋公園
日期：2017 年 10 月 24 日
時間：10:00 AM 至 12:00 PM

香港科技活動

Immortality may be far from possible, but some cells have been passed on for decades. Scientists call them immortalised cell lines for the simple fact that they can be grown for extended periods of time in a standardised laboratory setting. For many years, a number of immortalised cell lines were harvested from different tissues and species, revolutionising bioscience research on the study of gene function, toxin and medication testing, and the development of vaccines and antibodies.

The first and most well-known human immortalised cell lines came from a cancer patient called Henrietta Lacks. While she was pregnant with her fifth child, Lacks complained of a "knot" sensation in her abdomen. After giving birth, Lacks suffered from a haemorrhage (or a ruptured blood vessel). The bleeding was later diagnosed to be caused by a malignant cervical tumour. Lacks died in 1951, but unbeknownst to her, the cancerous tissue samples removed by her surgeon were sent to Dr. George O. Gey, a cell biologist at the Johns Hopkins Hospital. He noticed that the cancerous tissues were unique in that they were able to divide indefinitely in vitro from a single cell, something that he had never seen in the laboratory before. This became the first line of human cells to be isolated and immortalised, now known as the HeLa cell line [1].

Normal human cells undergo a process called cellular senescence, where repeated divisions cause the genetic materials to age and become unstable. In a laboratory setting, this happens typically after around 50 cell divisions in healthy human cells. The mechanism is in place to prevent abnormal or faulty cells to self-destruct. However, cancer cells do not experience senescence and will continue to divide. In fact, HeLa cells are particularly aggressive and care had to be taken to prevent them from contaminating other cells.



Since 1951, HeLa cells have made a significant impact in many areas of science and research.

Image credit: European Molecular Biology Laboratory (EMBL)/Jonathan Landry

Requests for HeLa were made by other researchers not long after Dr. Gey's publication, which gave rise to zillions of HeLa cells for scientific purposes. Since then, HeLa became a companion of researchers. Its impact on modern medicine is profound. For instance, the polio vaccine was tested on HeLa cells before going to human trial [1, 2]. Thereafter, HeLa became an instrument for studying the effects of viral infections for the invention of more vaccines. In the mid-1960s, HeLa was fused with mouse embryo cells to create the first

References 參考資料:

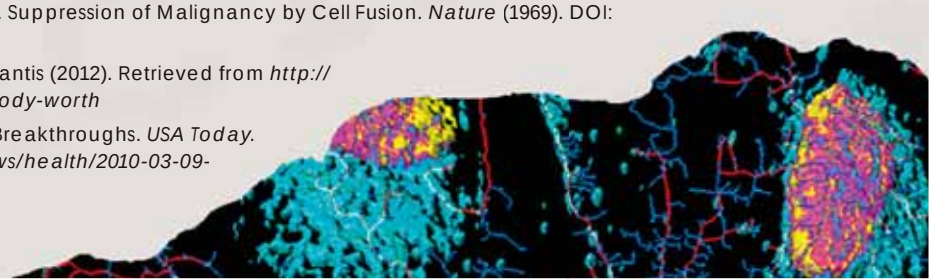
[1] Scherer, W. F., Syverton, J. T., Gey, G. O. Studies on the Propagation In Vitro of Poliomyelitis Viruses. *Journal of Experimental Medicine* (1953). 97(5): 695-710.

[2] Brownlee, K. A. Statistics of the 1954 Polio Vaccine Trials. *Journal of the American Statistical Association* (1955). Volume 50, Issue 272.

[3] Harris, H., Miller, O. J., Klein, G., Worst, P., Tachibana, T. Suppression of Malignancy by Cell Fusion. *Nature* (1969). DOI: 10.1038/223363a0

[4] Schulman, A. N. What is the Body Worth? The New Atlantis (2012). Retrieved from *http://www.thenewatlantis.com/publications/what-is-the-body-worth*

[5] Jones, B. How Henrietta Lacks' Cells Fueled Medical Breakthroughs. *USA Today*. Retrieved from *https://usatoday30.usatoday.com/news/health/2010-03-09-*



永生細胞 HeLa

The Immortalised HeLa

By Thomas Lee 李浩賢

永生或許是遙不可及，但有些細胞卻已流傳了數十載，科學家稱之為永生細胞系，因為它們可以在標準化的實驗室環境中長期生長。多年來，科學家從不同組織和物種獲得多株永生細胞系，為基因研究、毒素和藥物檢測、以及疫苗和抗體的開發，帶來革命性的變化。

首株也是最有名的人類永生細胞系來自一名癌症患者海瑞塔·拉克斯。當她懷有第五個孩子時，感到腹部有腫塊；產後血管破裂出血，診斷是子宮頸惡性腫瘤所致。拉克斯在1951年逝世。在她不知情的情況下，醫生把腫瘤樣本交給約翰·霍普金斯醫院的喬治·蓋伊博士。蓋伊博士是細胞生物學家，留意到這癌組織與別不同，細胞可以在體外無限分裂。他從未在實驗室見過這樣的細胞，稱之為 HeLa，是第一株可離體持續培養的人類細胞系 [1]。

正常的人類細胞經歷細胞衰老過程，因為重複分裂會導致遺傳物質不穩定。在實驗室中，細胞衰老通常發生在大約50次細胞分裂之後。這機制可以讓異常或有缺點的細胞不必自毀。不過，癌細胞卻不會老化，並且會持續分裂。HeLa 細胞系尤其富侵略性，要小心處理，免得污染其它細胞。

蓋伊博士的研究成果出版後，馬上有科學家求取樣本，結果有無數 HeLa 細胞用於科研。HeLa 從此成為研究員的夥伴，對現代醫學有深遠影響。 例如：脊髓灰質炎疫苗便是先用 HeLa 細胞測試，然後才在人體作試驗 [1,2]。其後，HeLa 用於研究病毒感染，開發出更多疫苗。至上世紀 60 年代中期，科學家將 HeLa 與鼠胚胎細胞融合，創建第一個雜交細胞，這對人類基因組圖譜的繪製大有幫助 [3]。目前，HeLa 廣泛應用於病毒和癌症研究、醫學診斷和毒理學。 值得一提的是，HeLa 是第一株送上太空的人類細胞系；它在外太空跟地球一樣，也能積極進行分裂 [4]。

可惜，拉克斯從未有機會同意提供細胞作醫學研究。她的家人對之後的應用也是不知情，直到研究員找到他們，希望了解 HeLa 細胞為何能夠如此積極地複制，甚至可以污染其它樣本 [5]。HeLa 細胞的應用觸發許多倫理議題，但毫無疑問，在過去數十年這些細胞拯救了數以百萬人的生命。

cell hybrid, which helped researchers in mapping the human genome [3]. At present, HeLa has been widely used in virus and cancer research, medical diagnostics, and toxicology. It was also the first human cell line to be taken to outer space, where it divided just as fiercely as it did on Earth [4].

Sadly, Lacks never had the chance to give consent for her cells to be used for medical research. Furthermore, her family was not informed of further application until researchers tried to contact them to find out why HeLa cells were so aggressive in their replication, to the extent of contaminating other samples [5]. While the use of HeLa cells has raised numerous ethical concerns, it is undoubtable that its existence has saved millions of lives for decades.

Double Act

Mankind's yearning to acquire knowledge of the vast unknown spaces of the universe has been a mission stretching back to antiquity. And when Earth inevitably succumbs to the test of time, it is to the great unknown we hold our hopes of survival. While we dream of grand interstellar voyages and the ultimate conquest of nature, understanding how our biology reacts to space travel is essential. Does the arduous task of space travel and zero gravity take a toll on our bodies?

In an attempt to further understand the effects of space travel on the human body, the National Aeronautics and Space Administration (NASA) initiated the Twins Study – a major research project encompassing ten separate researchers from diverse backgrounds, featuring multi-faceted collaboration between academia, government and industry.

This unprecedented project took Scott Kelly, a NASA astronaut, on an orbital journey between April 2015 and March 2016, while his twin brother Mark Kelly stayed on Earth for a ground-based control subject. Identical twins share nearly 100% of their genome, making them close to perfect test subjects for genetic and epigenetic research. Whether the stressors caused by zero gravity or other external stimuli from space travel trigger epigenetic changes will be more apparent with an exact genetic copy to compare to. The researchers were thus able to document any unique biological changes between Scott and Mark as a response of being in space. In addition, the 340 day-long investigation incorporated exciting new techniques from an array of emerging fields. For instance, genetic sequencing was employed in the establishment of individual molecular profiles, contributing to the development of personalised medicine.

Preliminary results were released during the Human Research Programme Investigators' Workshop in January 2017 and revealed several intriguing physiological phenomena as a result of Scott's stay at the International Space Station. Measurements before, during and after demonstrated notable divergences in DNA structure and gene expression patterns.

One structure that showed change was telomeres. Protective sequences of repetitive DNA situated at the ends of chromosomes, telomere shortening is a hallmark of aging and age-related diseases. While in space, the length of Scott's telomeres increased, but eventually dropped back to his pre-flight levels after he returned to Earth. The implications of these results are still yet to be determined.

Meanwhile, changes in the gene-expression signatures were reported. Where such changes are associated with the ever-shifting environmental factors, like diet and sleep pattern, the changes in Scott seemed more pronounced than expected, prompting the question of whether there may be the activation of a "space gene". It has, however, been hypothesised that gene expression could be affected by diet.

The challenge now is to untangle how many of the observed changes are specific to the physical demands of spaceflight — and how many might be simply due to natural variations. And because the Kelly twins are just

NASA astronaut Scott Kelly

雙胞胎行動

By David Iu 姚誠鵠

two people, the results may not be generalised to others. For the most part, the jury's still out, and exactly how the stress of space travel leads to such changes remains to be delineated. As NASA eloquently put it, "Each investigation is like an instrument. On its own, it plays solo music. But put them all together and you have something incredible".

Further results are expected to be published later this year, while integrated theme papers are due in 2018.

宇宙浩瀚無垠，人類自古以來就渴望探索這未知的領域。地球終有一日會崩壞，到時人類就只能把生存的希望寄託於穹蒼之上。在我們嚮往偉大的星際歷險和幻想征服大自然的同時，必須要了解人體對太空旅行的反應。艱鉅的太空旅行會否損害我們的健康？零重力的生活又會怎樣影響身體機能？

為了進一步了解太空旅行對人體的影響，美國太空總署啟動了龐大的雙胞胎研究計劃。有 10 位來自不同背景的研究員參與項目，突顯了官商學之間的多元合作。

在這項前所未有的研究中，美國太空人斯科特凱利於 2015 年 4 月至 2016 年 3 月期間在國際太空站生活，他的同卵雙胞胎兄長馬克則留在地球作為對照。同卵雙胞胎的基因組近乎完全相同，所以在基因研究和表觀基因學研究中大派用場。通過比對完全相同的基因組合，由零重力或其他外來刺激所觸發的表觀遺傳變化就會顯而易見。研究員因此可以肯定斯科特和馬克表現出來的不同生物變化，是受到太空旅行的影響。此外，這項為期 340 天的研究動用多個新興領域的科技，例如：以基因測序建立個人的分子譜，這將對個人化醫學的發展大有貢獻。

研究的初步結果已於 2017 年 1 月在太空總署舉辦的專題討論會上公佈，並披露了不少斯科特逗留在國際太空站時產生的有趣生理現象。比較在升空前、太空中和返回地球後的測量數據，不論是斯科特的基因結構還是基因表達的模式，均出現了明顯的變化。

端粒是其中一個出現變化的結構。端粒是處於染色體尾端的一段不斷重複的序列，作用是避免染色體受損。端粒縮短是衰老和老年疾病的一大特徵。斯科特在居留太空期間，端粒變長了，但在回到地表後，端粒長度又縮短至飛行前的長度。這些結果的意義還有待研究。

科學家們還發現了基因表達特徵上的改變。雖然基因表達的模式會隨著睡眠和飲食習慣等環境因素而不斷變化，斯科特的基因表達變化似乎比預期更為明顯，因此有揣測可能是因為啟動了「太空基因」。不過，目前的假設仍然是認為這些基因表達上的變化，是由於長期食用太空餐造成的。

現在主要的挑戰是要分辨出有哪些觀察到的變化是來自太空旅行，有哪些是純粹自然變化。畢竟凱利兄弟只是兩個人，難以把實驗結果直接套用於大眾。很大程度上，要對太空旅行的影響下定論還是言之過早。穿梭太空造成的壓力是如何導致這些變化，也是有待考證。正如太空總署所作的生動比喻：「每項研究就像一件樂器，各自彈奏；但湊在一起時，你又會得到不可思議的結果。」

進一步的研究結果將在今年稍後刊登，整合後的主題論文則會在 2018 年發佈。

Identical twin brother Mark Kelly

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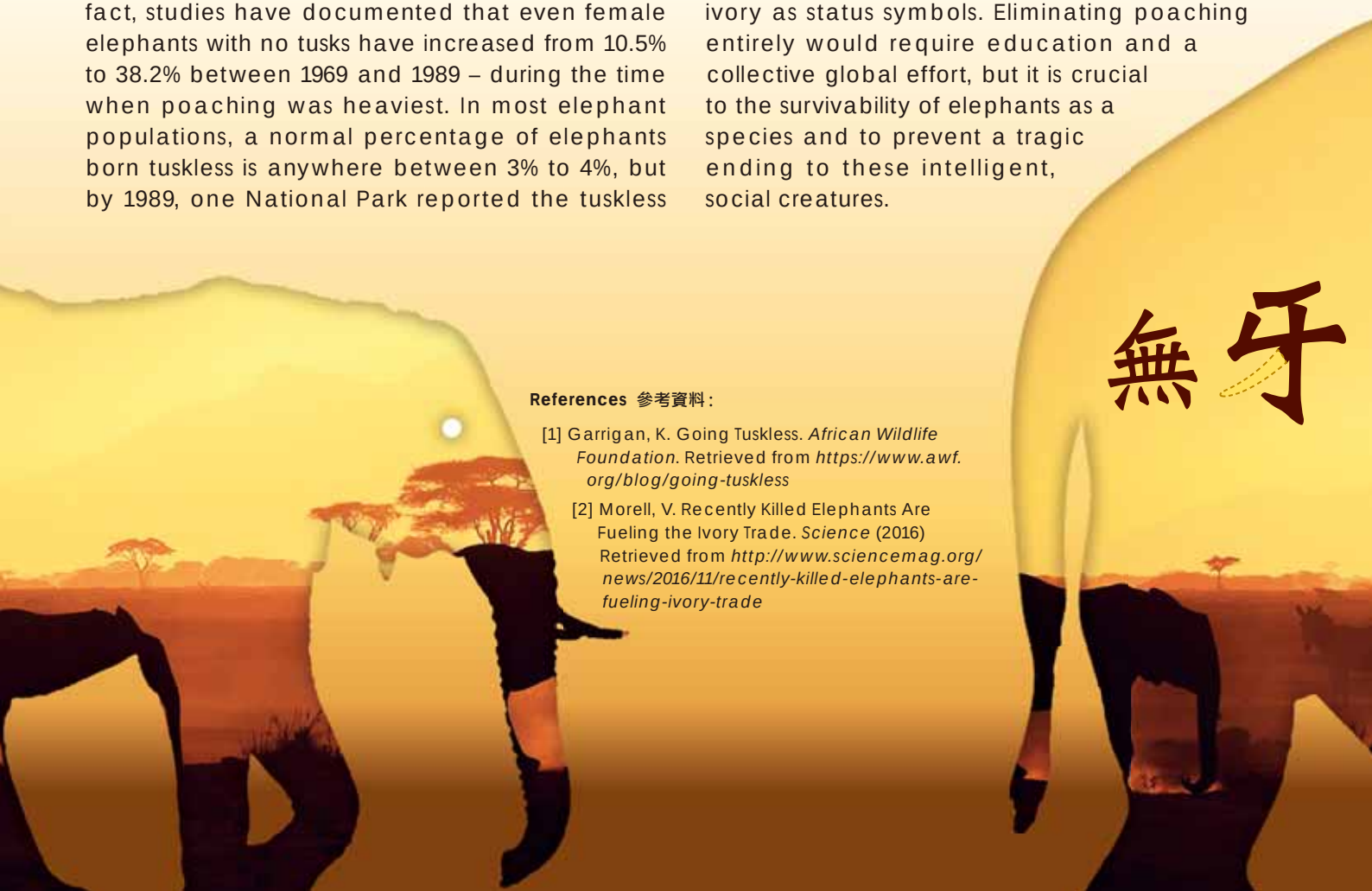
Elephants are often associated with their tusks. But these majestic biological structures are both a blessing and a curse. Highly sought for its ivory and poached to the point of endangerment, the number of African elephants born without tusks or born with smaller tusks has been increasing at an alarming rate, suggesting a selection disfavouring tusks.

The largest elephants typically bear the largest tusks, and in general, tusks grow as the elephant ages. Male African elephant tusks can grow up to seven times the weight of female elephant tusks [1]. Due to the fact that poachers tend to go after elephants with the largest tusks, male elephants are particularly targeted at their reproductive prime. Many of these elephants are therefore taken out of the population and can no longer pass on their genes related to the development of large tusks. Even if there are younger elephants that carry the genes for large tusks toward the line, either they are not ready to breed or their mating success is relatively low due to their smaller sizes. Poachers are artificially selecting for tuskless elephants, by eliminating large tusks from the gene pool.

Accelerated evolution of the tuskless elephant is not isolated in the African elephant alone. In fact, studies have documented that even female elephants with no tusks have increased from 10.5% to 38.2% between 1969 and 1989 – during the time when poaching was heaviest. In most elephant populations, a normal percentage of elephants born tuskless is anywhere between 3% to 4%, but by 1989, one National Park reported the tuskless elephant population to be between an alarming 9% and 25%.

The purpose of the elephant tusk is more far-reaching than an aesthetic feature. Tusks are specialised teeth that continue growing throughout an elephant's life. If a tusk is broken off due to natural circumstances, the tusk will keep growing, unless the root of the tusk is exposed, which leaves the elephant to a very slow and painful death. Elephants use their tusks to fight, dig or move things. They also have the function of protecting the trunk of the elephant when they charge and defending an elephant from predators, which increases its survivability. There is also the fact that female elephants prefer to mate with male elephants with tusks. Reduced mating could affect long term population size.

Despite the fact that the international ivory trade has been banned since 1989, illegal elephant poaching and ivory trafficking still run rampant in certain countries that lack the resources to battle poachers. Some surveys demonstrate that between 2007 and 2014, as many as 144 000 elephants were killed for their ivory [2]. Much of the demand of ivory stems from the nouveau-riche in Asian countries, who view owning items made of ivory as status symbols. Eliminating poaching entirely would require education and a collective global effort, but it is crucial to the survivability of elephants as a species and to prevent a tragic ending to these intelligent, social creatures.



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[2] Morell, V. Recently Killed Elephants Are Fueling the Ivory Trade. *Science* (2016) Retrieved from *http://www.sciencemag.org/news/2016/11/recently-killed-elephants-are-fueling-ivory-trade*

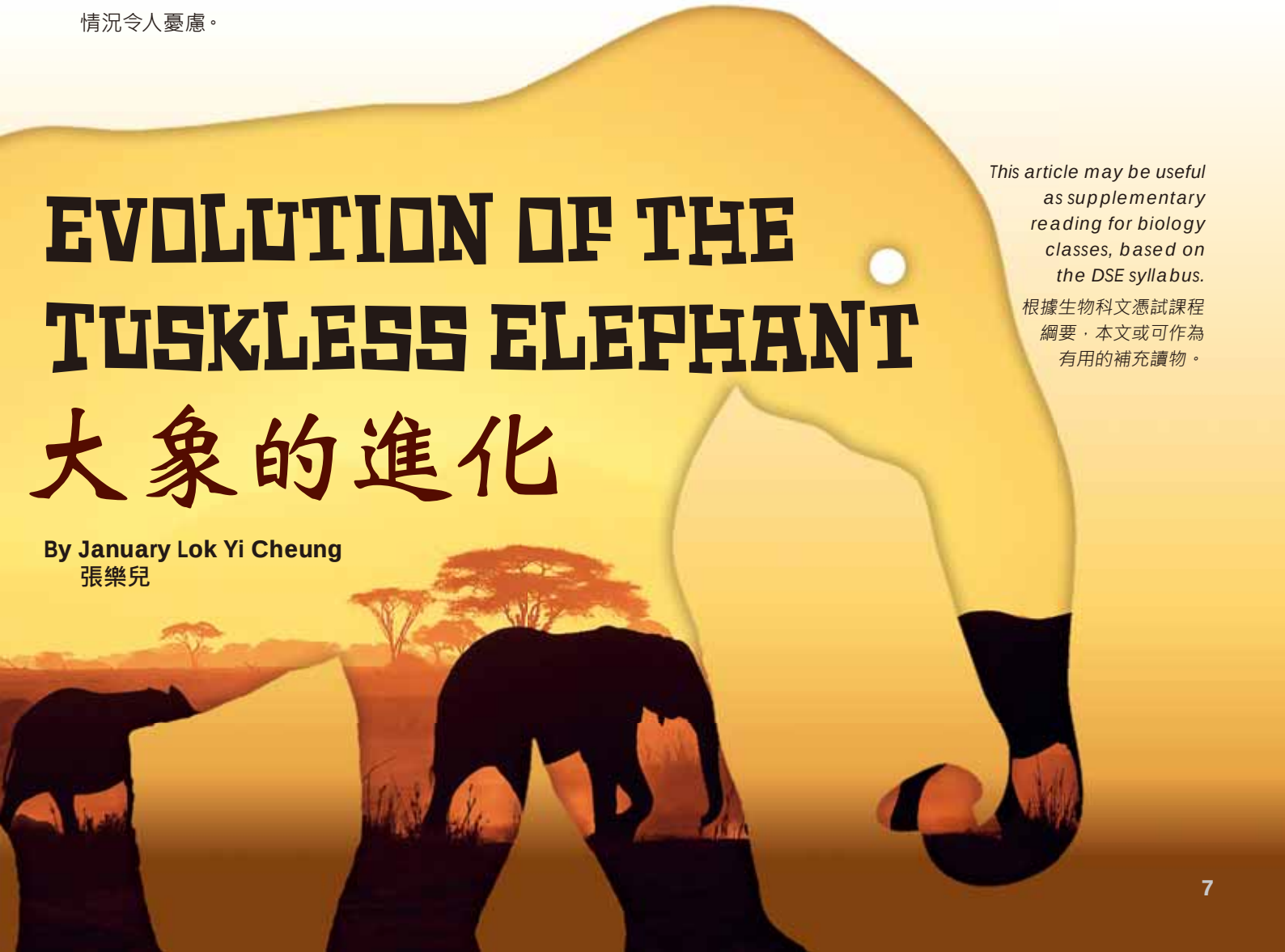
想起大象就往往想起牠們的長牙，這耀眼的生物結構既是祝福，也是詛咒。象牙需求殷切，以致非洲大象被非法獵殺至瀕危。天生沒有象牙或是象牙偏小的非洲大象，以驚人的速度增加，顯示象牙正在被淘汰。

最大的大象通常擁有最大的象牙。一般來說，象牙會隨著大象年齡而生長。雄性非洲象的象牙重量可達到雌象象牙的 7 倍 [1]。由於偷獵者傾向於捕獵有最大象牙的大象，在生殖期的雄性大象就成為重點目標，許多會因此從象群中消失，不能把大象牙相關的基因傳遞給下一代。即使有較年輕的大象攜帶著大象牙的基因，牠們還未成熟，或者是由於身型太小而難於成功交配。偷獵者將大象牙基因從基因庫剔除，人工選擇了無牙大象。

無牙大象的加速演化不僅僅發生在非洲大象中。事實上，研究指出在盜獵最嚴重的時期（1969 至 1989 年），無牙雌象也由 10.5% 增至 38.2%。在大多數的象群中，天生無牙大象的正常比率約在 3% 至 4% 間。但到了 1989 年，有國家公園報告無牙大象竟然在 9% 至 25% 之間，情況令人憂慮。

象牙的作用遠不止是為了美觀。象牙有特別的功能，一生都在不停生長，在自然環境中折斷後也能繼續。但若是牙根露出了，就沒法再長出來，大象也會非常緩慢而痛苦地死去。大象用象牙來打架、挖掏或移動東西。象牙還能在大象衝刺時保護象鼻，也能對抗大象的天敵，增強大象的倖存能力。此外，雌象偏好與有牙雄象交配。長遠而言，減少交配會影響象群的數量。

雖然自 1989 年國際象牙貿易已被禁止，但在某些缺乏資源打擊偷獵者的國家，非法盜獵大象和象牙販賣活動依然猖獗。一些調查顯示，2007 年至 2014 年期間，有多達 144,000 隻大象因為象牙而被殺 [2]。大部分的象牙需求來自於亞洲國家的暴發戶，他們認為擁有象牙製品是身份的象徵。要完全消滅盜獵活動，就需要教育和全球共同努力，這關乎到讓大象這種聰明的群居動物脫離厄運，物種得以延續下去。



EVOLUTION OF THE TUSKLESS ELEPHANT

大象的進化

By January Lok Yi Cheung
張樂兒

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

根據生物科文憑試課程綱要，本文或可作為有用的補充讀物。

MVP of 移民太阳

Extinction – the end of a species of biological organisms. An estimated five billion species that have ever existed on Earth are believed to be extinct. It happens for a variety of reasons, sometimes due to human interference, but also due to unforeseen natural circumstances. It can be marked by the death of the last organism of the species, but equally importantly is the quantity of the Minimum Viable Population (or MVP), denoting the smallest size of a population required to avoid extinction for a certain period of time [1].

Extinction of a species is a common event that is currently estimated to occur at a rate of one species per year. One of the factors that lead to a species' demise is stochastic perturbation, or random deviation of a system. These could be natural disasters for instance. An example of stochastic perturbation is in the extinction of the heath hen (*Tympanuchus cupido cupido*) [2].

Following a steady decline of the heath hen population during the early 1900s, conservation efforts rebounded the birds' population to about 800 by 1916. In the summer of that year, a forest fire destroyed their nests and habitat, compounded by unusually high predation. These two random factors dramatically reduced the population to 100-150 birds. Then, in 1920, a disease outbreak caused the population to dip below 100. By 1932, the last heath hen was gone.

A species cannot hold off extinction once its population goes below its MVP. With a stable population around or above its designated MVP, a species is thought to be able to ward off stochastic pertubations. What about humans? By some counts, there were only 10 000 of us at one point in time [3], but being the resilient species that we are, we have now rebounded to around 7.5 billion and growing. With limited resources on Earth, the question is – what is the MVP we need for sustained space travel?

According to University of Florida's Dr. John Moore, the number is 160 [4]. He obtained this number through a series of computer modelling he ran in 2002, setting the space travel period to 200 years and around 8 to 10 generations long. In fact, this model still held when extrapolated for 60 to 80 generations or approximately 2000 years. While that sounds low, history speaks otherwise. Clans of hunter gatherers, villages in pre-industrial societies and infantry groups in armies have been well-maintained by 150 to 180 people [5].

However, a more recent study by anthropologist Cameron Smith, reported a minimum of 10 000 people would be needed to weed out inbreeding and maintain genetic diversity. He mapped out the trajectories of five starting populations (150, 500, 2 000, 10 000 and 40 000) in their percent of genetic variation against the number of years, demonstrating that with a starting population of 150 people, inbreeding would cause a loss of over 80% of genetic diversity for a given hypothetical gene over 200 years. A starting population of 10 000 or more showed a high percent of variation, at close to 100% over at least 200 years.

"With 10 000", Smith says, "you can set off with good amount of human genetic diversity, survive even a bad disease sweep, and arrive in numbers, perhaps, and diversity sufficient to make a good go at Humanity 2.0." [6]

By Rinaldi Gotama
李嘉德

SPACE Travel 的最小族群數

滅絕 — 生物物種的完全消失。據估計，有 50 億種曾經在地球上存在的物種已經滅絕了。原因有多方面，有的是人為干擾，也有的是出於不可預見的自然環境。最後一位成員的死亡固然標誌著物種的滅絕，但同樣重要的指標是最小存活族群數（Minimum Viable Population, MVP），代表著物種族群要在一段時間內避免滅絕所需的最小族群數量 [1]。

物種滅絕經常發生，估計每年就有 1 種物種消失。導致物種滅亡的其中一個因素是隨機擾動或系統的隨機偏差，例如是自然災害。新英格蘭黑琴雞的滅絕便是隨機擾動的一個例子 [2]。

二十世紀初期，新英格蘭黑琴雞的數量漸漸下降，但由於保育工作，在 1916 年數量回升到 800 隻左右。同年夏天，牠們的巢穴和棲息地卻被森林火災摧毀，加上被捕食率異常的高。新英格蘭黑琴雞的數量因此而驟減至 100 至 150 隻。在 1920 年，一場疾病爆發導致種群數下降到不足 100 隻。直到 1932 年，最後一隻新英格蘭黑琴雞也死去了。

這個例子指出一旦種群低於 MVP，滅絕就不可避免。若能維持種群在其特定的 MVP 左右或以上，這物種就能夠免受隨機擾動。那人類呢？據說人口曾經只有 10,000 左右 [3]，但人類作為堅韌的物種，數目已經達到 75 億，而且還在繼續增長。不過，地球資源始終有限，假若要開展持續的太空旅程，我們需要的 MVP 到底是多少呢？

佛羅里達大學的約翰·摩爾博士認為是 160 [4]。他在 2002 年以一系列計算模型進行推算，將太空旅行的時間設定為 200 年，即是約 8 到 10 代的時間，得出了這個數值。事實上，將這模型延伸至 60 到 80 代，大約是 2,000 年，結論依然相同。雖然這數值似乎偏低，但歷史卻說明，150 至 180 人就已足夠支撐採獵者族羣、前工業社會村莊、以及軍隊步兵團 [5]。

不過，人類學家卡梅倫·史密斯最近發表的一項研究報告，指出至少需要 10,000 人方能排除近親交配所帶來的風險，以及維持遺傳多樣性。他繪製了起始數目不同的人口發展軌跡 (150、500、2,000、10,000 和 40,000 人)，說明遺傳變異的百分比如何隨著年數遞增而變化。結果顯示在 150 人的羣體中，一個假設基因的遺傳多樣性將會因近親繁殖而在 200 年後失去了 80%。至於 10,000 人以上的起始人口，卻最少能在 200 年之內保持接近 100% 的高度變化。

史密斯說：「有了 10,000 人，就可以有充盈的人類遺傳多樣性，即使是遇到疾病也能倖存；並且在到達目的地時，可以有足夠的多樣性，或許還有人數，成功繁衍人類 2.0。」[6]

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Unboiling An Egg?

By Twinkle Poon
潘晴

Boiling an egg is a chemical change that uses heat to permanently alter the proteins in an egg in a process called denaturing. In this process, chemical bonds are broken and new ones are formed, causing chains of proteins to unfold and reset. Unlike physical changes, most chemical changes are difficult to reverse, but for the first time, international chemists have cracked the challenge of "unboiling an egg" – or really, folding protein.

Proteins are made up of long chains of amino acids and have a variety of essential functions within living organisms. DNA replication, molecule transportation and various metabolic reactions are all processes that rely on proteins. Folding proteins are important to many industries, from chemical reactions to medical research, but is a difficult process because proteins come out "tangled". The same thing happens when an egg is boiled.

Gregory Weiss, a researcher at the University of California at Irvine and his team set out to untangle and fold the proteins of a boiled egg back into that of an unboiled egg. They separated the yolk from the egg white and boiled the latter for 20 minutes at 90 degrees Celsius to allow the proteins to become tangled clumps. The physical manifestation of the tangled proteins is the hard and solid appearance of the egg white. The chemists then added urea, the primary component of urine, to break down the proteins and rearrange them into liquid egg white form using a specialised machine known as the vortex fluid device. The device is key, as it uses the shear forces of stress to separate the entangled proteins and allow them to refold.

Weiss initially happened upon the vortex fluid device while visiting Australia's Flinders University. The device was designed for intricate chemical reactions, where molecules are placed in a liquid to be spun in test tubes. The centripetal forces from spinning these test tubes are then applied to molecules in a controlled manner. Weiss realised that the same thing could be applied to tangled proteins when he saw that the device was able to separate molecularly-thin graphene from a block of graphite.

But while one of the main proteins that make up egg white – lysozyme – refolded nicely using this technique, different proteins have different shapes and not all behave the same way, calling for an investigation of the procedures required for each type of protein. Researchers discovered that a protein produced by *E. coli* for instance would not refold. To encourage folding, they locked down one end of the protein to a bead, similar to that of a weight, reproducing the natural way of folding for the protein.

Aside from being a fun science experiment, the effort has noble goals. To begin with, this discovery has the potential to minimise the cost of cancer treatment. Pharmaceutical companies typically create cancer-associated proteins by cultivating them inside costly hamster ovary cells, because this method prevents manufactured proteins from "misfolding" into undesired shapes during formation. Possessing a method to re-mold proteins from common *E. coli* bacteria or yeast could cut down costs of cancer research substantially by circumventing the need to cultivate proteins in the aforementioned method. In addition, the traditional path of salvaging protein is lengthy, whilst "unboiling an egg" only takes a few minutes.

翻轉煮雞蛋

將雞蛋煮熟是一種化學變化，使用熱能永久性地把蛋中的蛋白質變成新物質。在這變性過程中，化學鍵斷裂及重組，導致蛋白質鏈伸展和重新折疊。與物理變化不同，大多數化學變化都是難以扭轉，但終於有化學家克服了「翻轉煮雞蛋」，也就是折疊蛋白質的挑戰。

蛋白質是由氨基酸長鏈組成，在活生物體內具有多種不可缺的功能，DNA複製、分子轉運和各種代謝反應都是依靠蛋白質才能進行。在多個產業領域，包括化學反應到醫學研究等方面，蛋白質折疊都是重要卻難於控制的環節，因為蛋白質會「糾結」在一起，就像雞蛋煮熟時一樣。

加州大學歐文分校的研究員雷戈裡·韋斯和他的團隊研究熟蛋內的蛋白質，能否解開糾結，重新折疊成生蛋的蛋白質。他們將蛋黃與蛋清分離，將蛋清在90攝氏度下煮了20分鐘，讓蛋白質變成糾纏的團塊。蛋清的硬實樣子其實就是蛋白質糾結的物理表徵。化學家跟著以尿液的主要組分尿素來分解蛋白質，再用特製的渦旋射流裝置將蛋白質重新排列成液態蛋清。關鍵是該裝置能夠使用剪切應力來分離纏結的蛋白質，讓它們可以重新折疊。

最初韋斯是在訪問澳大利亞福林德斯大學時，發現這渦旋射流裝置。該裝置是為了在旋轉試管中進行的複雜化學反應而設計，產生的向心力在受控的情況下，施加到試管內液體中的分子。當韋斯看到它能夠將石墨烯分子薄層從石墨分割下來，就意識到可以應用同樣的方法處理纏結的蛋白質。

這技術可以有效重構溶菌酶，即是組成蛋清的其中一種主要蛋白質；但是不同的蛋白質有不同的形狀，亦有不同的運作模式，所以每類蛋白質的重構步驟還有待研究。研究人員發現大腸桿菌產生的一種蛋白質就不會重新折疊。他們將蛋白質的一端鎖定到珠子上，好比附上重量，幫助蛋白折疊回復自然。

這項研究固然是一個有趣的科學實驗，但也有著崇高的目標。首先，這一發現有可能降低癌症治療的成本。通常製藥公司會在成本高昂的倉鼠卵巢細胞內生產與癌症相關蛋白質，以避免製造出來的蛋白質在形成期「錯誤折疊」成不理想的形狀。若能將從普通大腸桿菌細菌或酵母得到的蛋白質重構，就無須用上述方法培養蛋白質，大大降低癌症研究的成本。此外，要回收利用蛋白質，傳統途徑是很耗時的，「翻轉煮雞蛋」只需要幾分鐘便能完成。

Further Reading 延伸閱讀：

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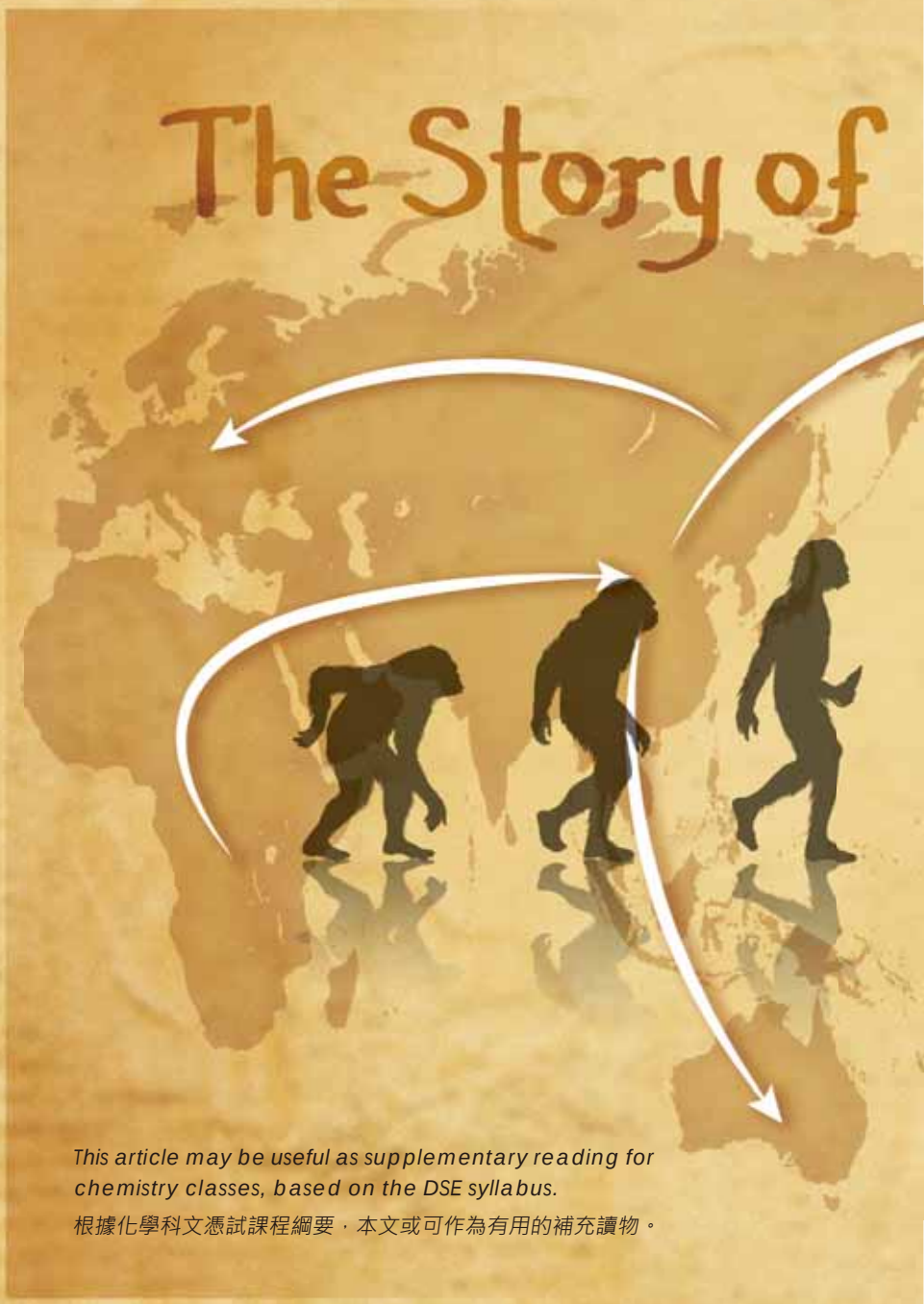
The Story of



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

根據化學科文憑試課程綱要，本文或可作為有用的補充讀物。

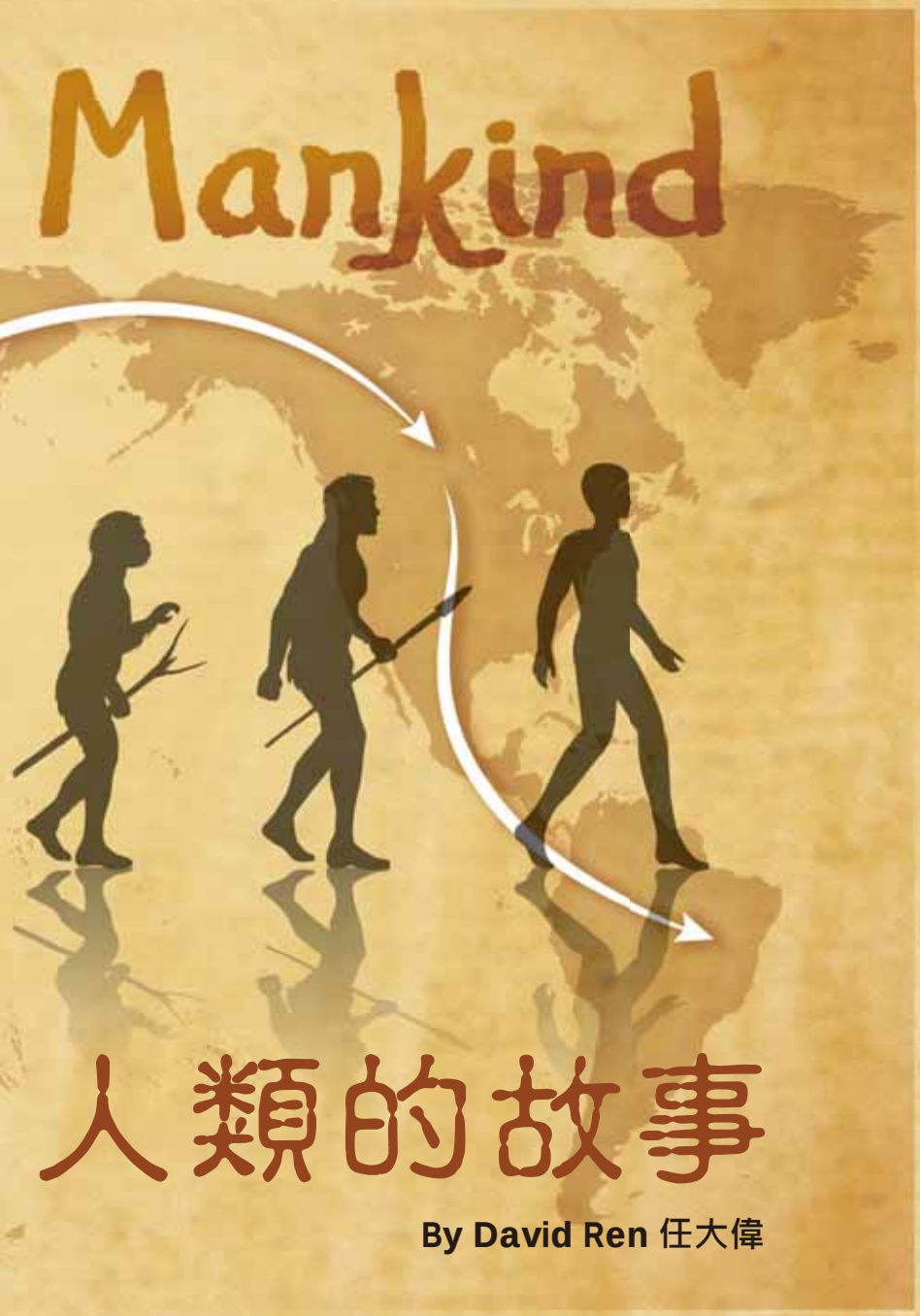
From a valley in Tanzania came a long-armed, hairy, ape-like creature whose genus would one day spread across the world. *Homo habilis*, or "Handy Man", marks a bold step forward for the evolution of modern *Homo sapien*. Their brain capacity of 550cm^3 to 687cm^3 [1] was far larger than that of their predecessors. Improved cranial capacity lent credence to their name, especially since *Homo habilis* are thought to have mastered simple stone tools to butcher and skin animals [2]. *Homo habilis* continued to thrive in previously uninhabitable territory until they disappeared around 1.8 million years ago.

During the same time period, a new species named *Homo erectus* became anatomically distinct to *Homo habilis*. Perhaps due to changing environments, *Homo erectus* developed shorter arms that allowed for easier upright walking. Their larger skulls (800 cm^3) and less robust skeleton suggested that *Homo erectus* depended on wit rather than brute strength. It was *Homo erectus* that first made the journey out of Africa. In around one million years, they spread into Europe, the Middle East and Asia.

The reason why early man left Africa is still disputed. Some suggest the population size approached the woodland carrying capacity (maximum population size that an environment can support indefinitely). Exploration of the savannah offered opportunities for a few. These few would evolve over time to become better suited toward traversing the open ground and so would *Homo erectus*.

What is indisputable is how widely spread the ape-men lived: from Africa to eastern Asia, skeletal remains of *Homo erectus* exist. One famous example is the 750 000 year-old Peking Man, found near Beijing. The excavation site became a battlefield as the Japanese invaded, so it was decided that all archaeological discoveries should be shipped away to New York for safety. No one knows whether a vehicle ever came to haul away the discoveries, but if it did, no one knows where the vehicle went. However, the Peking Man was photographed meticulously, leaving behind a detailed description of late *Homo erectus*. He was far heavier in build than early *Homo erectus*, a sign of evolutionary adaption to colder climates.

Dating archaeological artifacts proves rather difficult. It was not until the discovery of carbon dating could archaeological finds be dated accurately. The technique relies on a radioisotope of carbon – C14. Living matter contains a mixture of



人類的故事

By David Ren 任大偉

有一種來自坦桑尼亞的山谷，長臂、毛茸茸、像猿的動物，衍生的後裔遍佈世界各地。「能人」標誌著人類演化過程中的一大步，腦容量達到前所未有的 550 至 687 毫升 [1]。顱骨容量提升了，顯示他們是名符其實的「能人」。一般相信他們已懂得使用簡單的石頭工具屠宰剝皮 [2]。能人在原本不宜生息的地方艱苦奮鬥，直至約 180 萬年前才消失。

在同一時期，一種名為「直立人」的新物種出現，在解剖學上與能人明顯不同。或許是為了應付變化多端的環境，直立人的手臂較短，方便直立行走。他們的頭骨 (800 毫升) 較大，骨架較弱，說明直立人靠的是機智而非勇力。首先遷離非洲的是直立人。他們在 100 萬年左右的時間內，擴散到歐洲、中東和亞洲。

早期人類為什麼要離開非洲，仍然未有定論。有人認為，當時人口規模已接近叢林的承載力 (環境可以支援的最大人數)。少數物種如直立人在大草原找到了機會，之後隨著時間進化，有更大能力跨越開闊的地域。

可以確定的是猿人曾經遍佈大地：從非洲到東亞，都能發現直立人的遺跡。一個著名的例子是在北京附近發現的「北京人」，生活在距今大約 75 萬年前。在日軍侵華時，發挖地點成為戰場，當時為保安全決定將所有考古發現運往紐約。沒有人知道是否曾有車子來接載這些珍品，但即使有，也沒有人知道車子的下落。幸好北京人已被仔細拍攝下來，留下晚期直立人的詳細記錄。與早期直立人相比，北京人的體形健碩得多，應該是為了適應較寒冷的氣候而出現的演化改變。

要為考古遺物定年代其實是相當困難的，在碳測年技術出現後才能準確做到。這技術是利用碳的放射性同位素碳 -14。生命物質含有碳 -12(碳最豐富和穩定的同位素)和碳 -14; 生物體死亡後，碳 -14 隨放射衰變而減少。碳 -14 原子核中的中子，在釋放 β 粒子 (電子) 和反中微子後成為質子，碳 -14 蛻變成氮 -14。以蓋革計數器測量有機樣品的 β 放射活性，就可以確定樣品的年代。用這方法檢測在最近一次冰河期形成的冰川下的泥炭年代，得出的結果合符預期，由此可以確定碳測年技術的有效性。

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C12 (carbon's most abundant and stable isotope) and C14, but C14 radioactively decays after the organism expires. A neutron in the carbon nucleus will convert into a proton after releasing a beta particle (electron) and an antineutrino, resulting in an N14 atom. By using a Geiger counter to measure the beta decay activity of an organic matter sample, it becomes possible to date the sample. To verify the validity of this method, peat trapped under glacier was carbon dated. Since it was known that the glacier formed during the last ice age, the accuracy of carbon dating was confirmed.

To understand whether *Homo erectus* could communicate in a recognisably modern way, carbon dating is not sufficient. An excavation on the island of Flores in Indonesia revealed an adult skeleton no more than 106 cm tall. Initially, the specimen was declared to be *Sundathropus floresianus* but the relatively large size of its skull compared with its height convinced researchers to classify it as *Homo floresiensis*. It seemed that *Homo erectus* had managed to reach Flores and lost height to become *Homo floresiensis* [3].

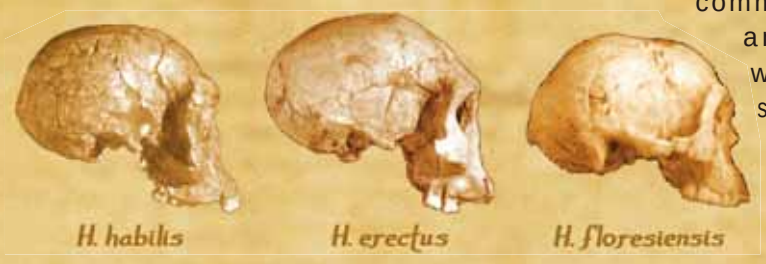
The island of Flores lies east of the Wallace line that marks the faunal ecospheres of South Asia and Australia. For most of history, the Wallace islands have been difficult to reach by land. For this reason, the evolutionary development of fauna only kilometres apart are vastly different [4]. The presence of humans east of the Wallace line 800 000 years ago suggests a great degree of cooperation and complex communication. Indeed, stone tools have been found on the island of Crete estimated to be 130 000 years old and on Flores since 800 000 years ago [5].

The loss of Flores Man's height is a surprisingly common occurrence for large animals confined to small environments. Smaller hominids require less food, have shorter gestation periods and have improved thermoregulation in tropical climates. The dodo experienced the reverse phenomenon – gigantism. Upon first arriving on Mauritius, the dodo was approximately the size of a pigeon. But after centuries without natural predators, the dodo grew larger; until it grew so large it lost its ability to fly [6].

Far away in sub-Saharan Africa, a distinctly modern ape-man was emerging. The new species, appearing approximately 150 000 years ago, displayed flatter faces, less muscular limbs and larger neural canals than *Homo erectus* or its predecessors. The developments reflected a preference for bipedalism and a greater capacity for communication. Two ice ages between 190 000 and 90 000 BCE pushed humanity to its limits. The dearth of fossil records during this period attest to the harshness of the environment. Some estimate that there were only 20 000 Homo sapiens alive in 100 000 BCE on the planet [7]. Adverse conditions encouraged genetic mutations to flourish, particularly those favouring higher intelligence. When climate conditions settled, humans with those genetic mutations were able to out-compete other ape-men. As the human homelands struggled with an increase in population, people migrated out of Africa via the land bridge at Somalia into the Middle East.

Civilisation began soon after the end of the last Ice Age at around 13 000 BCE. Within the "lucky latitudes", hunter-gatherers were able to begin to leverage a warmer planet. No place was better suited than the Hilly Flanks, incorporating the Tigris, Euphrates and Jordan Valleys in the Middle East. Desperate and hungry, people had deposited seeds into fertile soil and stayed year-round to ensure they grew into healthy crops.

Humanity spread to every corner of the globe in a relatively short period of time, overcoming multiple ice ages on the way. We have demonstrated our ability to adapt to new challenges like no other creature due to complex communication, cooperation and intelligence – and we continue to show this spirit as the challenges of the 21st century loom overhead.



人類的故事

不過，要知道直立人是否懂得用現代方法溝通，單靠碳定年技術是不足夠的。在印度尼西亞的弗洛勒斯島上發掘了一具身高不足 106 厘米的成人骸骨。最初，這個標本被誤判為獨立人種 *Sundanthropus floresianus*。但是與其高度相比，顱骨顯得太大，所以研究人員重新把他分類為「佛羅勒斯人」。看來直立人曾經到達弗洛雷斯，之後變得矮小而成為佛羅勒斯人 [3]。

弗洛勒斯島位於區分南亞和澳大利亞動物生態圈的華萊士線東面。長久以來，華萊士線兩側的群島難於從陸路到達，因此相距只有數十公里的動物群，也會經歷很不一樣的演化過程 [4]。但人類早在 80 萬年前便在華萊士線東留下蹤影，由此表現出高度的合作和複雜的溝通技能。在克里特島和弗洛勒斯島找到的石器工具，估計分別有 13 萬年和 80 萬年的歷史 [5]。

佛羅勒斯人的身高變矮，在狹小環境生活的大型動物經常會出現這種現象。身型較小的原始人類需要較少的食物，有較短的妊娠期，在熱帶氣候中更能有效地調節體溫。渡渡鳥卻經歷了相反的巨大化過程。渡渡鳥剛抵達毛里求斯時，身型跟鴿子差不多，但由於缺乏天敵，越長越大，甚至失去了飛行的能力 [6]。

在遙遠的撒哈拉以南的非洲地區，具有明顯現代猿人特徵的物種崛起。他們出現在大約 15 萬年前，與直立人或前人相比，面部輪廓較平坦，四肢肌肉較少，神經管較粗。這種種特徵顯示他們偏向以雙足步行，溝通能力更強。在西元前 19 萬年至 9 萬年之間的兩次冰河期，將人類推向極限。這段時期留下的化石記錄稀少，足證當時環境惡劣。估計在西元前 10 萬年，地球上的人口只剩下 20,000 [7]。惡劣的環境催動了基因突變，尤其是偏向產生高智商人類。在氣候緩和時，這些攜帶遺傳變異的人類淘汰了其他猿人。隨著家鄉人口增加，人們從非洲經過索馬里大陸橋移居入中東。

西元前 13,000 年，最後一次冰河期結束，人類文明旋即開始。在「幸運緯度帶」中，採獵者可以開始運用地球回暖所帶來的資源。滙聚了中東的底格里斯河，幼發拉底河和約旦河谷的「丘陵兩翼」地區，更是提供了安居樂土。窘迫飢餓的人羣將種子撒在肥沃的土壤中，守候整年以確保作物健康成長。

人類克服了多次冰河期，在相對短的時間內擴散到世界各個角落。我們掌握複雜的溝通技能、懂得互相合作，而且擁有高度智慧，所以比別的生物更能應對新挑戰。在面對 21 世紀所帶來的種種挑戰時，我們也能一如既往，迎難而上。

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Crystals, microscopically arranged in a highly ordered fashion of molecules, are common solid structures of many compounds and elements. The crystal's structure is distinguished by its unit cell, which consists of at least one atom within an imaginary cube, repeated and stacked in three-dimensional space in any direction. Snowflakes, for instance, are typically a single crystal or a conglomerate of several crystals. Solid sodium chloride, or more commonly known as table salt, is another example of a common crystal. Or perhaps most iconic is the giant covalent network of diamond. These tangible structures are easy to grasp, because they exist in space. Yet, in 2012, the same idea of a repeated structural unit in time was proposed by Nobel laureate Frank Wilczek, known as a time crystal.

These abstract structures would pulse without the need of energy input. Instead of a repeating unit of molecules that extend in all directions of a crystal lattice in space, a time crystal would theoretically consist

新物質狀態：時間晶體

A New State of Matter:

By Long Him Cheung 張朗謙

晶體是常見的固體結構，微觀分子排列高度有序。含有最少一個原子的單元晶胞，像方盒子般在三維空間中重複疊加，形成晶體結構。例子有雪花，它是單晶體或多個晶體的聚集物。 固體氯化鈉，即是俗稱的食鹽，也是一種常見的晶體。至於最具標誌性的例子，可能要算鑽石的巨型共價網狀結構。這些有形結構存在於空間之中，很易理解。在2012年，諾貝爾獎得主弗朗克．韋爾切克就以重複結構的概念，提出了「時間晶體」的設想。

這種抽象的結構無需輸入能量就能夠呈現週期性的運動狀態。普通晶體的結構單元往不同方向重複排列，而理論中的時間晶體則是在時間軸上重複同一模式，就像「不用上發條也能永遠行走的時鐘」[1] 。時間晶體與週期性振動還不一樣：時間晶體的振動是內在的特性，一般波動就需要靠外力驅動。要將韋爾切克的深奧構思付諸實踐，就要面對多方面挑戰。

of a pattern that repeats in time, akin to "a clock that ticks forever without being wound". [1] Furthermore, unlike a wave's periodic oscillations, time crystal oscillations would be intrinsic, whereas wave patterns demand a driving force. Wilczek's proposition to physically manifest this abstruse idea was met with several challenges.

Most fundamentally, the idea that a pulse could oscillate perpetually without initial energy input seems to defy the laws of thermodynamics. Symmetry in physics, for instance, means that the laws apply to all points in space and time, yet there are exceptions to the rules. At the ground state or its lowest energy level, a magnet will fall toward either north or south – that is they are asymmetrical since they look different on both sides. Crystals in space are also asymmetrical at their ground states, and do not look the same on all sides. However, regardless of symmetry, crystals in their ground states do not move unless given energy because by definition, that is where something is at its most stable state. Objects with asymmetry across time instead of space would then be considered as time crystals. The notion is counterintuitive because it is analogous to an object in its ground state moving around without added energy. For example, if one were to drop a marble in a bowl, the marble should intuitively come to an eventual rest, but instead the marble continues to roll around perpetually.

However, scientists from the University of Maryland and Harvard reported to have independently created time crystals, except neither party's time crystals fit the definition of what Wilczek initially proposed. Alternating lasers were fired at a chain of ytterbium ions so that the chains are constantly oscillating in random directions. What was significant was that even after the frequency of the initiations were changed, the oscillation did not change. Crystals in space are likewise stable to changes to their repeated structures.

There is still some debate as to whether this type of system is a time crystal as it fulfills time asymmetry but still requires some type of energy input to begin with. Scientists believe that the stability from these systems may have applications in quantum computing.

Time Crystals

最根本的問題是：在沒有輸入啟動能量的情況下，永遠重複的振動模式似乎違背了熱力學定律。基於物理學中的對稱性，這些定律應可適用於空間和時間上的每一點，不過也有例外。磁鐵在基態或最低能量的狀態時，會指向南或北方向，所以兩邊看來不一樣，也就是不對稱。處於基態的空間晶體同樣是不對稱的，從不同方向觀察的話會看到不同的面貌。然而，無論對稱與否，除非得到足夠能量，處於基態的晶體都不會移動 ，因為基態就是最穩定的狀態。在時間而非空間中擁有不對稱性的物體，可以被視為時間晶體。這個概念與常理背道而馳，因為這意味著處於基態的物體，可以在沒有增加能量的情況下移動；等如說丟進碗內的一顆波子不斷滾動，卻不會停息在碗底。

然而據報導，馬里蘭大學和哈佛大學的研究團隊已分別製造了時間晶體，只是雙方的成果都未能完全符合韋爾切克最初所提出的定義。科學家以激光交替射擊由鐿離子構建的鏈條，讓鐿離子鏈在隨機方向不斷振蕩。重點是即使改變了脈衝的頻率，鐿離子振動的頻率也能保持不變。這就跟空間晶體一樣可以維持穩定的重複結構。

這類系統能否稱得上是時間晶體，仍然存在爭論。雖然它滿足了「時間不對稱性」的條件，但還是需要輸入能量才能啟動。科學家相信這些系統的穩定性或許可以應用在量子計算中。

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Missing continent: houses 20 million people, last seen 5 feet to the south. Notice any problem? Yes, the title is misleading and the adjustment is actually quite small compared to Australia's gigantic size of 8,600,000 square kilometres. Though, putting in perspective how points are projected on the Global Positioning System (GPS), how the continent moves does affect the accuracy of locating someone, or impair the ability to differentiate between two neighbouring places. As Australia continues on its journey to the north, the GPS would be due for a major adjustment once every couple of years – and the next one is scheduled at the end of this year.

The GPS is basically a giant map of our three-dimensional world. That is, we can know precisely where everyone, everything, every place is by simply obtaining three numbers – the x, y and z coordinates (or r, θ, φ in polar coordinates). These numbers form the basis for a global positioning system; by treating the world as one giant grid of coordinates; one can assign numbers to each and every specific place, thus annotating their respective positions.

If you would like to know more about the physics of the GPS, please read Science Focus Issue 10 "The GPS and Its Connection to Relativity".

Cartography, the study of maps and pinpointing locations, is a delicate art. Maps are but static projections of our dynamic, ever-changing world. Every once in a while, the sets of reference points used to locate places on the coordinate system have to be updated. These reference points constitute a geodetic system which approximates or defines geographical distances, i.e. the latitude, longitude and altitude relative to these reference points, and make up the virtual framework on which we overlay the real world on. Usually, these reference points are major static structures such as roads and electrical grids, surveyed relative to other existing reference points. However, this implies that densely populated areas would be much better mapped than sparsely populated ones – in fact, Alaska is only as well mapped as Mars in this sense! [1]

Now, back to Australia. The current geodetic datum for the land down under was formalised way back in 1994. Updates for these datums have been sporadic – Australia has only ever seen three renditions of geodetic datums. Any further changes to the grid matrix of locations are based on the 1994 datum, minor edits and marks on a stationary canvas. The thing is, all continents are essentially tectonic plates floating around, colliding and sliding against each other – land masses shift over time, and Australia is no different. Except, Australia's movement has exceeded that of other continents due to its unique geology. The last update for the geodetic datum, i.e. the 1994 one, corrected its latitude and longitude by a staggering 656 feet.

"Some countries are more stationary than others. When there is a significant shift in land masses over time we need to revise the models of the Earth from which GPS coordinates are calculated, so for example your neighbour doesn't end up with your old coordinates," said Damien Saunders, Director of Cartography for National Geographic.

Aside from the adjustment set for the end of this year, plans have been made for a modernised datum to be introduced by 2020, in order to keep up with the pace of advancements in mapping technologies – which is probably way faster than the rate at which Australia drifts. It's not that Australia is drifting away so fast the GPS couldn't keep up – we know where it is, we are just constantly pushing the very boundaries of precision and accuracy.

失物啟示:2,000 萬人的家園,最後一次出現偏離南方 5 呎。留意到有甚麼問題嗎?沒錯,文章標題確是有點誤導;跟澳洲 860 萬平方公里面積相比,這誤差算不了什麼。不過,從全球定位系統(GPS)的運作原理出發,這大陸漂移確實會影響定位的準確度,也會妨礙系統分辨兩個鄰近地點。澳洲不停向北漂移,GPS 每隔幾年就得好好調校,下一次更新將會在今年年底進行。

GPS 基本上就是一幅巨型的三維世界地圖;也就是說,我們只要得到 3 個數字,即是座標 x、y、z(或者是極座標的 r、θ、φ),就能知道任何人、物件或地點的準確位置。將地球表面劃分為許多網格,再給每個具體地方一組數字,就可以標示相對位置,建立全球定位系統。

若讀者希望更深入了解 GPS 的物理原理,請閱讀第 10 期「科言」的「全球定位系統與相對論」。

地圖學是研究地圖與定位方法的科學,也是精妙的藝術。地圖是變化無休的世界的靜態投影。每隔一段時間,便要更新座標系統中用於定位的參照點。這些參照點組成「大地測量座標系統」,是反映真實世界的虛擬架構,提供相對的經

緯度和地平緯度 (高度) ，作為計算地理距離的依據。通常這些參照點都是鮮有變動的大型結構，例如道路、電網等，與其它參照點的關係經過勘量。不過，這就意味著在人口稠密地區繪製的地圖要準確得多。事實上，目前阿拉斯加的地圖質素才剛及得上火星 [1] ！

回歸正傳，究竟本文主角澳洲的情況是怎麼樣的呢？澳洲的大地測量基準資料只是偶爾更新，至今才重新詮釋了 3 次，現在的版本自 1994 年起便沿用至今。後來出現的網格矩陣變化都是以 1994 的數據為基礎，就像在定格的畫布上補上幾筆而已。可要知道，所有大陸其實都是浮動的板塊，這些板塊互相碰撞和摩擦，所以陸地會隨著時間演變。澳洲亦不例外，並且因地質獨特而移動較快。澳洲在上次即 1994 年調整大地測量基準時，就把經緯度大幅校正了整整 656 呎。

國家地理雜誌的製圖部主任桑德斯說：「有些國家的位置相對穩定。陸地移位隨著歲月而變得明顯時，我們就必須修正這些用以計算 GPS 座標的地球模型。不然，你原本的座標可能就會指向你的鄰居。」

除了在今年年終進行的調整，澳洲政府亦計劃在 2020 年推出一套現代化的測量基準，以追上製圖技術的發展；當然，這方面的發展速度遠比澳洲漂移快。澳洲的確是在漂走，但 GPS 還是能跟得上。我們清楚知道澳洲在哪裡，只是要追求更高的精準度。

全球定位系統能跟上澳洲飄移嗎？

Could Australia Be Drifting Away So Fast That GPS' Can't Keep Up?

By David Iu 姚誠鵠

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We Need Our Nails To Grow, So STOP Biting Them!

You have most likely seen a friend whose nails are uneven and jagged stubs, or perhaps you have them yourself. Onychophagia is the compulsive act of biting one's nails. It is surprisingly prevalent at young ages, around 45% of adolescents are nail-biters, and it may become a lifelong habit [1]. Onychophagia is also identified as an obsessive-compulsive disorder (OCD). For long-term nail biters, to rid of this compulsive behaviour, a 2012 study suggested that becoming aware of the need to rid the habit is one of the first steps [2]. So, to fellow nail biters, friends and families supporting our kind, let us understand why our nails are gifted to us and what biting them might incur.

Our nails have several important functions, including protecting the delicate phalangeal tissue and allowing for precise mechanical movements. Fingertips have high innervation density and are very sensitive. Our fingernails contribute to this feature by providing a counterforce to the fingertips when the finger's skin is touched, which heightens two-point discrimination [3]. Nails also enhance the precision of the pincer grip, the finest grip that humans can muster [4]. So how do these useful appendages grow and how can onychophagia stunt this?

The visible nail or the nail plate is composed of keratin, a fibrous protein which gives the nail its translucent nature. This plate is held down to the nail bed by the proximal nail fold (the skin around your nail) and the eponychium (the cuticle) [5]. The growth of the nail begins at the nail matrix which is part of the nail bed. The cells near the matrix replicate and undergo enlargement. The nail folds limit the direction of growth and the duplicated cells grow distally toward the nail plate. The newly formed cells flatten and elongate from the resistance of the established nail [6]. Eventually, the nail plate is pushed out forming the free edge of the nail.

Nail-biting can cause irreversible damage to this growth process. By removing the free edge of the nail plate and exposing the distal nail bed to the environment, the nail bed can "irreversibly disappear" thereby permanently shortening the nail plate [7]. With less of a free edge, not only is a smaller area of the finger protected, but precision manoeuvres are impeded. Onychophagia can also increase the risk of onychomycosis, a fungal infection of the nail, which can stunt nail growth and contribute to the destruction of the nail bed or the matrix [8]. With such deleterious effects, compounded by the unhygienic factor, it is hardly surprising that a social stigma exists for nail-biting.

For young children, when onychophagia has yet to develop beyond occasional biting, we can make use of biological responses and use topical bitter concoctions to discourage biting [2].

Research suggests that incentive is a useful remedy, and can be particularly effective for older children. But when all else fails and no amount of instinctive responses or information gathering can strike the habit, stigmatisation may be the only way left.

Let me end with a confession and the unfortunate irony of the nail-nibbling that occurred when I wrote this article. I am beyond the age of relying on biological responses, and I have learnt (even written) about onychophagia. I guess I have no choice but to ready myself for the only other option now: come my way, social stigma!

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

要長指甲啊，不能再咬了！

By Natalie Si Yeung Yu 俞思扬
King George V School, Year 12

你很有可能見過朋友的指甲呈不均的鋸齒狀，甚至你自身的指甲也是這模樣。咬甲癖是啃咬指甲的強制性行為，在年輕人中出奇地普遍，大約 45% 的青少年有這怪癖，且有可能成為終身的習慣 [1]。咬甲癖已被認定是一種強迫症。2012 年的一項研究顯示，要擺脫這種強迫行為，首先要意識到有這個需要 [2]。所以，讓我們這些咬甲者、以及支持我們的親友們，看看我們的指甲有多重要，咬指甲又會帶來什麼後果。

我們的指甲有幾個重要功能，包括保護精巧的指骨組織和允許精確的機械活動。指尖的神經分佈高度密集，非常敏感。指甲在指尖皮膚被觸碰時生出的反作用力，可以提升兩點辨別覺 [3]。指甲也能提高手指鉗夾物體的準繩，這是人類能掌握的最細緻動作 [4]。那麼這些有用的附屬物是如何生長的？咬甲癖是如何阻礙指甲的生長？

指甲的可見部分或甲板是由一種半透明的纖維蛋白，即是角蛋白組成。甲板被近端甲皺（指甲周圍的皮膚）和甲上皮（角質層）包圍，與甲床緊貼在一起 [5]。指甲是從位於甲床的甲母基開始生長，在甲母基附近的細胞複製和擴大。甲皺限制了生長方向，複製的細胞只好朝向遠端的甲板生長，新形成的細胞在遇到指甲攔阻時就會延展開來 [6]，甲板最終亦會被推出邊緣。

咬指甲可以對這生長過程造成不可逆轉的損害。除去了甲板的邊緣就會將甲床的遠端暴露出來，讓甲床不可逆轉地消失了，甲板也會永久地縮短了 [7]。指甲邊緣減少，受保護的手指面積也會減少，精準的動作也受到影響。咬甲癖也可增加患甲癬的風險，妨礙指甲生長並會破壞甲床或甲基質 [8]。基於這種種害處，加上衛生考慮，咬指甲自然會被污名化。

對於咬甲尚未成為癖好的幼童，我們可以利用生物反應，使用有苦味的外塗混合劑來阻攔偶爾出現的咬甲行為 [2]。研究指出獎勵措施很有用處，對大齡兒童尤其有效。但是，當用盡所有招數，即使是本能反應及資訊都不能打破習慣時，恐怕只有靠社會壓力。

最後我要承認在寫這篇文章時，我其實是在不停地咬指甲。我早已過了跟隨生物反應的年齡，也已清楚（甚至寫下）關於咬甲癖的種種問題。看來我別無選擇，只能接受社會標籤了！

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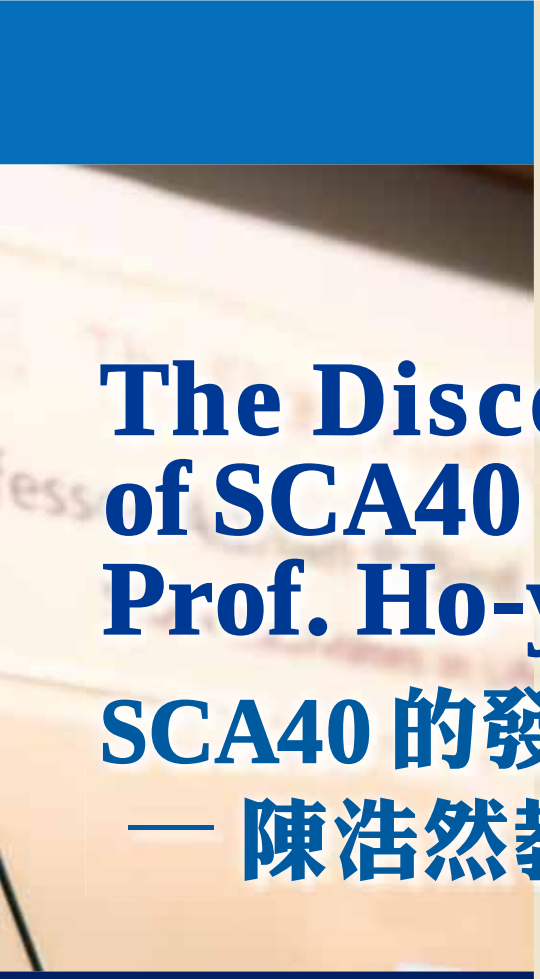
Genetic diseases, caused by abnormalities in a person's genome, are typically extremely difficult to treat, and many are incurable. Spinocerebellar ataxia (SCA) is one of them. It is a degenerative disease that eventually strips patients of their ability to coordinate movement. There are numerous types of SCA, with each type affecting a different gene and manifesting into a specific set of symptoms. Physiotherapy is often prescribed but only relieves symptoms to a certain degree. SCA40 was discovered by Prof. Edwin Ho-yin Chan during a three-year study of the genetic sequence of SCA patients in Hong Kong. He is Professor of Life Sciences at The Chinese University of Hong Kong and Director of the Laboratory of Drosophila Research.

In the latter part of the 90s, it was nigh impossible to identify genes that caused genetic diseases. The gene mutation responsible for causing cystic fibrosis, for instance, was not discovered until 1988 by Francis Collins, Lap-Chee Tsui and John R. Riordan. As a high school student and college freshman, Prof. Chan aspired to follow in their footsteps. In his junior year in college, Prof. Chan met his mentor, Dr. Sandy Luk, who urged him to first understand the biology of normal individuals before investigating pathological conditions. Equipped with his mentor's sage advice, he began studying developmental biology on the fruit fly, *Drosophila melanogaster* and conducting research which helped explain how genes control an organism's egg development, as many genetic diseases unfold from young. Thus began his commitment to biomedical research.

Prof. Chan's life's work began in 2002 after he had returned to Hong Kong from University of Pennsylvania, bringing with him the dream to dedicate himself to the SCA patient society. Together with the Hong Kong Spinocerebellar Ataxia Association, scientists, neurologists and genetic counsellors, they created a spinocerebellar ataxia patient registry that compiled patient data. It was during this period that they came across the SCA40 family – where five individuals were diagnosed with adult-onset spinocerebellar ataxia. Using genetic and biochemical analyses, Prof. Chan and his team were able to isolate the mutation in a gene called CCDC88C in these patients. Carriers of this gene mutation will develop SCA40.

Identification of the mutation is pivotal to developing better treatment and a potential cure to SCA40. "We take a structure-based drug design approach to develop inhibitors that can neutralise RNA-toxicity in SCAs. Based on the toxic RNA structure, we virtually fit molecules to the RNA and attempt to find one that binds tightly to the toxic RNA," said Prof. Chan, "After we identify such molecule(s), we then synthesise these compounds in the lab and then test them in our disease models, including iPSC, *Drosophila* and mice".

Parallel to the research on SCA40, Prof. Chan and his team also investigate other neuromuscular diseases, including myotonic dystrophy and amyotrophic lateral sclerosis (ALS). The latter garnered viral attention in 2014 in an activity called "the ALS Ice Bucket Challenge", aiming to bring awareness and donation toward ALS research.



The Discovery of SCA40 with Prof. Ho-yin Chan

SCA40 的發現 — 陳浩然教授

By Teresa Ming Shan Fan
樊銘姍

人類基因組異常引起的遺傳性疾病通常極難治療，有許多甚至是不治之症，小腦萎縮症便是其中之一。小腦萎縮症是一種退行性疾病，最終會剝奪患者協調動作的能力。有很多種小腦萎縮症，每種影響不同的基因，並表現出一組特定的症狀。通常會以物理治療處理，但只能在一定程度上緩解症狀。在一個為期三年的研究中，陳浩然教授比對香港小腦萎縮症患者的基因序列，發現稱為 SCA40 的小腦萎縮症。陳教授是香港中文大學生命科學學院教授，亦是果蠅研究實驗室主任。

在 90 年代後期，要確定導致遺傳疾病的基因幾乎是不可能的；例如，一直到了 1988 年，才由法蘭西斯．柯林斯、徐立之和約翰．賴爾登，發現造成囊腫性纖維化的基因突變。陳教授在高中求學及初入大學時，嚮往追隨他們的腳步。在大學三年級時遇到啟蒙導師 Sandy Luk 博士，勸勉他在研究病理之前應先要了解正常的生物學狀態。他銘記導師的忠告，開始研究黑腹果蠅的發育生物學，和卵發育的基因調控機制，因為有許多遺傳疾病都是從小出現。從此開始了他的生物醫學研究旅程。

陳教授的職涯始於 2002 年，當時他從美國賓夕法尼亞大學帶著抱負返回香港，矢志為小腦萎縮症患者謀福。他和其他科學家、神經內科醫生和醫學遺傳學家，與香港小腦萎縮症協會合作彙編患者資料冊。期間遇到患有 SCA40 的家庭，其中 5 人被診斷患有成人發病型小腦萎縮症。陳教授和他的團隊利用遺傳和生物化學分析，發現在名為 CCDC88C 的基因上出現的變異，帶有這種基因突變的人就會患上 SCA40。

識別突變是開發更好的治療甚至是根治 SCA40 之法的關鍵。陳教授說，「我們採取「基於結構的藥物設計方法」來開發可以中和小腦萎縮症 RNA 毒性的抑製劑。我們把不同的分子與有毒 RNA 的結構作虛擬配對，試圖辨識最能與有毒 RNA 緊密結合的結構。找到這樣的分子後，我們就可以在實驗室合成這些化合物，然後以誘導性多功能幹細胞、果蠅和小鼠等不同的疾病模型作測試。」

除了 SCA40 的研究，陳教授及其小組還探討了其他神經肌肉疾病，包括肌強直性營養不良症和肌萎縮性側索硬化症(ALS)。後者在 2014 年的「ALS 冰桶挑戰」中獲得廣泛關注，該活動的目的就是要提高對 ALS 的認識及為相關研究募捐。

Prof. Chan insists that creativity, paying attention to unobvious phenomena and perseverance are the keys to being a successful scientist.

He also suggested, "Work hard, play harder!"

陳教授堅信創造力、留意不起眼的現象和毅力都是科學家成功的關鍵。

他還建議：「努力工作，盡情玩樂！」

Photo credits: Yale Medicine

Many myths shroud the rhinovirus, the culprit responsible for causing the common cold, including the set of circumstances in which the virus transforms from being dormant to causing infections in humans. We know surprisingly little about this common virus and attempting to set right the old wives' tale on staying away from cold weather to avoid getting sick, is more complex than it seems. Yale University's Dr. Ellen Foxman and her research team investigate this very problem, exploring the mechanisms in which the rhinovirus manifests itself into symptoms and the body's defense in response to infection.

Dr. Foxman's research career began as an undergraduate, where she received a wide spectrum of research experiences. "During my first summer in college I did field research on Cliff Swallows, birds that migrate from the Midwestern United States to South America and back every year. The next summer, I was a research assistant in a virology lab, and the next year I worked in a yeast genetics lab", she said. These experiences paved the path for her future career in medical research.

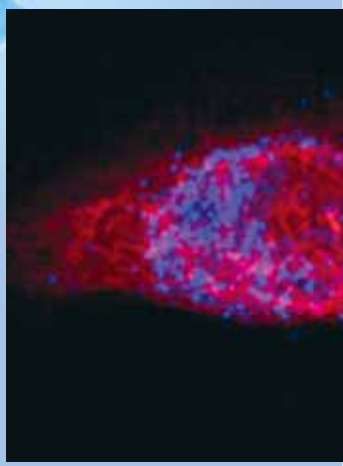
By the time she was in her medical residency, the normalisation of the use of polymerase chain reaction (PCR) to detect viruses helped to show that viral infections occur much more frequently in patients than had been previously thought. "In fact, the data that emerged from use of these techniques revealed that certain viruses visited the body so often that they could be considered part of the microbiome", said Dr. Foxman. However, as common as viruses were present, only half of the infections cause diseases and symptoms. "I became very interested in how variations in host-virus interactions affect the outcome of common viral infections". This intrigue fuels her current research on the topic.

Several factors enhance the replication of the rhinovirus; one of which is temperature, and whether the rhinovirus replicates more readily in cooler environments. Previous observations have suggested that many strains of the virus do become more virulent at temperatures of 33 – 35 °C in the nasal cavity, as opposed to an average body temperature of 37 °C. Dr. Foxman and her group discovered with compelling evidence that at a lower temperature, the defense response of infected cells are compromised, thus allowing the virus to become more infectious. Other factors that lower the defense response of cells include patients who have asthma, but the mechanism is not yet understood.

Hong Kong's flu seasons are marked with seas of mask-donning citizens in an effort to curb the spread of viruses. While masks act as barriers for airborne germs and viruses, Dr. Foxman believes there may be a possibility that warming up the nasal airway by wearing masks could also be a factor in extra protection against infection.

"The best strategy to prevent symptoms is probably to prevent replication from happening in the first place, which is what our airway epithelial cell defence mechanisms aim to do".

Meanwhile, Dr. Foxman and her team will continue to study and uncover the mechanisms that change the way our airway defence systems deal with viruses. A deeper understanding of these mechanisms is essential to the development of better strategies for the prevention of ubiquitous infections such as the common cold.



鼻病毒與 Ellen Foxman 博士

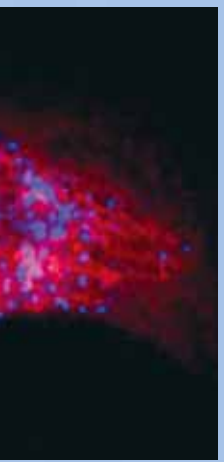
The Rhinovirus with Dr. Ellen Foxman

By Teresa Ming Shan Fan 樊銘姍

它是引起感冒的罪魁禍首。鼻病毒的誤解有很多，包括病毒從休眠狀態轉變為引起人類感染的一系列情況亦有很多說法。但我們對這種常見的病毒了解並不深。常覺得寒冷的天氣會帶來流感，容易生病，但到底這有沒有道理呢？耶魯大學的 Dr. Ellen Foxman 及其研究團隊調查了這個非常棘手的問題，探索了鼻病毒表現為症狀的機制，以及身體對感染的防禦。

Foxman 博士的研究生涯開始於本科，在那裡她獲得了廣泛的研究經驗。她說，「在我大學的第一個暑假，我對懸崖燕子進行了現場研究。這種鳥類每年從美國中西部遷移到南美洲然後再回去。第二個暑假，我是病毒學實驗室的研究助理，再下一年又是在酵母遺傳學實驗室工作。」這些經歷為她未來的醫學研究事業鋪平了道路。

當她在做醫療培訓時，使用聚合酶鏈反應 (PCR) 來檢測病毒的正常化有助於證明患者的病毒感染比以前認為的頻率更高。她說，「事實上，使用這些技術的數據顯示，某些病毒經常訪問身體，以至於被認為是微生物群體的一部分。」但雖然病毒一樣常見，只有一半的感染引起疾病和症狀。「我對寄主病毒相互作用的變化如何影響常見病毒感染的結果非常感興趣。」這個問題推動了她目前對該主題的研究。



Micrograph of human bronchial epithelial cells, seven hours after exposure to rhinovirus 1B. Rhinovirus infection of airway epithelial cells results in accumulation of double stranded RNA (dsRNA; blue) during viral genome replication. Cells are also stained with Mitotracker red to reveal the location of mitochondria, the cellular structures associated with innate immune signaling via the RIG-I like receptor pathway(red).

Photo credits: Ulysses Isidro, Yale University senior thesis student.

幾個因素增強了鼻病毒的複制；其中之一是溫度，以及在較冷的環境中鼻病毒是否更容易複製。以前的觀察表明，許多病毒株在鼻腔溫度為 33-35℃的溫度下變得更加劇毒，而平均體溫為 37℃。Foxman 博士及其小組發現有令人信服的證據表明，在較低的溫度下，感染細胞的防禦反應受到影響，從而使病毒變得更具傳染性。降低細胞防禦反應的其他因素包括患有哮喘的患者，但其機制尚未明了。

以免病毒傳染，口罩標誌著香港的流感季節。雖然面具作為空氣傳播的細菌和病毒的障礙，Foxman 博士認為，可能通過戴口罩來預熱鼻腔氣道也可能是額外保護感染的一個因素。

「預防症狀的最佳策略可能是首先防止複發，這是我們的氣道上皮細胞防禦機制所要做的。」

同時，Foxman 博士和她的團隊將繼續研究和揭示改變我們的氣道防禦系統處理病毒方式的機制。更深入地了解這些機制對於製定更好的預防普遍感染如普通感冒的策略至關重要。

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