Shamelessness Shouldn't Be Anyone's Nature
——An Open Letter to *Nature* (Part XX)

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【Summary】 On Sept 14, 2012, Fang published an article in *Xinhua Daily Telegraph* about caloric restriction and longevity. While introducing Clive McCay's classic study in the first paragraph, Fang made 10 low level mistakes, suggesting he didn't read the original paper. When facing a plagiarism allegation that he plagiarized a professor of Peking University, Fang issued a statement saying instead he plagiarized the professor, the professor plagiarized him, because his article was a newer version of an old one which was published ten years earlier. The fact is, both the old and new articles are plagiarism, and the victims including at least three American professors and a registered nurse. Fang's accusation against the professor could not be substantiated.

【Content】

Fang's Plagiarism History: The Longevity Case

The major players

From left: Dr. Robert Arking and Prof. Tian Qinglai, the victims; Fang, the thief and accuser.

The Story

1. A Scifool Article

Since 2007, Fang has been known in China as a "scifool writer," a writer who fools his readers in the name of science[1]. So far, Fang has published hundreds of scifool articles, and many of them have been analyzed and documented[2].

On Sept. 14, 2012, Fang, pretending as a nutritionist, published *Can You live Longer by Eating Less?* in *Xinhua Daily Telegraph*, a newspaper affiliated with Xinhua News Agency where Fang's wife Liu Juhua works as a chief reporter. The article is about the caloric restriction research, and its first paragraph, which contains only 6 sentence, introduces the classic experiment conducted by Clive Maine McCay and his colleagues in 1935[3]. According to my analysis, Fang made at least 10 low level mistakes in this paragraph[4]. For example, Fang claimed that the object of McCay's experiment was to demonstrate that longevity is inversely proportional to **developmental** rate. The fact is, the authors stated explicitly that "[t]he object of this study was to determine the effect of retarding **growth** upon the total length of life and to measure the effects of retarded **growth** upon the ultimate size of the animal's body." They didn't measure any developmental parameters. Another example of Fang's mistakes is that Fang said by caloric restriction, McCay et al. extended the average lifespan of the male rats by about 50%. The fact is, according to the paper, the average lifespan of the male rats in the two restricted groups were 820 and 894 days, respectively, and that of the control group was 483 days, so the average extensions were 70% and 85%, respectively[3].

The most outrageous mistakes made by Fang were in the last sentence of the paragraph: "Among the normally fed rats, the longest lifespan was 965 days, and among the calorically restricted rats, some lived for more than 1,800 days, equivalent of about 200 years in humans." Anyone who has read McCay's paper could tell that the longest lifespans of the control rat and calorically restricted rat were 1,189 days and 1,421 days, respectively. There was not a single rat among the 104 rats used in the experiment lived for 965 days. And during McCay's lifespan, he had never raised a rat that lived for 1,800 days, to my knowledge. Furthermore, according to Henry H. Donaldson, the very person who established albino rat as a model organism, "a rat three years old ……may be regarded as corresponding to a man ninety years old."[5] Therefore, Fang's equivalency is not right either.

Obviously, Fang didn't read McCay's paper when he was introducing McCay's experiment. The question is: How did he achieve that?

On Sept. 21, 2012, I wrote an open letter to Mr. Xie Guoji, the editor-in-chief of the *Xinhua Daily Telegraph*, in which I analyzed Fang's scifool article, exposed Fang's ignorance, accused him of deceiving his readers with false information, and suggested that his mistakes were made by plagiarizing other, yet to be identified sources[4]. Based upon my experience, I expected no-response from Fang, since no-response had been Fang's response to most of my allegations and accusations in the past five years. However, something strange happened.

2. A Self-incrimination Statement

Based on my suggestion in the open letter, an internet user found two pieces of evidence showing that Fang might have committed plagiarism indeed[6]: in an article published in 2009 and authored by Professor Tian Qinglai of Peking University, there were above-mentioned mistakes of 965 days/1800 days/200 years; also, both Fang and Prof. Tian wrote the following passage:

> "The diet of the residents of Ryukyu Islands contains adequate nutrition, but the calorie is much

> lower than the norm in Japan. Their lifespan is also longer than the norm in Japan, the incidence of centenarians is 2-40 times as many as the number on other Japanese islands."[7]

The only difference between Fang and Tian was that the latter said the incidence of centenarians in Ryukyu was 2-4 times, instead of 2-40 times as Fang said, as many as the rest part of Japan.

On Sept. 25, Fang issued a statement, entitled *A Statement Regarding the Plagiarism Committed by Professor Tian Qinglai of the Life Science School at Peking University*, which reads as follows:

> "The article entitled *The Gate to Longevity - Restricting Calories and Enhancing Nutrition* by Tian Qinglai, a professor in the School of Life Sciences of Peking University, and the research director of Aging Research Center at Peking University, published in *Health Guide* (October, 2009, http://www.vloho.com/Article/P6110052204.html), was mainly based on my article written in 2002, *Eat Less, Live Longer*, published in the 21st issue of *Globe* semimonthly, in 2002. The article was later published in *Disillusionment of Longevity* and *Science Makes You Healthy* (http://tech.sina.com.cn/oi/2003-07-24/1407213150.shtml). The forth part of Tian's article, *Examples of Longevity and Anti-aging*, is complete plagiarism; the only difference is that he foolishly changed my '2-40 times' to his '2-4 times.'
>
> "Recently, there has been new progress in the research on the relationship between caloric restriction and aging, [the results] challenging the traditional opinions, therefore I wrote *Can You Live Longer by Eating Less?*, and published in *Xinhua Daily Telegraph*. Some paragraphs, including the one plagiarized by Tian Qinglai, were rewritten from the old article. Based on that fact, a Fang-hater accused me of plagiarizing Tian Qinglai, and the senior Fang-haters, such as Yi Ming, Han Han's father Han Renjun, Sun Haifeng, Without V (Muzi Mei), and Yi Tian, outcried even louder. My popular science articles have been plagiarized many times, and I didn't want to make a fuss over such plagiarism involving only one or two paragraphs. However, since Fang-haters want to use this case to level an allegation against me, I'd better make a statement."[8]

Fang must have thought that he hit the jackpot in this case, because he posted the statement everywhere: twice on his New Threads, twice on his microblogs, and four times on his blogs[9]. Fang's statement also made his followers festively joyous, one of them, hqabc_, many people believe it's the account controlled by Fang's wife Liu Juhua, even demanded an apology from me[10].

Why this statement was so important to Fang and his followers? Because it was the first one since August, 2011, when Dr. Robert Root-Bernstein of Michigan State University made a public plagiarism allegation against Fang in his *An Open Letter to Shi-Min Fang*, and Fang swore repeatedly that he would not respond to any questions regarding to the charge, and anyone who continued commenting the incident on his microblog would by blocked[11]. To most Chinese people, including Fang's followers, Fang's behavior was equivalent to admitting wrong doing. Therefore, Fang and his followers hoped that the statement against Prof. Tian could serve as a pivotal turning point: instead of a plagiarizer, Fang had been a victim of double crimes: he was first plagiarized by Professor Tian, then he was falsely accused of plagiarism by Fang-haters. How innocent and pitiful the Great Leader Fang is, and how hateful and evil these Fang-haters are!

However, to veteran "Fang experts," Fang's performance revealed more than what he intended. The fact is, Fang has repeatedly claimed that the standard of plagiarism for popular science articles is different from that for academic papers, and in the former, there is no need to give citations or attributions[12]. So Fang's accusation against Professor Tian actually put himself in an extremely awkward situation: if his accusation was valid, then the 92 plagiarism cases documented before that time against him must also be valid; if his accusation was false, then his so called "fraud busting" must be fraudulent, at least partially

so, and his claim that he's fraud busting had been flawless was falsified. Also, Fang's accusation against Prof. Tian clearly indicates that Fang does know that there is no different standard of plagiarism for popular science writings, since Tian's article was just one of such. It also shows that Fang has been intentionally using double standards to defend himself and to attack other people. Furthermore, Fang's allegation against Tian showed clearly how imprudent, reckless, irresponsible, and evil his "fraud busting" is: except for similarities, Fang had no other evidence - he even dared not to give a comparison.

The fact is, Professor Tian studied the effect of caloric restriction on aging as early as in 1986, and he has published numerous related books[13]. On the other hand, Fang has no training, no background, and no basic knowledge in these areas. So, why would an expert plagiarize a few sentences from a layman? On the other hand, there had been several articles before Tian's talking about the centenarian incidence in Okinawa[14]. So, even if Professor Tian did commit plagiarism, why he had to plagiarize Fang?

Professor Tian Qinglai's gerontological books

3. A Mammoth Thief

Fang's another miscalculation by issuing the statement was that he revealed the information of his original article. Based on this information, I cracked Fang's another plagiarism case, the 93rd case identified by then.

According to Fang, his *Eat Less, Live Longer* was originally published in *Globe* magazine, and in two books. The fact is, according to the link he provided, the article was also published in *Southern Weekend* in 2003, and, I found out, was present in the book *Are You Eating Supplements or Poisons?*, published in 2008 in Taiwan. In other words, Fang published the article five times in less than 6 years, and *Can You live Longer by Eating Less?* is its 6th appearance in print media.

Between 2002 and 2008, Fang's *Eat Less, Live Longer* appeared 5 times in China's print media

From left: *Globe* magazine (2002); *Disillusionment of Longevity* (2002); *Southern Weekend* (2003); *Science Makes You Healthy* (2007); *Are You Eating Supplements or Poisons?* (2008).

The publishers of Fang's fraudulent article and books

From left: Mr. Ji Bin (姬斌), the editor-in-chief of *Globe* magazine, a subsidiary magazine of Xinhua News Agency, published Fang's *Eat Less, Live Longer* in 2002; Mr. Mao Wentao (毛文涛), the president of Shanghai Science and Technology Press, published Fang's *Disillusionment of Longevity* in 2002; Mr. Zhang Baixin (张百新), the president of Xinhua Publishing House, a subsidiary company of Xinhua News Agency, published *Fang's Science Makes You Healthy* in 2007; and Mr. Xie Guoji (解国记), the editor-in-chief of *Xinhua Daily Telegraph*, a subsidiary newspaper of Xinhua News Agency, published at least 9 articles plagiarized by Fang in 2012, including *Can You live Longer by Eating Less?*.

According to Fang, only "some paragraphs…were rewritten from the old article" in *Can You live Longer by Eating Less?*. However, a careful examination reveals that among the 12 paragraphs, 1,866 characters, in the article, 8.5 paragraphs, 1,350 characters are the same as the old version, *Eat Less, Live Longer* (see figure below).

If this is not self-plagiarism, then what is self-plagiarism?


吃得少能否活得老？

新华每日电讯

新华通讯社出版

好问

■方舟子（科普作家）

1917年，奥斯本等三位美国生物学家在用大鼠做营养实验时，发现那些没有喂饱的老鼠，生长迟缓，而其寿命似乎也延长了。受到这个结果的启发，1935年，美国康奈尔大学的麦克凯等人直接验证是否动物寿命真的与发育速度成反比，在大鼠断奶后，他们给其中的一组提供完备的营养物质，但是严格限制其饮食，让它们一直处于饥饿中，而另一组老鼠则任其吃饱。饮食受限制的老鼠发育几乎停止，身体也不再长大，一些老鼠夭折了，但是存活下来的老鼠中，寿命明显增长了。雄鼠所受的影响更显著，寿命平均延长了约50%，喂食正常的老鼠中，寿命最长的为965天，而限制喂食的老鼠，有的活到了1800多天（相当于人活到200岁）。

以后许多实验室都做了类似的实验，得到了相当一致的结论，多项实验结果都表明，如果让鼠类的食物包含完备的营养物质，但是把食物中的热量减低25%～60%，它们在中年后得慢性病的危险减低了，而寿命也延长了大约30%，对其他动物，包括脊椎动物和无脊椎动物所做的类似实验，也得到了类似的结果。**限制饮食热量，是迄今为止我们所知道的唯一能够有效地延长动物生命的环境因素。**

饮食热量受限制的老鼠不仅活得长，而且显得更健康。麦克凯等人当时已发现这些老鼠得各种癌症的危险性降低了，心血管和肾脏的老化也延缓了，后来的研究者还发现其他的延缓衰老的标志，如慢性炎症减少，免疫力提高，对血糖的耐受性增强，跑年记忆力提高等。这些老鼠对环境致癌物的抵抗力也增强了，用几种不同的致癌物做试验，发现它们因此患癌症的概率显著低于对照组。限制饮食热量还显著地减少了体内脂肪细胞的数目，从而防止了晚年肥胖；而如果仅仅限制饮食中的脂肪含量，达不到这个效果。

总之，各种研究都表明限制饮食热量对动物的衰老过程有根本性的影响，而不只是某些表面效果。

限制饮食热量对老鼠和其他动物的健康和寿命的积极作用已被充分证明了，我们更关心的是：它基不基也适用于灵长类和人类？在上世纪八十年代末，美国有两个研究小组开始对罗猴做限制饮食热量实验，由于罗猴的寿命大约为40年，直到最近，才有了实验结果，不幸的是，这两个研究小组的实验结果并不一致。

第一个研究小组是威斯康星国家灵长类研究中心，根据他们在2009年发布的结果，限制饮食热量能够显著延长罗猴的寿命，他们发现，对照组罗猴已有37%死于与衰老有关的疾病，而饮食热量少30%的罗猴死于这类疾病的只有13%，但另一个研究小组（国家衰老研究所）在近日发布的结果则表明，虽然限制饮食热量的罗猴显得更健康，在免疫、运动协调性等方面有较好的表现，但是寿命却没有延长。

这两组实验在设计方面有些差异，国家灵长类研究中心的罗猴吃的是不健康的饮食，蔗糖在食物中的含量高达28.5%，而国家衰老研究所的罗猴吃的食物较健康，蔗糖只占3.9%，还添加了鱼油和抗氧化剂。国家灵长类研究中心的对照组罗猴吃的食物是不受限制的，让他们随便吃，而国家衰老研究所的对照组罗猴吃的食物也是定量的，这些差异也许导致了不同的结果。如果饮食健康的话，热量是否受限也许并不重要。

那么人类又是如何呢？我们没法对人类也做类似的实验。战争时期的战俘、饥荒地区的难民被迫忍饥挨饿，饮食热量是受限制了，但是他们也往往营养不良，所以不能说明问题。**不过，有一些间接证据表明限制饮食热量可能对人体也有积极作用。**琉球群岛的居民的饮食有充足的营养，但热量低于普通日本人，他们的寿命也长于普通日本人，其百岁寿命的数目是日本其他地区的2～40倍，世界各地的百岁寿星也极少有肥胖的。在1991～1993年间，四男四女在一个与外界隔绝的生态系统"生物圈2"生了两年，在此期间，他们的饮食营养齐备，但热量大约为一般饮食的90%，他们的体重明显降低了（男的降低18%，女的降低10%），并出现了与卡路里受限制的老鼠类似的生理变化。

"生物圈2"的居民中包括研究衰老的分子生物学，加州大学洛杉矶分校的教授洛伊·瓦尔佛德。他成了限制卡路里寿法的倡导者和实践者，声称他因此至少能活到120岁。正常人一天需要从食物中吸收2000～2500大卡热量，其中大约30%来自脂肪，30%来自蛋白质和40%来自碳水化合物。如果把热量供应减低30%，则每天大约只吸收1500大卡，那些长期严格执行这个饮食计划的人，身体将会变得非常精瘦，总是感到饥饿和寒冷。由于他们体内脂肪已大部分丧失，他们将没有足够的脂肪充当骨骼缓冲物，他们坐下的时候，臀部的骨骼会因被压着而感到疼痛。由于脚底没有脂肪垫着，走路也会很得疼。食物的热量将主要来自水果和蔬菜，再加一点蛋粉和肉。

瓦尔佛德曾在其新书发布会上，请来窦晶霖拖根据长寿配方制作的膳食。来宾们都觉得难以入口，有的人认为如果要长期吃这样的食物，还不如短命。本来的是，在忍饥挨饿多年后，瓦尔佛德并没能实现其长寿目标，他在2004年因运动神经疾病导致的呼吸系统衰竭而去世，享年79岁。

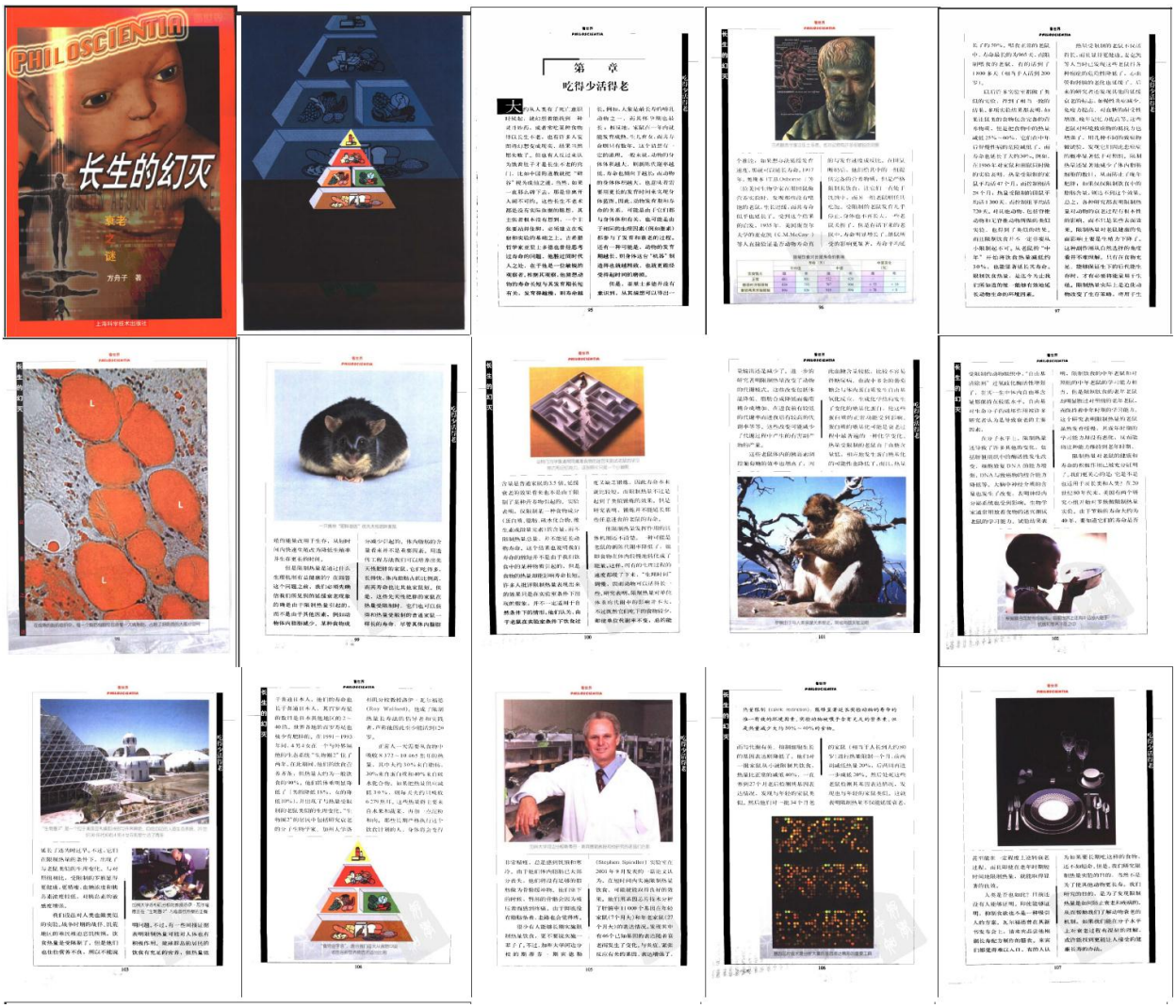
各种实验都表明，限制饮食热量即使不能延长寿命，也能延缓衰老，对健康有益，即便如此，抑制食欲也不是一种吸引人的方案，不过也有研究表明，只在短时间内限制热量饮食，可能就能取得良好的延缓衰老的效果。吃得少也许不能活得老，却能活得好。

Garbage Recycling

On Sept. 14, 2012, *Xinhua Daily Telegraph* published Fang's *Can You Live Longer by Eating Less?*, which has nearly three quarters of the same content (highlighted in yellow) as his original article published in 2002, *Eat Less, Live Longer*, which was published 5 times between 2002 and 2008.

An astonishing finding was that, when the article appeared in *Disillusionment of Longevity* in 2002, it was accompanied by 13 images, none of them with a source acknowledgement, therefore they must have been pirated by Fang from the internet. Yes, from ancient Greek Aristotle to a present day African hungry boy to American scientists Stephen Spindler and Roy Walford; from fat cells to a fat mouse to a Barbary ape; from a humorous poster to a menu picture to a gene chip photo; from the pyramid of food to the pyramid of Biosphere 2: anyone, anything, could be stolen by Fang, and sold by him over and over, whether they are protected by the copyright law or not.

If this is not stealing, then what is stealing?



Q: How does a thief write books? A: By stealing.

Fang stole 1 table and 13 images in the chapter of *Eat Less, Live Longer* in his *Disillusionment of Longevity*, none of them was attributed. The images alone occupied more than a half of the space in the chapter. (The entire book contains 134 pages, 145 images.)

The Evidence of Plagiarism

1. A Revealing Table

The above finding was astonishing indeed, but the most valuable finding in the book was a table, "The effect of calorie-restricted diet on the longevity of field mice." The numbers in the "lifespan" columns were from McCay's papers on white rats[15], but the numbers in the "percent change in median lifespan" columns didn't appear in these papers.

The effect of calorie-restricted diet on the longevity of field mice

限制饮食对田鼠寿命的影响

Diet 饮食情况	寿命（天）Lifespan (days) 平均值 Mean 雄♂	雌♀	中值 Median 雄♂	雌♀	Percent Change in Median Lifespan 雄♂	雌♀
正常 Normal	483	801	522	820	–	–
Restrict from weaning	820	755	797	904	+ 53	+ 10
Restrict 2 wks after weaning	894	826	919	894	+ 76	+ 9

A mysterious table

A table appeared in Fang's *Disillusionment of Longevity* (p. 96) (above) and *Science Makes You Healthy* (p. 38) (not shown). The two tables are exactly the same, except that the first one indicated the animals used were field mice (田鼠) and the other were rats (大鼠). The English translations in the above table are mine, and due to space limitation, some Chinese characters were covered by their English translations.

I searched the internet, and found out that Fang's table was stolen from the *Biology of Aging: Observations and Principles* by Dr. Robert Arking, a biology professor at Wayne State University in Detroit, about 90 minutes away from Michigan State University, Fang's alma mater.

Table 7.1 **The Effect of Calorie-Restricted Diet Longevity in Rats**

Diet	Life span (days) Mean ♂	Mean ♀	Median ♂	Median ♀	Percent change in median life span ♂	♀
Normal	483	801	522	820	—	—
Deficient from time of weaning	820	755	797	904	+53	+10
Deficient from 2 weeks after weaning	894	826	919	894	+76	+9

Source: After McCay and Crowell 1934, and McCay, Crowell, and Maynard 1935.

Table 7.1 of Robert Arking's *Biology of Aging* (1998 edition, p.314)

The table is a composite from two of McCay's papers, but the data in the column of "percent change in median life span" (red-boxed) were not present in either of them, so the values in the column must be calculated by Dr. Robert Arking himself. Fang's table contains exactly the same content as that of Dr. Arking's, but he has never acknowledged the fact.

Dr. Arking's book has three editions: 1991, 1998, and 2006, and more than one half of Fang's *Eat less, Live Longer* was directly translated from the 2nd edition of *Biology of aging*, including its table 7.1.

2. Mistakes from Misreading

As mentioned above, the case was initiated from the finding of many stupid mistakes in the first paragraph of Fang's *Can You live Longer by Eating Less*, and these mistakes were the duplication of his 2002 article, *Eat Less, Live Longer*. Then, how did Fang make these mistakes? The answer is, mostly by misreading Dr. Arking, a few by copying Dr. Arking and other people. Let's examine the misreadings first.

According to Fang, the average lifespan of underfed male rats in McCay's experiment was extended by about 50% (See sentence II-5 in the table below). Obviously, Fang didn't know the difference between mean and median, so he interpreted Dr. Arking's Table 7.1 in a wrong way.

Another example of Fang's misreading includes his following statement:

> "In 1917, three American biologists, T. B. Osborne et al., conducted a nutrition experiment with rats, and they found that those underfed grew retarded, and seemed lived longer." (Sentence II-1)

The fact is, the paper by Osborne et al. was published in March 1917 in *Science*, and their experiment lasted about 3 three years[16]. Therefore, neither the experiment was conducted, nor the finding was made, in 1917. And the only reason Fang said so was because in Dr. Arking's book, there is the following sentence:

> "This idea was derived partly from the works of philosophers such as Aristotle and partly from the experimental work of Osborne, Mendel, and Ferry (1917), whose data suggested, but did not prove, that underfed rats live longer." (p.313)

The fact is, even the sentences which Fang used to accuse Professor Tian of plagiarism were originally plagiarized by Fang from Dr. Arking, and the only difference was that Fang translated Okinawa, an island of Ryukyu Islands, into Ryukyu Islands:

> Fang: "The diet of the residents of Ryukyu Islands contains adequate nutrition, but the calorie is much lower than the norm in Japan. Their life span is also longer than the norm in Japan, the incidence of centenarians is 2-40 times as many as the number on other Japanese islands. Centenarians around the world have rarely been obese." (Sentences X-4 and X-5)
>
> Dr. Arking: "In the past, the caloric intake of much of the population of Okinawa was much lower than the norm in Japan, but the nutrition of the Okinawans was otherwise adequate. Okinawa has a high incidence of centenarians: 2 to 40 times as many as may be found on any other Japanese island. ⋯Other anecdotal evidence suggests that very few, if any, centenarians or other long-lived people have been obese." (p.324).

3. Mistakes from Believing

So, why did Fang say McCay's experiment was to test whether animal's lifespan was really inversely proportional to the **developmental** rate, while the title of McCay's paper was *The effect of retarded **growth** upon the length of life span and upon the ultimate body size*? Because Dr. Arking wrote:

> "The experiments of McCay and his colleagues, which we will discuss shortly, grew out of the idea that longevity is inversely proportional to developmental rate. This idea was derived partly from the works of philosophers such as Aristotle⋯." (p.313)

The fact is, what Aristotle said was, it is a general rule that the larger animals live longer than the smaller ones[17], so his idea was more like "longevity is inversely proportional to **growth** rate" rather than "to **developmental** rate."[18] ("Aristotle" was deleted in the 3rd edition of Biology of Aging.)

In fact, Dr. Arking only implied what Aristotle thought, but Fang developed that implication into an explicit statement, saying Aristotle thought that animal's longevity was related to their developmental duration, the slower the development rate, the longer the lifespan. (Sentence I-4).

Fang's over extrapolation was not limited to Aristotle. In his book, Dr. Arking mentioned many times the negative effect of protein glycation or glycosylation on aging, such as "advanced glycosylation end products." Obviously based on the information, Fang wrote:

> "Protein glycosylation is probably the most common chemical reaction during the aging process." (Sentence VII-3)

It is very clear that this famous "biochemist," trained by Michigan State University, praised by his advisor[19], and "certified" by *Science* magazine[20], didn't know the difference between the non-enzymatic glycation and enzymatic glycosylation, the latter is essential for the functions of many bio-molecules, mainly proteins[21].

So, where did Fang's mistakes of 965/1800/200 come from? Some people suggested that Fang might have plagiarized the articles in *Life Extension Magazine*. In two articles published in the magazine in 1995 and 2001, respectively, there are following sentences:

> "The longest lived calorie restricted rat survived for more than 1,800 days (the equivalent of about 200 years in humans) in the laboratory of Morris H. Ross at the Institute For Cancer Research in Philadelphia."
>
> "McCay's oldest control rat died at 965 days, whereas his oldest CR rat lived 1,456 days (150 years in human terms)."[22]

One might doubt that an American Ph. D. could make a statement like Fang's ("Among the normally fed rats [by McCay et al.], the longest lifespan was 965 days, and among the calorically restricted rats, some lived for more than 1,800 days, equivalent of about 200 years in humans") based upon the above two sentences, because they actually conflict with what Fang wrote. However, it seems impossible to underestimate Fang's intelligence and knowledge. In the 12th paragraph of *Eat Less, Live Longer*, Fang introduced a PNAS paper by Dr. Stephen Spindler of UC Riverside. Apparently unable to understand the original paper, Fang based his introduction on an interview of Dr. Stephen Spindler by *Life Extension Magazine*. In the interview, Dr. Spindler said:

> "We took a group of animals that had been allowed to eat almost all they wanted their whole life and we intervened when they were quite old-34 months of age. These mice would be the equivalent of people who are probably 80 years old or older - I'm just guessing at the human equivalent age."[23]

And Fang wrote:

> "They then underfed a group of 34 months old mice (equivalent of people who are about 80 years old)…" (Sentence XII-6)

It is common knowledge that rats have longer lifespan than mice, so how come 34 months old mice are equivalent to 80 years old people, while 60 months old rats are equivalent to 200 years old people? Even if the rodents have the same lifespan, the numbers still do not add up, unless Fang had a queer formula.

4. East Lancing Ph. D. Plagiarizing North Carolina Master

As I mentioned before, Fang values his Ph. D. credential dearly, and if anyone without a doctoral degree wants to argue with him, Fang would for sure laugh at his overreaching. For example, in 1999, Fang told one of his opponents:

> "In the biology domain, anyone without a doctorate is not qualified to conduct research; those with a Master's degree can only provide technical labor, let alone other people."[24]

And in 2011, because of his wife's plagiarism case, Fang declared, in *Xinhua Daily Telegraph*, that a Master degree graduate student has no need to write a degree thesis, obviously implying that plagiarism in a Master's degree thesis is not a big deal due to its non-necessity[25]. However, in *Eat Less, Live Longer*, Fang plagiarized an article written by a Master from North Carolina. In the article, Fang wrote:

> "Many results showed that if rodents were on a diet with complete nutrients, but with 25-60% calorie reduction, their risks of chronic diseases after middle age would be reduced; and their lifespan extended by about 30%. For example, one experiment conducted in 1986 with mice and rats showed that the calorie restricted mice lived 47 months on average, and the control group lived 28 months; the calorie restricted rats lived 1300 days on average, and the control group lived 720 days." (Sentences III-2 and III-3)

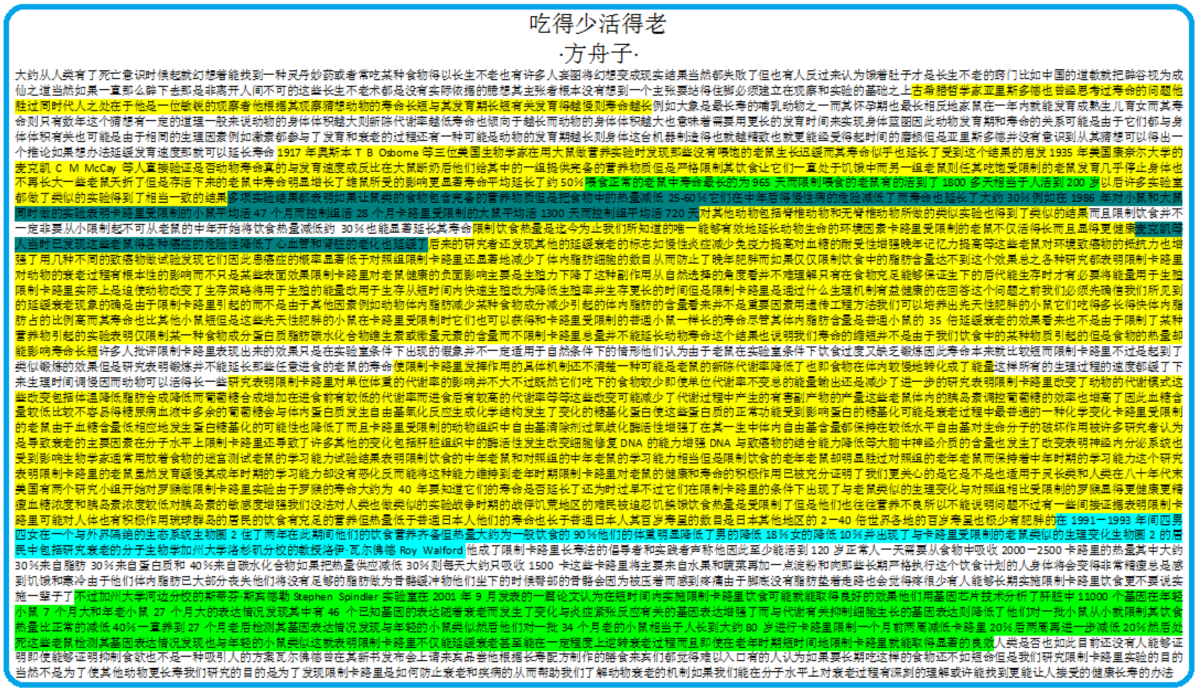
On the internet, there is an article written by Jean E. Pierog (R.N., M.S., NC), in which it says:

> "One such study with mice and rats by Weindruch (1986) showed that fully fed mice lived on the average 28 months versus the calorie restricted group who lived 47 months. Rat survivals were shown to be approximately 720 days old for those eating ad lib and 1300 days of life if calories were restricted. In these and other studies, calorie restriction is defined as a reduction in calories of 25-60% from ad lib feeding levels while providing an adequate intake of essential vitamins and nutrients."[26]

Just by looking at the Arabic numbers in the two passages, you should be able to tell their relationship. Doctor Fang must have been extremely loyal to Master Pierog when he was plagiarizing the article. Unfortunately, the passage he stole contains a crucial mistake: in 1986, Dr. Richard Weindruch only published one paper as the first author, and not only that paper did not contain any of these numbers, it also did not deal with rats[27].

Concluding Remarks

In summary, Fang's *Eat Less, Live Longer* contains 3,902 Chinese characters, among them, 2078 (53%) were translated from Dr. Arking's *Biology of Aging*, 400 (10%) from articles published in *Life Extension Magazine*, 170 (4.4%) from Master Pierog's internet article, and 140 (3.6%) from one of Dr. Walford's papers. The total proportion of plagiarism is 71%. Considering the space occupied by the stolen images in his books, Fang's own contribution to "his article" is merely about 10%, assuming those sentences without identified sources were indeed written by himself.



吃得少活得老

·方舟子·

Multiple plagiarisms

The text of Fang's *Eat Less, Live Longer* contains 3,902 Chinese characters, among them, 53% (highlighted in yellow) were translated from Dr. Robert Arking's book, *Biology of Aging*. Besides Dr. Arking, Fang also plagiarized Ms. Jean E. Pierog (dark blue), Dr. Walford, et al. (light blue), and articles in *Life Extension Magazine* (green). The total percentage of plagiarism is more than 70%.

On Oct. 17, 2012, three weeks after Fang's self-incriminating statement, I published another open letter, reporting the above findings[28]. The open letter was sent via email to Mr. Xie Guoji and Fang, the editor-in-chief and the columnist of *Xinhua Daily Telegraph*, respectively, on the same day of its publication. However, neither chief editor Xie nor columnist Fang has responded to the letter yet. Fang's non-responsiveness is understandable and expected, because that's the last strategy available to him whenever his fraud is exposed. In other words, by not responding to an allegation without any excuses (such as having no time, unnecessary, etc.), Fang admitted his plagiarism, as well as his false accusation against Prof. Tian.

The non-responsiveness from the newspaper is also understandable and expected, for the reasons I will reveal sometime later.

The thing I don't understand and didn't expect is the non-responsiveness from *Nature*. Of course you have your own, probably unspeakable, reasons. However, no matter how secretive they are, they will be known to the world eventually. It is amusing that a prestigious science journal in the Western world behaves just like a communist newspaper in China on the matter involving Fang. Maybe he does have some demonical power of transformation!

On the other hand, if a demon can be praised, prized, and promoted just because he is wearing a "fraud fighter" uniform, holding a "stand-up for science" banner, yelling the "root out the fakers" slogan, then does it make pseudoscience, antiscience, frauds, and fakers look so innocent and benign, make science look so evil and malicious?

A Complete Comparison between Fang's *Eat Less, Live Longer* with Its Sources			
Note: The Chinese text of Fang's article is retrieved from his New Threads website and presented here in its entirety. The article is translated by me (The Roman numerals indicate the paragraph order, and the Arabic numerals indicate the sentence order in a paragraph.)			
Fang's article			**The victims' articles** Unless specified, the page numbers are of Dr. Arking's Biology of Aging, 2nd edition. Sinauer Press, 1998.
Seq.	**Chinese**	**English Translation**	
I-1 I-2 I-3	大约从人类有了死亡意识时候起，就幻想着能找到一种灵丹妙药，或者常吃某种食物得以长生不老，也有许多人妄图将幻想变成现实，结果当然都失败了。但也有人反过来认为饿着肚子才是长生不老的窍门，比如中国的道教就把"辟谷"视为成仙之道，当然，如果一直那么辟下去，那是非离开人间不可的。这些长生不老术都是没有实际依据的臆想，其主张者根本没有想到，一个主张要站得住脚，必须建立在观察和实验的基础之上。	Since the time human beings had the consciousness of death, they have been dreaming of finding a wonder medicine, or a food, which could make them forever young. There were some people who tried to realize this dream, and of course they failed. However, some people believes hunger is the way leading to longevity, for example, the Taoism teaches that by bì gǔ, or refraining from eating grain, one could become an immortal. Sure, if a person keeps refraining, he will leave man's world. All these immortal arts are unpractical fantasies, and the practitioners never realized that to be tenable, a proposition must be built on the basis of observation and experiment.	
I-4 I-5	古希腊哲学家亚里斯多德也曾经思考过寿命的问题，他胜过同时代人之处，在于他是一位敏锐的观察者。他根据其观察，猜想动物的寿命长短与其发育期长短有关，发育得越慢，则寿命越长。	Ancient Greek philosopher Aristotle thought about the question of lifespan, he was better than his contemporaries because he was a keen observer. Based on his observation, he conjectured that animal longevity was related to their developmental duration, the slower the development rate, the longer the lifespan.	The experiments of McCay and his colleagues, which we will discuss shortly, grew out of the idea that longevity is inversely proportional to developmental rate. This idea was derived partly from the works of philosophers such as Aristotle ……. (p.313)
I-6	例如大象是最长寿的哺乳动物之一，而其怀孕期也最长，相反地，家鼠在一年内就能发育成熟、生儿育女，而其寿命则只有数年。	For example, elephants are one of the mammals with longest lifespan, and its pregnancy is the longest also. On the contrary, mice can mature and reproduce in one year, and their lifespan is only a few years.	
I-7 I-8 I-9 I-10 I-11	这个猜想有一定的道理。一般来说，动物的身体体积越大，则新陈代谢率越低，寿命也倾向于越长；而动物的身体体积越大，也意味着需要用更长的发育时间来实现身体蓝图。因此动物发育期和寿命的关系，可能是由于它们都与身体体积有关。也可能是由于相同的生理因素（例如激素）都参与了发育和衰老的过程。还有一种可能是，动物的发育期越长，则身体这台"机器"制造得也就越精致，也就更能经	The conjecture makes some sense. Generally speaking, the bigger an animal's size is, the lower its metabolic rate, and the longer its lifespan. Also, the bigger size of an animal means that it needs longer time to develop the body plan. Therefore, the relationship between the length of animals' developmental periods and their lifespans may be determined by their body sizes, or because that the same physiological factors (such as hormones) are involved in the processes of development and aging. Another possibility is that the longer the developmental period is, the more elegant the body	

	受得起时间的磨损。	machine is, and thus more resistant to wear.	
II-1	但是亚里斯多德并没有意识到，从其猜想可以得出一个推论：如果想办法延缓发育速度，那就可以延长寿命。	However, Aristotle didn't realize that from his conjecture one could draw a conclusion that by delaying the development, an animal's lifespan could be extended.	
II-2 II-3 II-4 II-5	1917 年，奥斯本（T. B. Osborne）等三位美国生物学家在用大鼠做营养实验时，发现那些没有喂饱的老鼠，生长迟缓，而其寿命似乎也延长了。受到这个结果的启发，1935 年，美国康奈尔大学的麦克凯（C. M. McCay）等人直接验证是否动物寿命真的与发育速度成反比。在大鼠断奶后，他们给其中的一组提供完备的营养物质，但是严格限制其饮食，让它们一直处于饥饿中，而另一组老鼠则任其吃饱。受限制的老鼠发育几乎停止，身体也不再长大，一些老鼠夭折了，但是存活下来的老鼠中，寿命明显增长了。雄鼠所受的影响更显著，寿命平均延长了约 50%。	In 1917, three American biologists, T. B. Osborne et al., conducted a nutrition experiment with rats, and they found that those underfed grew retarded, and seemed lived longer. Inspired by this result, in 1935, C. M. McCay et al. at Cornell University directly verified whether animal's lifespan was really inversely proportional to the development rate. After weaning, one group of rats was fed a nutritionally complete but strictly restricted diet so they were always hungry; the other group was provided with unlimited food. The development of the restricted rats almost ceased, their bodies grew no more, some of them died, but the surviving rats lived significantly longer. The effect was more significant on the male rats, the average lifespan was extended by about 50%.	This idea was derived partly from the works of philosophers such as Aristotle and partly from the experimental work of Osborne, Mendel, and Ferry (1917), whose data suggested, but did not prove, that underfed rats live longer. McCay, Crowell, and Maynard (1935) demonstrated that rats that were fed a nutritionally complete but calorie-restricted diet from the time of weaning had significantly increases in the values of mean, median, and maximum life span when compared to animals fed a normal diet conducive to rapid growth (Table 7.1). The animals provided with unlimited calories grew and matured normally. In the restricted group, maturation was greatly slowed, although these animals held their weaning weight and suffered from no other nutritional deficiency, since their diet included adequate amounts of protein, vitamins, and minerals. Growth and development in the restricted animals resumed only after they were given additional calories at about 2 years of age. The restricted animals never attained a normal body size or body weight; they remained about 15% smaller than their normal controls. (p.313)
II-6	喂食正常的老鼠中，寿命最长的为 965 天，而限制喂食的老鼠，有的活到了 1800 多天（相当于人活到 200 岁）。	Among the rats fed normally, the oldest lived for 965 days; among the restricted rats, some lived for more than 1,800 days, equivalent of about 200 years in humans.	The longest lived calorie restricted rat survived for more than 1,800 days (the equivalent of about 200 years in humans) in the laboratory of Morris H. Ross at the Institute For Cancer Research in Philadelphia. (Anonymous. Dietary Manipulation Of Aging. Life Extension Magazine, June 2001.) McCay's oldest control rat died at 965 days, whereas his oldest CR rat lived 1,456 days (150 years in human terms). In the 1960s, CR rats in the laboratory of Morris H. Ross at the Institute For Cancer Research in Philadelphia, survived for more than 1,800 days (180 years in human terms). (Saul Kent. Aging Research Becomes A Science. Life Extension Magazine, December 2001.)
III-1	以后许多实验室都做了类似的实验，得到了相当一致的结果。	Since then, many laboratories have conducted similar experiments, and obtained similar results.	These observations have since been confirmed and extended by many other investigators. (p.313)
III-2 III-3	多项实验结果都表明，如果让鼠类的食物包含完备的营养物质，但是把食物中的热量减低 25-60%，它们在中年后得慢性病的危险减低了，而寿命也延长了大约 30%。例如在 1986 年对小鼠和大鼠同时做的实验	Many results showed that if rodents were on a diet with complete nutrients, but with 25-60% calorie reduction, their risks of chronic diseases after middle age would be reduced; and their lifespan extended by about 30%. For example, one experiment conducted in 1986 with mice and rats	This finding, that animals on a low calorie, nutrient rich diet far outlived animals allowed to eat as much as they wanted, has been replicated a great number of times. One such study with mice and rats by Weindruch (1986) showed that fully fed mice lived on the average 28 months versus the calorie restricted group who lived 47 months. Rat survivals were

	表明，卡路里受限制的小鼠平均活 47 个月，而控制组活 28 个月；卡路里受限制的大鼠平均活 1300 天，而控制组平均活 720 天。	showed that the calorie restricted mice lived 47 months on average, and the control group lived 28 months; the calorie restricted rats lived 1300 days on average, and the control group lived 720 days.	shown to be approximately 720 days old for those eating ad lib and 1300 days of life if calories were restricted. In these and other studies, calorie restriction is defined as a reduction in calories of 25-60% from ad lib feeding levels while providing an adequate intake of essential vitamins and nutrients. (Jean E. Pierog. RECIPE FOR LONGEVITY.)
III-4	对其他动物，包括脊椎动物和无脊椎动物所做的类似实验，也得到了类似的结果。	Similar experiments on the other species, including both vertebrate and invertebrate, gave similar results.	The basic observation has been found to apply to other species, both vertebrate and invertebrate, and is hallmarked by its ease of repeatability. (p.313)
III-5 III-6	而且限制饮食并不一定非要从小限制起不可。从老鼠的“中年”开始将饮食热量减低约 30%，也能显著延长其寿命。	Furthermore, the restriction does not have to start from youth. The rodents start the restriction by 30% from middle age could also extend their lifespan significantly.	
III-7	限制饮食热量，是迄今为止我们所知道的唯一能够有效地延长动物生命的环境因素。	Caloric restriction is the only environmental factor that we know so far which can significantly extend the lifespan of animals.	In fact, caloric restriction is the only environmental means that has been shown to significantly slow the mortality rate of any mammal. (p.313)
IV-1	卡路里受限制的老鼠不仅活得长，而且显得更健康。	The calorically restricted rodents not only live longer, but also healthier.	Are these animals that live longer also healthier, or are they sick and feeble? Is the boon of extended longevity a blessing or a curse? What, in other words, is the effect of caloric restriction on age-related pathologies? Many studies have shown that the dietary history of the rodent has a major effect on the age of onset and the incidence of the various age-related pathologies. (pp.314-315)
IV-2	麦克凯等人当时已发现这些老鼠得各种癌症的危险性降低了，心血管和肾脏的老化也延缓了。	McCay et al. also discovered that these calorically restricted rodents had reduced risk of a variety of cancers and delayed age-related deterioration of the vascular system and the kidneys.	McCay also discovered that calorie restriction inhibited a large variety of cancers and delayed age related deterioration of the vascular system and the kidneys. (Jean E. Pierog. RECIPE FOR LONGEVITY.)
IV-3	后来的研究者还发现其他的延缓衰老的标志，	Later researchers also discovered that many age-related changes were delayed,	A large body of data (reviewed by Masoro 1988a, 1992a; Weindruch and Walford 1988; Finch 1990) shows that caloric restriction, in addition to having an effect on the age-related pathologies, delays or eliminates the onset of many normal age-related physiological changes. (p.316)
	如慢性炎症减少、	such as the reduced incidence of chronic tissue inflammations,	and the incidence of chronic tissue inflammations (for example, chronic glomerulonephritis, myocardial fibrosis) and of endocrine hyperplasias is significantly reduced. (p.315)
	免疫力提高、	enhanced immunity,	The early effects of restriction seem to depend on the strain, but a general response to the restriction of calories seems to be a decrease in antibody production coupled with an enhanced cell-mediated immunity. (p.316)
	对血糖的耐受性增强、	enhanced tolerance to blood glucose,	The ability of calorie-restricted animals to satisfy energy

			requirements with low levels of blood glucose implies that they can minimize the age-related effects of glycosylation. (p.322)
	晚年记忆力提高等。	improved memory at old age, etc.	prevention of the decline in the mouse's learning ability, (p.316)
IV-4	这些老鼠对环境致癌物的抵抗力也增强了，用几种不同的致癌物做试验，发现它们因此患癌症的概率显著低于对照组。	These rodents had stronger resistance against environmental carcinogens. Using several different carcinogens in experiment, it was found that they had significantly lower probability of causing cancer [in the restricted group] than in the control group.	Third, the restricted animals have a greater degree of protection against exogenous carcinogens; these rodents showed significantly fewer tumors after exposure to any of several different carcinogens tested. (p.316)
IV-5	限制卡路里还显著地减少了体内脂肪细胞的数目，从而防止了晚年肥胖；而如果仅仅限制饮食中的脂肪含量，达不到这个效果。	Calorie restriction also reduced the number of fat cells, thus prevented later years obesity. Restricting the amount of fat intake only did not have this effect.	An example of a normal trait that is eliminated in restricted animals is the normal increase in the number of fat cells found in particular fat depots in the rat. Not only does caloric restriction eliminate the increase in fat cells, but it brings about a significant decrease in the fat depot mass as a result of a reduction in the number of fat cells (Masoro 1992). Restricting the amount of fat without restricting the total energy intake did not have this effect. (p.316)
IV-6	总之，各种研究都表明限制卡路里对动物的衰老过程有根本性的影响，而不只是某些表面效果。	In summary, various studies have shown that CR affects the aging process fundamentally, not superficially.	It seems reasonable to assume that caloric restriction is affecting, either directly or indirectly, some fundamental process(es) involved in the regulation of biological aging. (p.317)
IV-7 IV-8 IV-9	限制卡路里对老鼠健康的负面影响主要是生殖力下降了。这种副作用从自然选择的角度看并不难理解。只有在食物充足、能够保证生下的后代能生存时，才有必要将能量用于生殖。限制卡路里实际上是迫使动物改变了生存策略，将用于生殖的能量改用于生存，从短时间内快速生殖改为降低生殖率并生存更长的时间。	The major negative effect of CR on the health of the rodents was decreased reproductivity. This side-effect is not difficult to understand from the point of view of natural selection. Only when food is abundant, enough to ensure the offspring survival that using energy for reproduction makes sense. CR actually forces the animals to adjust their survival strategy by relocating the energy from reproduction to survival, from rapid reproduction over a short time period to a reduced rate of reproduction and a longer lifespan.	Clearly, caloric restriction works. But why should mammals come equipped with a mechanism that enables them to live long if they stay hungry? What is the evolutionary sense behind this concept? One proposal suggests that caloric restriction is best viewed as a special application of the disposable-soma theory (see chapter 4), which is based on the premise that an organism can devote its excess calories, beyond the amount needed for basic and essential functions, to reproduction and/or somatic maintenance. In this view, caloric restriction evolved as the set of mechanisms by which an organism adjusts its reproductive strategy to the conditions of its environment by shifting from rapid reproduction over a short time period to a reduced rate of reproduction over a longer life span (Holliday 1989; Richardson and Pahlavani 1994). (p.325)
V-1 V-2	但是限制卡路里是通过什么生理机制有益健康的？在回答这个问题之前，我们必须先确信我们所见到的延缓衰老现象的确是由于限制卡路里引起的，而不是由于其他因素，例如动物体内脂肪减少、某种食物成分减少引起的。	Through what physiological processes does CR benefit health? Before answering the question, we must make sure that the lifespan extension we see are indeed caused by CR, not by other factors, for example, the reduced the amount of body fat, or the decreased intake of certain food components.	It seems reasonable to assume that caloric restriction is affecting, either directly or indirectly, some fundamental process(es) involved in the regulation of biological aging. But what might these process(es) be? And what specific aspect of dietary manipulation is involved? At a minimum, one could hypothesize that the critical variable is the amount of body fat, or the total amount of food eaten, or the total amount of

			calories taken, or the decreased intake of specific (toxic?) food components such as fats or carbohydrates or proteins, or perhaps more subtle effects, such as the lack of exercise in well-fed laboratory animals or delayed onset of degenerative disease in the restricted animals. (p.317)
V-3 V-4 V-5	体内脂肪的含量看来并不是重要因素。用遗传工程方法我们可以培养出先天性肥胖的小鼠，它们吃得多，长得快，体内脂肪占的比例高，而其寿命也比其他小鼠短。但是这些先天性肥胖的小鼠在卡路里受限制时，它们也可以获得和卡路里受限制的普通小鼠一样长的寿命，尽管其体内脂肪含量是普通小鼠的 3.5 倍。	It seems that the amount of body fat is not an important factor. Genetically obese mice can be obtained by genetic engineering method; they eat more, grow fast, have a high percentage of fat in their body, and live a shorter life than other mice. However, when these animals are calorically restricted, they have a lifespan comparable to that of normal CR mice, even though their body fat was about 3.5 times as much as the normal mice.	The amount of body fat is not what is important. The mice in one genetically obese strain eat more, gain weight very rapidly, live a shorter time than other mice, and have a high percentage of fat in their body weight (Table 7.2). Yet when these animals are calorically restricted, they exhibit a median and maximum life span comparable to that of their long-lived, calorically restricted controls, even though they still have about 3.5 times as much body fat as do the controls. The increased longevity appears to be related to food consumption as such in these animals, and not to body composition. (p.317)
V-6 V-7 V-8	延缓衰老的效果看来也不是由于限制了某种营养物引起的。实验表明，仅限制某一种食物成分（蛋白质、脂肪、碳水化合物、维生素或微量元素）的含量，而不限制卡路里总量，并不能延长动物寿命。这个结果也说明我们寿命的缩短并不是由于我们饮食中的某种物质引起的，但是食物的热量却能影响寿命长短。	It looks that aging delay effect is not caused by the restriction of certain nutrients. Experimental result showed that restriction of only single food component (proteins, fats, carbohydrates, vitamins, or minerals), but without CR, could not extend animals' life. The result suggests that our lifespan is not shortened by a certain component in our diet, but calorie intake could affect longevity.	Furthermore, the diet restriction does not appear to work if it consists of the elimination of any single deleterious component of the diet. The individual restriction of any single food component (such as protein, fats, carbohydrates, fibers, or minerals) to the same extent as observed in the complete diet restriction regime does not markedly affect longevity (Iwasaki et al. 1988; Masoro et al. 1989). It now appears unlikely that diet restriction experiments extend the life span by reducing the intake of a particular single component of the food. This observation suggests that our life span is not shortened as a result of toxic components in our diet, but it does support the idea that longevity is affected by the daily amount of food (calories) eaten. (p.318)
V-9 V-10 V-11	许多人批评限制卡路里表现出来的效果只是在实验室条件下出现的假象，并不一定适用于自然条件下的情形。他们认为，由于老鼠在实验室条件下饮食过度又缺乏锻炼，因此寿命本来就比较短，而限制卡路里不过是起到了类似锻炼的效果。但是研究表明锻炼并不能延长那些任意进食的老鼠的寿命。	Many people criticized that the effects of caloric restriction is false appearance under laboratory condition, which may not represent what really happens naturally. They believe that because of overeating and lack of exercise, the lifespan of the rodents in labs are already shorter, and the effect of caloric restriction is nothing but the effect similar to exercise. However, research shows that exercise cannot extend the lifespan of the unrestricted rodents.	
VI-1 VI-2	使限制卡路里发挥作用的具体机制还不清楚。一种可能是老鼠的新陈代谢率降低了，也即食物在体内较慢地转化成了能量。	The mechanisms underlying the effectiveness of caloric restriction are not clear. One possibility is by decreasing the rodent's metabolic rate, i. e. food transforms to energy slowly in the body.	The mechanisms underlying the effectiveness of caloric restriction are not clear. ……As Masoro (1988a) has pointed out, recent studies have eliminated two hypotheses regarding the mechanism of action of dietary restriction and forced the reconsideration of a third.……The third hypothesis was the idea that dietary restriction increases life span by decreasing

			the metabolic rate. (p.321)
VI-3	这样，所有的生理过程的速度都缓了下来，“生理时间”调慢，因而动物可以活得长一些。	Thus, all the rates of physiological processes slow down, “physiological time” is down tuned, and the animals can live longer.	
VI-4 VI-5 VI-6 VI-7	研究表明，限制卡路里对单位体重的代谢率的影响并不大，不过既然它们吃下的食物较少，即使单位代谢率不变，总的能量输出还是减少了。进一步的研究表明限制卡路里改变了动物的代谢模式。这些改变包括体温降低、脂肪合成降低而葡萄糖合成增加、在进食前有较低的代谢率而进食后有较高的代谢率等等。这些改变可能减少了代谢过程中产生的有害副产物的产量。	Studies showed that CR did not significantly affect the metabolic rate per unit body weight. However, since the intake was reduced, the total energy output was reduced also. Further research shows that caloric restriction changed the animal's metabolic mode. The changes included a lowering of body temperature, a reduced fat synthesis and increased glucose synthesis, a lower than normal metabolic rate before feeding but a higher-than-normal metabolic rate after feeding. These changes could reduce the production of harmful metabolic by-products.	Recent information suggests that this third hypothesis is too simple to be entirely correct, but it is also not entirely wrong. Dietary restriction does affect metabolism, but not in the simple manner envisioned by this theory. Data from the National Institute on Aging–National Center for Toxicological Research (NIA–NCTR) joint biomarker study have shown that caloric restriction induces a major metabolic reorganization in animals (Duffy et al. 1989; Feuers et al. 1991, 1995). This reorganization includes a lowering of core body temperature, a shift away from fat synthesis and toward glucose synthesis, a change in motor activity such that it is concentrated about the feeding time, and an alteration in the body’s metabolic rate such that restricted animals have a lower than normal metabolic rate before feeding but a higher-than-normal metabolic rate after feeding. One result of such a metabolic shift would be the lowering of the organism’s steady-state production of harmful metabolic by-products that result in oxidative stress and damage (Sohal and Weindruch 1996). (p.321)
VII-1 VII-2 VII-3 VII-4 VII-5 VII-6	这些老鼠体内的胰岛素调控葡萄糖的效率也增高了，因此血糖含量较低，比较不容易得糖尿病。血液中多余的葡萄糖会与体内蛋白质发生自由基氧化反应，生成化学结构发生了变化的糖基化蛋白，使这些蛋白质的正常功能受到影响。蛋白的糖基化可能是衰老过程中最普遍的一种化学变化。卡路里受限制的老鼠由于血糖含量低，相应地发生蛋白糖基化的可能性也降低了。而且，卡路里受限制的动物组织中，“自由基清除剂”过氧歧化酶活性增强了，在其一生中体内自由基含量都保持在较低水平。自由基对生命分子的破坏作用被许多研究者认为是导致衰老的主要因素。	The efficiency of insulin regulation of glucose in the rodents increased, so the blood glucose was lower, and the animals were less likely to have diabetes. The excess glucose in the blood could result in radical oxidation reactions with proteins in vivo, generating glycosylated proteins with modified chemical structures, affecting their normal functions. Protein glycosylation is probably the most common chemical reaction during the aging process. Because the calorie-restricted rodents had lower levels of blood glucose, the possibility of corresponding protein glycosylation was also lower. In addition, in the tissue of the calorie-restricted rodents, the activity of free radical scavenger superoxide dismutase was enhanced, and throughout their lifespan, they maintained a lower level of free radicals in their body. Many researchers believe that the damage to life molecules by free radicals is the major factor contributing to the degeneration of old age.	The ability of calorie-restricted animals to satisfy energy requirements with low levels of blood glucose implies that they can minimize the age-related effects of glycosylation. Maintaining an efficient flow of glucose through glycolysis enables calorie-restricted animals to modulate their NADPH pools better. These latter cofactors are known to play an important role in maintaining some of the enzyme systems responsible for the detoxification of free radicals. Thus the ability to maintain “youthful” regulation of this enzyme may spare the organism the harmful effects of glycosylation and free-radical, or oxidative, damage, two harmful processes that can interact synergistically in contributing to the degeneration characteristic of old age (Kristal and Yu 1992). Caloric restriction has been shown to reduce the age-dependent accumulation of advanced glycosylation end products (AGEs) in both red blood cells and skin collagen (Cefalu et al. 1995). In addition, calorie-restricted animals have, in some but not all tissues, a higher level of superoxide dismutase enzyme activity and a lower level of superoxide and/or hydroxide radicals throughout their life span (Lee and Yu 1990). (p.322)
VIII-1	在分子水平上，限制卡路里还导致了许多	At the molecular level, CR results in many other	In addition to these changes in energy metabolism, a multitude

VIII-2	其他的变化，包括肝脏组织中的酶活性发生改变，细胞修复 DNA 的能力增强，DNA 与致癌物的结合能力降低等。大脑中神经介质的含量也发生了改变，表明神经内分泌系统也受到影响。	changes, including changes in liver enzyme activity, enhanced DNA repair activity, decreased binding of DNA to carcinogens, etc. The content of neurotransmitters in the brain was also altered, suggesting the neuroendocrine system was affected.	of other enzyme reactions are affected by diet restriction, including liver enzymes known to be involved in drug metabolism and elimination (Leakey et al. 1989). The complexity of these changes is illustrated by the observation that DNA repair activity increases in diet-restricted rodents (Lipman et al. 1989), while the same treatment simultaneously decreases both normal DNA synthesis and the binding of a chemical carcinogen to DNA in vivo (Chow et al. 1993). The observation that caloric restriction brings about various alterations in brain neurotransmitters suggests neuroendocrine involvement (Kolta et al. 1989). (p.322)
VIII-3 VIII-4 VIII-5	生物学家通常用放着食物的迷宫测试老鼠的学习能力。试验结果表明，限制饮食的中年老鼠和对照组的中年老鼠的学习能力相当，但是限制饮食的老年老鼠却明显胜过对照组的老年老鼠，而保持着中年时期的学习能力。这个研究表明限制卡路里的老鼠虽然发育缓慢，其成年时期的学习能力却没有恶化，反而能将这种能力维持到老年时期。	Biologists usually test the learning abilities of rodents in a standard maze test. The results showed that middle-aged rodents in either diet-restricted group or control group had comparable learning abilities. However, the old diet-restricted rodents were clearly superior to the old controls, maintaining the learning abilities comparable to the middle-aged ones. This study indicates that although the development of the calorie restricted rodents was delayed, their adult learning abilities were not deteriorated, instead, they maintained these abilities well into the aging process.	One unexpected beneficial outcome of diet restriction is its effect on learning performance in mice (Ingram et al. 1987). Both middle-aged and old mice were tested for their learning abilities in a standard maze test. The control and diet-restricted middle-aged adults had comparable learning levels, as indicated by their number of errors per trial. However, the old diet-restricted animals, exhibiting scores comparable to the middle-aged mice, were clearly superior to the old controls. This study is very important because it indicates that the delayed growth and maturation characteristic of diet-restricted animals have no deleterious effect on adult learning abilities but instead maintain these abilities well into the aging process. (p.323)
IX-1 IX-2 IX3 IX-4 IX-5 IX-6	限制卡路里对老鼠的健康和寿命的积极作用已被充分证明了。我们更关心的是：它是不是也适用于灵长类和人类？在八十年代末，美国有两个研究小组开始对罗猴做限制卡路里实验。由于罗猴的寿命大约为 40 年，要知道它们的寿命是否延长了还为时过早。不过，它们在限制卡路里的条件下，出现了与老鼠类似的生理变化。与对照组相比，受限制的罗猴显得更健康，更精瘦，血糖浓度和胰岛素浓度较低，对胰岛素的敏感度增强。	The positive effects of caloric restriction on the health and lifespan of rodents have been fully proven. What we are more interested in is whether it affects primates and human being the same way? At the end of 1980s, there were two groups started the studies on the effects of caloric restriction in rhesus monkeys. Because the lifespan of rhesus monkeys is about 40 years, so it is too early to know whether their lifespan has been extended. However, under reduced caloric intake, the monkeys' physiological changes resembled those of the rodents. Compared with the controls, the restricted rhesus monkeys are healthier, leaner, with decreased blood glucose and insulin levels, and increased insulin sensitivity.	Caloric restriction works wonders for rodents, but what about other mammals? How does caloric restriction affect primates in general and human being in particular? At least two ongoing studies are focusing on the effects of caloric restriction in rhesus monkeys─one located at the National Institute of Aging (Ingram et al. 1990), the other at the University of Wisconsin (Kemnitz et al., 1993). In both studies the treatment is a reduction in caloric intake of about 30 percent. At the end of the first 5 years of the studies, this level of caloric restriction appears to be well tolerated by the animals, and the treatment outcomes identified so far resemble those of the rodent studies (Weindruch 1995b). These results include decreases blood glucose and insulin levels, increased insulin sensitivity, and increased HDL ("good cholesterol") levels. Interestingly, long-term caloric restriction appears not to affect the animals' energy metabolism, percent lean body mass, or percent body fat (Lane et al. 1995). (p.323)
X-1 X-2	我们没法对人类也做类似的实验。战争时期的战俘、饥荒地区的难民被迫忍饥挨饿，饮食热量是受限制了，但是他们也往	We could not do the similar experiment on humans. Prisoners in time of war, refugees in famine areas suffer from caloric restriction, but they suffer from	No well-controlled, long-term studies deal with the effects of caloric restriction on humans. The severe malnutrition too often practiced on prisoners and refugees in time of war

X-3	往营养不良，所以不能说明问题。不过，有一些间接证据表明限制卡路里可能对人体也有积极作用。	malnutrition too, so such data cannot be used as evidence in this question. However, there is some indirect evidence showing CR has possible positive effect on human bodies.	clearly has devastating short- and long-term effects on the health of these people (Mohs 1994a), but such data cannot be used as evidence one way or the other in this question. There is, however, some anecdotal evidence. (p.324)
X-4 X-5	琉球群岛的居民的饮食有充足的营养，但热量低于普通日本人，他们的寿命也长于普通日本人，其百岁寿星的数目是日本其他地区的 2－40 倍。世界各地的百岁寿星也极少有肥胖的。	The diet of the residents of Ryukyu Islands contains adequate nutrition, but the calorie is much lower than the norm in Japan. Their lifespan is also longer than the norm in Japan, the incidence of centenarians is 2-40 times as many as the number on other Japanese islands. Centenarians around the world have rarely been obese.	In the past, the caloric intake of much of the population of Okinawa was much lower than the norm in Japan, but the nutrition of the Okinawans was otherwise adequate. Okinawa has a high incidence of centenarians: 2 to 40 times as many as may be found on any other Japanese island. Other anecdotal evidence suggests that very few, if any, centenarians or other long-lived people have been obese. (p.324)
X-6 X-7 X-8 X-8	在 1991－1993 年间，四男四女在一个与外界隔绝的生态系统“生物圈 2”住了两年。在此期间，他们的饮食营养齐备，但热量大约为一般饮食的 90％。他们的体重明显降低了（男的降低 18％，女的降低 10％），并出现了与卡路里受限制的老鼠类似的生理变化。“生物圈 2”的居民中包括研究衰老的分子生物学、加州大学洛杉矶分校的教授洛伊·瓦尔佛德（Roy Walford）。	In 1991-1993, four men and four women maintained themselves inside an isolated ecosystem Biosphere 2. During this period, they had nutritionally adequate diet, but the calorie intake was about 90% of the normal diet. Their body weights were significantly decreased (18% for men, 10% for women), and they had physiological changes similar to those observed in calorically restricted rodents. The residents of Biosphere 2 included the aging researcher, professor at UCLA, Roy Walford.	Finally, the seven people who voluntarily entered Biosphere 2 for 2 years and reduced their caloric intake while there are reported to have shown physiological changes similar to those observed in calorically restricted rodents (Walford et al. 2002). (p.324) Sealed inside Biosphere 2 in September 1991, four women and four men, including two of the authors, maintained themselves and the various systems for 2 yr, ……Major medical problems encountered during the 2 yr included adaptation to a low-calorie (1800-2200 kcal.d-1 per person) but otherwise nutritionally adequate diet, with substantial weight loss (18% for men, 10% for women), and a declining oxygen atmosphere (down to 14.2%). (Walford RL., et al. 1996. "Biospheric medicine" as viewed from the two-year first closure of Biosphere 2. Aviat Space Environ Med. 67:609-17.)
X-9	他成了限制卡路里长寿法的倡导者和实践者，声称他因此至少能活到 120 岁。	He became the leader and practitioner of caloric restriction movement, claiming he would live at least to 120 years old.	
XI	正常人一天需要从食物中吸收 2000－2500 卡路里的热量，其中大约 30％来自脂肪，30％来自蛋白质和 40％来自碳水化合物。如果把热量供应减低 30％，则每天大约只吸收 1500 卡。这些卡路里将主要来自水果和蔬菜，再加一点淀粉和肉。那些长期严格执行这个饮食计划的人，身体将会变得非常精瘦，总是感到饥饿和寒冷。由于他们体内脂肪已大部分丧失，他们将没有足够的脂肪做为骨骼缓冲物。他们坐下的时候，臀部的骨骼会因为被压着而感到疼痛。由于脚底没有脂肪垫着，走路也会觉得疼。	Normal people need to intake 2,000-2,500 calories from food daily, among the calories, 30% from fats, 30% from proteins, and 40% from carbohydrates. If reducing the calorie supply by 30%, the total intake will be 1,500 calories, they come mainly from fruits and vegetables, plus starch and meats. The people who follow the diet strictly will be skinny, feel cold and hungry all the time. Because the fats in their bodies have been lost completely, they do not have enough fats to cushion the bones. When they sit, the body weight falls on the bones in the buttock, they feel the pain. Because there are no fats in the soles of feet, they feel pain when walking.	

XII-1	很少有人能够长期实施限制卡路里饮食，更不要说实施一辈子了。	Few people are able to practice CR for a long term, let alone whole life.	
XII-2	不过，加州大学河边分校的斯蒂芬·斯宾德勒（Stephen Spindler）实验室在 2001 年 9 月发表的一篇论文认为，在短时间内实施限制卡路里饮食，可能就能取得良好的效果。	However, Stephen Spindler's lab at University of California at Riverside published a paper in September, 2001, and they believed that practice CR for a short-term could achieve a good result.	Dr. Spindler, who is a professor at the Department of Biochemistry at the University of California at Riverside… Stephen Spindler: I think the conclusion you can reach from the paper is that even in very old animals, caloric restriction will very rapidly produce most of the gene expression effects that you see in long-term calorie-restricted animals. (LE Magazine December 2001.)
XII-3	他们用基因芯片技术分析了肝脏中 11000 个基因在年轻小鼠（7 个月大）和年老小鼠（27 个月大）的表达情况，发现其中有 46 个已知基因的表达随着衰老而发生了变化。	Using gene chip technology, they examined the expression of 11,000 genes in young (7 months old) and old (27 months old) mice, and they found that the expressions of 46 known genes changed with aging.	L.E.: …First, you examined over 11,000 genes and found only 46 known genes that went either up or down with age. The old controls were mice that always ate almost all they wanted until being analyzed at 27 months of age. …We also had a young (seven month-old) control group… (LE Magazine December 2001.)
XII-4	与炎症、紧张反应有关的基因，表达增强了，而与代谢有关、抑制细胞生长的基因表达则降低了。	The expressions of the genes involved in inflammation and stress increased, while the expressions of the genes involved in metabolism and block cell division decreased.	We found, …that the older the animals got, the more there was expression of genes that seemed to indicate that the animal was undergoing inflammatory stress and other kinds of stresses. We and others have shown that there is a decline in the expression of enzymes that are involved in drug metabolism in the liver, … and certainly there was a decline in genes that are important for cell division and an increase in genes that tend to block cell division. LE Magazine December 2001.)
XII-5	他们对一批小鼠从小就限制其饮食，热量比正常的减低 40%，一直养到 27 个月老后检测其基因表达情况，发现与年轻的小鼠类似。	They underfed a group of mice by 40% for 27 months, then examine their gene expression, and found the profile was similar to those of young mice.	The long-term calorie restriction mice were those mice who had spent their whole lives being under-fed by 40% until the age of 27 months. We under-fed them first for two weeks by 20%-that is, 20% less than they had been eating previously-and then for two weeks after that we fed them an additional 20% less so that for the second two weeks they were eating 40% less than they had been eating most of their lives. (LE Magazine December 2001.)
XII-6	然后他们对一批 34 个月老的小鼠（相当于人长到大约 80 岁）进行卡路里限制一个月，前两周减低卡路里 20%，后两周再进一步减低 20%。	They then underfed a group of 34 months old mice (equivalent of people who are about 80 years old) for one month, the first two weeks by 20%, the next two weeks by additional 20%.	We took a group of animals that had been allowed to eat almost all they wanted their whole life and we intervened when they were quite old-34 months of age. These mice would be the equivalent of people who are probably 80 years old or older-I'm just guessing at the human equivalent age. We took a group of them and said okay gals, the party's over, it's time to diet. We under-fed them first for two weeks by 20%-that is, 20% less than they had been eating previously-and then for two weeks after that we fed them an additional 20% less so

			that for the second two weeks they were eating 40% less than they had been eating most of their lives. (LE Magazine December 2001.)
XII-7	然后处死这些老鼠检测其基因表达情况，发现也与年轻的小鼠类似。	They then sacrificed all the mice and examined their gene expressions, and found they were similar to those of young mice.	At the end of that time, at 35 months of age, we sacrificed all of the animals. We then compared the gene expression profiles in the livers of these mice to those in four other groups of mice.
XII-8	这就表明限制卡路里不仅能延缓衰老，甚至能在一定程度上逆转衰老过程，而且即使在老年时期短时间地限制卡路里，就能取得显著的良效。	The results indicated that CR not only can delay aging, but also can, to a certain extent, reverse the aging process. Moreover, even if practice CR at an old age for a short time, a good effect could be achieved. 【Note: Fang's above 7 sentences were written based on a cover story in LE Magazine, *Reversing Aging Rapidly With Short-Term Calorie Restriction*.】	Since the animals were already extremely old when you imposed short-term calorie restriction on them, and since their gene expression profiles appeared more like those of young animals after the short-term calorie restriction, it seems inescapable that calorie restriction is not only able to slow age-related changes, but that it is able to reverse age-related changes as well. And it is able to do so over a remarkably short period of time. (LE Magazine December 2001.)
XIII	人类是否也如此？目前还没有人能够证明。即使能够证明，抑制食欲也不是一种吸引人的方案。瓦尔佛德曾在其新书发布会上，请来宾品尝他根据长寿配方制作的膳食。来宾们都觉得难以入口，有的人认为如果要长期吃这样的食物，还不如短命。但是我们研究限制卡路里实验的目的，当然不是为了使其他动物更长寿。我们研究的目的，是为了发现限制卡路里是如何防止衰老和疾病的，从而帮助我们了解动物衰老的机制。如果我们能在分子水平上对衰老过程有深刻的理解，或许能找到更能让人接受的健康长寿的办法。	Whether it works the same way in human? No one is able to prove it. Even if it can be proven, suppressing appetite is not an attractive solution. In a press conference for his new book, Walford asked his guests to taste the food prepared according to the longevity recipe, they all felt it was hard to swallow. Some people even suggested that if they had to eat such food for a long term, they'd rather live shorter. However, our purpose of studying caloric restriction is not for other animals to live longer. Our purpose is to learn how caloric restriction prevents aging and diseases, and understand the mechanism of aging. If we can understand the aging process at molecular level, we might be able to find a more acceptable approach to healthy and longer live.	

Notes

[1] I first termed Fang's so called science popularization "scifool" (科唬) on November 11, 2007. (See:《方舟子论转基因：伪"科普"，真"科唬"》). The term has been well accepted by Chinese people: on March 31, 2013, google the term generates 21,000 results; baidu the term generates 47,300 results.

[2] See "*Special Collection of Fang Zhouzi's Fake Science Popularization*" on AIR-China. (《方舟子伪科普专辑》).

[3] McCay CM, Crowell MF, Maynard LA. 1935. *The effect of retarded growth upon the length of life span and upon the ultimate body size*. J Nutr.10:63–79.

[4] Fang's mistakes: 1. Translating McCay to "麦克凯" (mài kè kǎi), indicating he didn't know the basic English pronunciation rules; 2. Claiming Osborne et al. made their discovery in 1917; 3. Claiming McCay was testing the hypothesis that that longevity is inversely proportional to developmental rate; 4. Claiming McCay divided the rats into two groups; 5. Claiming McCay's underfed rats were calorically restricted all along; 6. Claiming McCay's underfed rats stopped growing; 7. Claiming the average lifespan of the underfed male rats was extended by 50%; 8. Claiming the longest lifespan of McCay's normal rats was 965 days; 9. Claiming McCay raised a rat that lived for more than 1,800 days; 10. Claiming a 1,800 days old rat is equivalent to a 200 years old man. (See: Yi Ming. *The Gigantic Cheater Fang Is Still Cheating: The Second Open Letter to the Editor-in-Chief of Xinhua Daily Telegraph*. Sept. 21, 2012. 亦明：《方巨骗，还在骗──给〈新华每日电讯〉总编辑的第二封公开信》).

[5] Donaldson, H. H. *The rat: reference tables and data*. *Memoirs of the Wistar Institute*, No. 6. Philadephia, 1915. p. 20.

[6] Six-fingered: *Little Fang is now increasingly good for nothing*. Sept. 22, 2012. (六指：《小方现在越来越没出息了》).

[7] The webpage of Prof. Tian's article, *The Gate to Longevity* (《长寿之门》）has been deleted. The related Chinese text is preserved in reference [6] and recorded here:

> Tian: 1935 年美国康奈尔大学的麦卡教授进行的小鼠实验发现，自由饮食的大鼠其骨架在 175 天后就停止了生长，在两年内大部分死去，个别的大鼠活了 965 天；而限制饮食热量的小鼠在 1000 天内骨架还在缓慢地生长，全部活到 4 岁半以上，寿命平均延长了约 50%，有的活到了 1800 多天(相当于人活到 200 岁)。
>
> Fang: 1935 年，美国康奈尔大学的麦克凯等人直接验证是否动物寿命真的与发育速度成反比。在大鼠断奶后,他们给其中的一组提供完备的营养物质，但是严格限制其饮食，让它们一直处于饥饿中，而另一组老鼠则任其吃饱。饮食受限制的老鼠发育几乎停止，身体也不再长大，一些老鼠夭折了，但是存活下来的老鼠中，寿命明显增长了。雄鼠所受的影响更显著，寿命平均延长了约 50%。喂食正常的老鼠中，寿命最长的为 965 天，而限制喂食的老鼠，有的活到了 1800 多天(相当于人活到 200 岁)。
>
> Tian: 人类很难做限食实验，只能靠间接证据表明限制热量可能对人体有积极作用。研究发现，琉球群岛的居民饮食富有营养，但热量低于普通日本人，他们的寿命长于普通日本人，其百岁寿星的数目是日本其他地区的 2~4 倍。又如在 1991~1993 年间，科学家让四男四女在一个与外界隔绝的生态系统"生物圈 2"住了两年。在此期间，他们的饮食营养齐备，但热量大约为一般饮食的 90%。结果他们的体重都明显地降低了(男的降低 18%，女的降低 10%)，并出现了与卡路里受限制的小鼠类似的生理变化。
>
> Fang: 不过，有一些间接证据表明限制饮食热量可能对人体也有积极作用。琉球群岛的居民的饮食有充足的营养，但热量低于普通日本人,他们的寿命也长于普通日本人，其百岁寿命的数目是日本

其他地区的 2～40 倍。世界各地的百岁寿星也极少有肥胖的。在 1991～1993 年间，四男四女在一个与外界隔绝的生态系统“生物圈 2”住了两年。在此期间，他们的饮食营养齐备，但热量大约为一般饮食的 90%。他们的体重明显降低了(男的降低 18%，女的降低 10%)，并出现了与卡路里受限制的老鼠类似的生理变化。

[8] Fang’s original Chinese: “北京大学生命科学学院教授、北京大学老龄问题研究中心科研部主任田清涞《长寿之门——需限制热量，增强营养》一文（登于《健康指南》2009 年 10 月，）主要参考我写于 2002 年的《吃得少活得老》（登于《环球》半月刊 2002 年第 21 期，后收入《长生的幻灭》《科学成就健康》），其中“四、长寿与抗衰老事例”一段全文照抄，只是自作聪明地把“2～40 倍”错误地改成“2～4 倍”。最近关于限制热量与衰 老的关系的研究有新进展，对传统的观点提出了挑战，我因此又写了一篇《吃得少活得老？》登在《新华每日电讯》上，其中部分段落改自旧文，包括被田清涞抄袭的那段。有方黑据此反过来指责我抄袭田清涞，亦明、韩寒之父韩仁均、孙海峰、不加 V（木子美）、易天等资深方黑更是跳得厉害。我的科普文章多次被人抄袭， 像这样只是抄袭一两段的，我本懒得计较，但方黑们既然借此大做文章倒打一耙，我还是说明一下。”

[9] See: http://www.xys.org/forum/db/10/137/133.html; http://www.xys.org/xys/ebooks/others/science/dajia13/tianqinglai.txt; http://xysblogs.org/fangzhouzi/archives/9985; http://blog.sina.com.cn/s/blog_474068790102e2kr.html; http://fangzhouzi.blog.hexun.com/80078351_d.html; http://t.hexun.com/fangzhouzi/19897858_d.html; http://blog.caijing.com.cn/expert_article-151278-43438.shtml; http://t.sohu.com/m/5100188570.

[10] hqabc_’s original Chinese: “@亦明 2010 这次是当君子道歉呢，还是当睁眼瞎，继续骗弱智？” (See: 2012-9-25 05:22).

[11] Fang’s original Chinese: “Root-Bernstein 博士污蔑我从新语丝拿工资发博文、我的文章 90%是其论文内容、我逐字照抄其文字和例子（其实我已做恰当的改写并举自己的例子）、我剽窃其论文和侵犯其版权，全是谣言，他授权“方学家”诋毁我，我不起诉他已算客气，他还敢扬言告我？这是我对该事件最后表态，以后有人再问一概拉黑。” (See: 2011-8-22 10:42). “关于‘方舟子剽窃美国教授’一事自去年 10 月份以来我已在微博和博客上澄清过很多次，最近的一次见：http://t.cn/aRbsmy 还有其他几篇，有疑问的自己去看。我不再回答这个问题，再问就拉黑。” (See: 2011-8-22 11:12).

[12] For example, Fang wrote on Mar. 25, 2006: “So, Wu Guosheng’s accusation against me of stealing coyotejoy’s article is totally infamatory. ……He obviously doesn’t know anything about the difference between science popularization articles and academic research papers, and he applied the standards for the latter to the former. Academic research papers are required to give citations for every sentence, and required to give a detailed reference sources, but there is no such a requirement for science popularization articles or essays. This is true not only in China, but also all over the world.” (Original words: “可见吴国盛指控我抄袭 coyotejoy 文章，完全是侮蔑。……他显然完全不懂科普文章与学术论文的区别，以学术论文的标准来衡量科普文章。学术论文要 求句句有出处，必须详细列出文献来源，但是科普文章、随笔却没有这样的要求。不仅是中国的科普文章、随笔如此，全世界的科普文章、随笔也都如此。” Fang Zhouzi. *A Reply to American Wu Guosheng*. XYS20060325. 方舟子：《答美国的“吴国盛”》, XYS20060325). For more examples, See: *Xin Ge: A few comments on Dr. Zachary Burton’s “Support for Dr. Shi-min Fang”*, Part IV.

[13] Wang Houde, Wang Wenlu, Bai Jiaxiang, and Tian Qinglai. 1986. *The Effect of Food Intake on Mice’s Lifespan*. *Chinese Journal of Gerontology* 1986 (4):48-50. (王厚德、王文录、白家祥、田清涞：《食量对小鼠寿命试验的干扰》，《老年学杂志》1986 年 4 期 48-50 页.) Tian Qinglai and Tian Feng. *Aging and anti-aging science*. China Social Press, 2009. (田清涞、田枫：《衰老与抗衰老学》，中国社会出版社 2009 年版.) Tian Qinglai and Tian Feng. *Nutrition for the elderly*. China Social Press, 2009. (田清涞、田枫：《老年营养学》，中国社会出版社 2009 年版.) Tian Qinglai and Tian Feng. *Traditional and modern science of health preserving*. China Social Press, 2009. (田清涞、田枫：《传统与现代养生学》，中国社会出版社 2009 年版.)

[14] For example, an article published on Dec. 2, 2005 says: “In the Okinawa Island, the incidence of centenarians is several times as many as the number on other Japanese islands.” (Original Chinese: “日本冲绳岛的百岁老人数比

其他地区高好几倍". See: Zhang Jiaqing. *Eat Less, Live Longer*. *Xinmin Evening News* Dec. 2, 2005. 张家庆:《吃得少活得长》, 2005 年 12 月 2 日《新民晚报》.) Also, in *A Mystery of Life & Death: Secrets of How to Live 120 Years* (Zhaohua Press, 2007, pp.136-138), there is similar content.

[15] See: Table 1 in McCay, C. M. and M. F. Crowell. 1934. *Prolonging the life span. Sci. Monthly* 39: 405–414, and Table 2 in McCay, C., M. Crowell and L. Maynard. 1935. *The effect of retarded growth upon the length of life and upon ultimate size*. *J. Nutr*. 10: 63–79.

[16] Osborne TB, Mendel CB, Ferry ER. 1917. *The effect of retardation of growth upon the breeding period and duration of life in rats. Science* 45:294–295.

[17] Aristotle: "Speaking generally, the longest-lived things occur among the plants, e.g. the date-palm. Next in order we find them among the sanguineous animals rather than among the bloodless, and among those with feet rather than among the denizens of the water. Hence, taking these two characters together, the longest-lived animals fall among sanguineous animals which have feet, e.g. man and elephant. As a matter of fact also it is a general rule that the larger live longer than the smaller, for the other long-lived animals too happen to be of a large size, as are also those I have mentioned." (See: Aristotle. *On Longevity and Shortness of Life*. Translated by G. R. T. Ross.)

[18] Ukrainian physiologist and gerontologist Vladimir V. Frolkis wrote: "Since Aristotle and later Buffon, Shmalgauzen, and Bidder, an important role in species-specific life span determination has been attributed to growth rate." (Vladimir V. Frolkis, Khachik K. Muradian. *Experimental Life Prolongation*. CRC Press, Oct 24, 1991. p.170.)

[19] Zachary Burton. *Support for Dr. Shi-min Fang*.

[20] Xiong Lei. 2001. *Biochemist Wages Online War Against Ethical Lapses*. *Science* 293:1039.

[21] See, for example, Reuter, G. & Gabius, HJ. 1999. *Eukaryotic glycosylation: whim of nature or multipurpose tool? Cell. Mol. Life Sci.* 55:368–422; Varki A, Cummings RD, Esko JD, et al., editors. *Essentials of Glycobiology*. 2nd edition. Cold Spring Harbor Laboratory Press; 2009.

[22] Anonymous. 1995. *Dietary Manipulation Of Aging*. *Life Extension Magazine*, June, 1995; Saul Kent. 2001. *Aging Research Becomes A Science*. *Life Extension Magazine*, December 2001.

[23] Anonymous. 2001. *Reversing Aging Rapidly With Short-Term Calorie Restriction*. *Life Extension Magazine*, December 2001.

[24] Fang's original Chinese: "在生物科学的领域，没有相关博士学位的人是没有任何资格从事科研的，那些只有硕士学位的人尚且只能干干技术活，更不要说其他人了。" (See: Fang Zhouzi. *The "Anthropological research" fellows*. XYS19990801. 方舟子：《"人类学研究"的难兄难弟》, XYS19990801.)

[25] Fang's original Chinese: "事实上我认为本科生甚至硕士研究生都没有必要写毕业论文。" (See: Fang Zhouzi. *College students need not write graduation thesis. Xinhua Daily Telegraph*. April 29, 2011. 方舟子：《大学生不必写毕业论文》，2011 年 4 月 29 日《新华每日电讯》).

[26] Jean E. Pierog. *RECIPE FOR LONGEVITY*. *healthlinks.net Newsletter*.

[27] Weindruch R, Walford RL, Fligiel S, Guthrie D. 1986. *The retardation of aging in mice by dietary restriction: longevity, cancer, immunity and lifetime energy intake.* *J Nutr*. 116:641-54.

[28] Yi Ming. *Old Stealer, Gigantic Cheater, Stealing First, Cheating Later: The Third Open Letter to Mr. Xie Guoji, The Editor-in-Chief of Xinhua Daily Telegraph*. (亦明：《老偷巨骗，先偷后篇：给〈新华每日电讯〉总编辑解国记先生的第三封公开信》).

THE PREVIOUS PARTS OF THE OPEN LETTER