南區青教組教員進修會
106.6.18 於喜樹

從醫學基因看同性戀

羅大德執事

同性戀的起源：天生抑或後天

香港的同志團體一直聲稱同性戀是與生俱來的，這種性傾向不應受到歧視。他們甚至要求制訂《反性傾向歧視條例》，防止同性戀者的「權利」被「剝奪」。究竟同性戀是天生的還是後天的呢？本文嘗試從生物學、心理學及社會學的角度分析同性戀的起源問題，並深入檢視一些支持同性戀天生論的遺傳學、產前神經激素、神經解剖學等方面的「證據」，盼望能減少和糾正一般人對同性戀起源的誤解及不正確觀念，引發這方面更多的討論，從而讓大眾對同性戀有更全面的認識。

同性戀的起源

甲，生物學對同性戀起源的解釋

1. 遺傳學的證據

A. 簡接的基因研究

雖然同性戀與家庭有關，但這並不表示同性戀是遺傳的。很多遺傳學家對同卵雙生兒及異卵雙生兒進行研究，試圖證明遺傳基因跟同性戀有關，其中最具影響力的是Bailey和Pillard的研究（1991, 1993）。他們在同性戀社群中找出一些有雙生兄弟姊妹的同性戀者，調查他們的雙生兄弟姊妹的性傾向。報告指出男性同卵雙生兒（Identical Twins）的一致比率[註一]是 52%，即 52%同卵雙生兒兩兄弟都有同性戀偏好，男性異卵雙生兒（Fraternal Twins）的一致比率是 22%，女性同卵雙生兒的一致比率是 48%，女性異卵雙生兒的一致比率是 16%。

Bailey 和 Pillard 的研究有一令人關注的地方，就是他們在一些支持同性戀的雜誌及小報刊登廣告招募雙生兒。同性戀者爲了這項研究能出現一些對他們有利的結果，於是有同性戀雙生兄弟姊妹的同性戀者便很願意參與研究，而沒有同性戀雙生兄弟姊妹的同性戀者便不大願意參與，樣本偏誤（Sample Bias）因而出現。

Bailey 之後所作的研究有力地推翻初期的研究結果。他獲准向在澳洲雙生兒登記處登記了的雙生兒寄出問卷，調查他們的性偏好及性經驗。Bailey 這個研究（Bailey, Dunne & Martin (2000)）的結果，男性同卵雙生兒的一致比率是 20%，男性異卵雙生兒的一致比率是 0%，女性同卵雙生兒的一致比率是 24%，女性異卵雙生兒的一致比率是 10%。

上述的研究結果指出了初期的研究明顯出了樣本偏誤，它們亦令人質疑遺傳因素對同性戀的形成有多大影響。Bailey 和他的同事承認新的研究結果未能提供有力的證據支持遺傳因素對同性戀傾向的形成起著重要作用的看法。換句話說，遺傳或許並不是同性戀形成的重要因素。

[註一] 基因相關的人共同擁有某特徵的比率稱爲一致比率 (Concordance Rate)。

B. 直接的基因研究

Dean Hamer 和其他研究員做了一個研究（Hamer, Hu, Magnuson, Hu & Pattatucci, 1993），他們聲稱即將發現「同性戀基因」。研究員先訂下一個假說，認爲有多種類型的同性戀，其中一種能通過母親的基因（即 X 染色體）遺傳給下一代。他們從一個愛滋病治療計劃挑選 76 個男人，這些男人都有同性戀兄弟，他們的母方家族大多有同性戀傾向，而父方家族則沒有。研究員檢查這一群男人的 X 染色體，發現 40 對同性戀兄弟中，33 對兄弟的 X 染色體某區域的模樣是相同的，遠高於預期的隨機併存水平（Random Concurrence Level），研究員便假定這區域涉及決定人類的性傾向。

根據 Stanton & Yarhouse（2000），Hamer 的研究有其問題和限制。首先，其他研究隊進行相同的研究，可是不能得出相同的實驗結果。其次，Hamer 和他的同事並非找到「同性戀基因」，因爲他們所指的「同性戀」不是一般的同性戀，他們只能從某一類男同性戀者發現這染色體標記，而這些男同性戀者都有一個同性戀兄弟，並且他們的家族出現了強烈的「母體傳遞」（Maternal Transmission）。事實上，兩兄弟都是同性戀者的情況並不普遍，我們亦不知道有這種母體傳遞的男同性戀者的數目有多少。最後，研究員發現這種染色體標記並不是引致同性戀的必需的（Necessary）或足夠的（Sufficient）條件——有這染色體標記並不表示就是同性戀者，所以不是一個足夠的條件；沒有它又不表示就不是同性戀者，所以不是一個必需的條件。

Jones 和 Yarhouse（2000）指出，如果 Hamer 的發現是可靠的，可能表示了一些染色體標記能夠使人發展出某種性格、性情或特徵，而這種性情或特徵成爲了部份同性戀者被同性吸引的原因。這些染色體標記並不會決定人的性傾向，它們只能夠使人有較大的機會發展出同性戀傾向，間接引致同性戀出現。

2. 產前神經激素假說（Prenatal Neurohormonal Hypothesis）

Ellis 和 Ames (1987)根據他們的動物實驗結果，提出懷孕期的第二至第五月，胚胎受到多種性激素刺激，性傾向便從此定型，但是人類的情況不一定和動物的相同。Money (1987)認爲單憑懷孕期的激素作用，並不足以注定一個人永遠是同性戀者，還要考慮他/她的成長經歷，況且，沒有證據顯示所有同性戀者都受到產前激素作用的影響。產後激素的研究指出，同性戀者和異性戀者的激素成份和生理結構都沒有明顯的分別。

3. 神經解剖學（Neuroanatomy）的證據

Le Vay (1991)對屍體進行檢驗，發現同性戀者的 INAH-3（腦部一種組織）較一般人細小。這個發現帶出多個問題。但研究所採用的方法出了嚴重問題。首先，他只檢驗了 35 具屍體，這數目對於進行研究實在是太少了。另外，LeVay 按著死者的醫療記錄來分辨他們的性傾向，凡醫療記錄上沒有被註明是同性戀者的人

士，便被列爲異性戀者。其實，接近一半死者的性傾向是不明確的。還有，愛滋病病毒及愛滋病療法均可能改變 INAH3 的大小及形狀，我們不能肯定他的研究結果是跟同性戀有關還是跟愛滋病或其療法有關。最後，研究員無法確定是細小的 INAH-3 導致同性戀傾向，還是同性戀傾向導致 INAH-3 出現變化。

乙，心理學對同性戀起源的解釋

Bieber (1976)根據他的臨床經驗及對一百個男同性戀者的調查，提出男人成爲同性戀者，是由於童年的成長受到嚴重干擾。這些男人的爸爸可能對他們漠不關心、又常常拒絕他們，他們心中便暗暗渴望跟男性有親密的關係。他們的媽媽可能太過愛護他們，甚麼都過問、甚麼都管束，以致他們不能建立完整的男性身份（Male Identity）。

關於同性戀的形成，行爲假說（Behavioral Hypotheses）指出，一個人童年的學習經驗（包括性經驗）塑造出他/她的性傾向。一個曾被同性戀者性侵犯的兒童，可能會將那次經歷作爲日後性幻想的依據，並且將自己介定爲同性戀者。

Storm (1981)指出性傾向通常在青春期確立。男孩子和女孩子一般都在青春期才有較多接觸機會，男孩子的性慾這時候開始旺盛，能有助他們發展出異性戀傾向。但是，男孩子的性慾如果過早旺盛，他們有可能將身邊的同性朋友作爲對象，發展出同性戀傾向。女孩子出現這種情況的機會較低，因爲她們的性慾較遲才旺盛。

丙，社會學對同性戀起源的解釋

Kinsey 等學者（1948,1953）認爲，童年如果有深刻的性經驗，那個經驗會有重複的傾向。如果那是一個同性性經驗，則日後很可能發展出同性戀傾向。

Bell 等學者（1981）做了路徑分式（Path Analysis），發現「童年性別不協調」（Childhood Gender Nonconformity）是男性成年性偏好的重要預兆。他們又認爲成年同性戀傾向的發展，性感受比性活動起著更重要的作用。

Van Wyk 和 Geist (1985)進行了路徑分析，發現青春期後期的社交經驗是成年同性戀傾向的重要預兆，青春期的經驗會被帶進成年階段。他們認爲樣貌長得有點像異性的兒童，可能會以爲自己是個同性戀者。如果這些兒童受到同性同伴排斥，他們日後可能不會對異性產生興趣。Van Wyk 和 Geist 又指出，兒童的性嬉戲如果太過分，如涉及手淫、口交、性交等，這些經驗都會形成性興奮和性滿足，導致兒童繼續幻想和參與同性的性行爲，使到成年發展出同性的性偏好。

不少人都認爲女同性戀者的關係是基於情感而不是情慾，心理治療事提出女同性戀關係有「融合」的現象。Briar Whitehead (1996)認爲女同性戀者對自己的女性身份（Female Identity）作了防衛性的拒絕，以致她們從與同性伴侶的親密關係尋找彌補或補償。所以，在女同性戀者關係的表面「融合」裡，蘊含著對自我女性身份的抗拒。

Moberly (1983)說兒童如果在幼年時遇到特別事件，以致他/她對同性父母的依戀受到破壞，他/她的性別身份認同和角式模仿會因而受到妨礙[註二]。由於他/她對同性父母的需要——就是愛、依靠和認同——仍然存在，這些需要如果被厭惡和敵意情緒充斥，便會產生「同性矛盾情感」(Same-Sex Ambivalence)，這種情感會流露在和同性伴侶的關係中，出現同性戀情況。關於女同性戀者的童年和青春期的研究結果都引證了Moberly的論說，並且指出女同性戀者通常和男性有很惡劣的關係。

Bell, Weinberg 和 Hammersmith (1981)從多個研究發現，女同性戀者和媽媽的關係通常都較一般女性惡劣。Nicolosi (1991)認爲兒童跟同性父母不和，會使他們融入同性群體時出現困難。女同性戀者回憶童年及青春期跟同性同伴的關係時，通常會感到很痛苦。Bell 等學者（1981）認爲「童年性別不協調」——覺得自己跟同性同伴「不同」——是女性日後發展出同性戀傾向的第二重要預兆。

Briar Whitehead (1996)指出很冷漠和很粗暴的男人都能夠令女人失去異性戀的興趣。女人如果曾和男人有惡劣的相處經驗，或曾被男人性虐待，她們很可能選擇同性戀。Van Wyk 和 Geist (1985)發現有些女孩子被其他女性撫摸後學會了手淫，並且發展出同性戀傾向。她們長大後表示只有女性能夠在性方面吸引她們。Bell 等學者（1981）的路徑分析顯示，女同性戀者與母親的惡劣關係、童年性別不協調、以及青春期與同性的性行爲都導致成年出現同性戀傾向。

七，總結

盼望以上關於同性戀成因的探討——它是先天的還是後天的——能夠讓讀者對這課題有一整全的概念。遺傳學家對基因的簡接及直接的研究都出現了不少漏洞，他們不能得到有力的證據支持任何結論。遺傳學的權威學者質疑這些研究的正確性，並指出生物學現在還沒有充分論據建立同性戀傾向是與生俱來的理論（Byne & Parsons, 1993, p.228）。直到現在，各項研究報告還未能讓生物學發展出任何性傾向理論，關於同性戀成因的生物學科研結果仍不能確立任何結論，換句話說，生物因素不會「導致」同性戀，它亦不是在生命初期便被注定了的。

很多學者認爲社會和心理因素對同性戀傾向的發展起著更重要的作用。Byne 和 Parsons（1993）就性傾向提出一個「互相作用模型」(Interactional Model)，他們認爲基因能影響人的個性發展，而人的個性則影響他/她怎樣面對還境，性傾向就在他/她成長時顯露出來。Anne Fausto-Sterling（1985）指出一個行爲可能是由很多因素導致的，生物因素雖然可以影響行爲，但行爲亦能反過來改變人的生理機能。Ruth Hubbard（1990）觀察到社會對恰當性行爲及性活動的規範，幾乎能夠對身體每個系統都構成影響。這些發現都指出基因或許能影響同性戀傾向的形成，但社會和心理因素對同性戀傾向的發展起著更大的作用，而人的同性戀經驗及行爲亦能導致他/她的生理機能出現變化。

[註二]兒童從同性父母獲得依戀（Attachment）、依靠（Dependence）和認同（Identification），然後進行性別身份認同（Gender Identification）和角色模仿（Role-Modeling）的成長步驟。

編輯及撰寫：臨床心理學家葛琳卡博士
翻譯：胡道航

參考書目

Bailey, J. M, Dunne, M. P & Martin, N. G. (2000). Genetic and environmental influences on sexual orientation and its correlates in an Australian twin sample. Journal of Personality and Social Psychology78, 33

Bailey, J. M. & Pillard, R. C. (1991). A genetic study of male sexual orientation. Archives of General Psychiatry,48, 1089-1096.

Bell, A. P., Weinberg, M. S., & Hammersmith, S. K. (1981). Sexual preference: Its development in men and women. Bloomington, Indiana: Indiana University Press

Bieber, J. (1976). A discussion of homosexuality: The ethical challenge. Journal of Consulting and Clinical Psychology,44, 163-166.

Byne, William & Parson, B. (1993). Human sexual orientation: The biological theories reappraised. Archives of General Psychiatry,50. 228-239.

Ellis, L. & Ames, M. A. (1987). Neurohormonal functioning and sexual orientation: A theory of homosexuality-heterosexuality. Psychological Bulletin,101, 233-258.

Fausto-Stering, A. (1985). Myths of gender: Biological theories about women and men. New York: Basic Books Inc.

Hamer, D. H, Hu, S., Magnuson, V. L., Hu, N. & Pattatucci, A. M. L. (1993). A Linkage Between DNA Markers on the X Chromosome and Male Sexual Orientation. Science261, 320-26

Hubbard, R. (1990). The politics of women's biology. New Brunswick: Rutgers University Press.

J. Michael Bailey, Richard C. Pillard, Michael C. Neale and Yvonne Agyei (1993), "Heritable Factors Influence Sexual Orientation in Women, Archives of General Psychiatry,50, 217-23

Kinsey, A. C., Pomeroy, W. B., & Martin, C. E. (1948). Sexual behavior in the human male. Philadelphia: W. B. Saunders.

Kinsey, A. C., Pomeroy, W. B., Martin, C. E. & Gehhard, P. H. (1953). Sexual behavior in the human female. Philadelphia: W. B. Saunders.

LeVay, S. (1991). A Difference in the Hypothalamic Structure Between Heterosexual and Homosexual Men. Science253, 1034-37

Moberly, E. R. (1983). Psychogenesis. The early development of gender identity. London: Routledge & Kegan Paul Ltd.

Money, J. (1987). Sin, sickness, or status? Homosexual gender identity and psychoneuroendocrinology. American Psychologist,42, 384-399.

Nicolosi, J. (1991). Reparative therapy of male homosexuality. Northvale, NJ: Jason Aronson Inc.

Stanton, L. J. & Yarhouse, M. A. (2000). Homosexuality: The use of scientific research in the church's moral debate. Illinois, InterVarsity Press.

Storms, M. (1981). A theory of erotic orientation development. Psychological Review,88, 340-353.

Van Wyk, P. H., &Geist, C. S. (1985). Psychosocial development of heterosexual, bisexual and homosexual behavior. Archives of Sexual Behavior,13, 505-544.

Whitehead, Briar (1996). Lesbianism: Causality and compassion. Journal of Psychology and Christianity,15 (4), 348-363.

(本文收在關啓文、戴耀廷、康貴華等著，《平權?霸權?——審視同性戀議題》，香港：天地圖書，2005 年。)

RESEARCH NEWS

Evidence for Homosexuality Gene

A genetic analysis of 40 pairs of homosexual brothers has uncovered a region on the X chromosome that appears to contain a gene or genes for homosexuality

How much of sexual orientation is determined by a person's genes, and how much by familial and cultural influences? That has proved to be an exceptionally controversial question. Several recent studies of twins and adoptive siblings have pointed toward a large genetic component in homosexuality, implying that a gene or genes should exist that create a predisposition for homosexuality, but there was no direct proof. Now, a team of geneticists at the National Cancer Institute has come closer to that proof.

On page 321, Dean Hamer and his colleagues Stella Hu, Victoria Magnuson, Nan Hu, and Angela Pattatucci report linking some instances of male homosexuality to a small stretch of DNA on the X chromosome. If the finding can be confirmed, it might eventually lead to a better understanding of the biological basis of homosexuality and of sexual orientation in general.

No one is breaking out the champagne just yet, however. The field of behavioral genetics is littered with apparent discoveries that were later called into question or retracted. Over the past few years, several groups of researchers have reported locating genes for various mental illnesses—manic depression, schizophrenia, alcoholism—only to see their evidence evaporate after they assembled more evidence or reanalyzed the original data. "There's almost no finding that would be convincing by itself in this field," notes Elliot Gershon, chief of the clinical neurogenetics branch of the National Institute of Mental Health. "We really have to see an independent replication."

Despite the caution, researchers familiar with the work say this study appears to have a very good chance of holding up because it avoids some of the methodological problems of earlier work. One way or the other, the verdict may be in before the end of the year since a replication can probably be performed quickly.

To look for a possible homosexuality gene, Hamer and his colleagues took a two-step approach. First they recruited 76 homosexual men and traced out pedigrees for each, determining which other members of each family were themselves homosexual. They found 13.5% of the gay men's brothers to be homosexual—much higher than the rate of 2% or so that the Hamer group measured in the general population. (While this is lower than previous estimates of 4% to 10%, other recent studies have come up with similar low figures.) Earlier studies had also found that brothers of homosexual men are more likely to be homosexual than are men in the general population.

But once Hamer and colleagues ventured outside the immediate family, they found something new. "When we collected the family histories," Hamer says, "we saw more gay relatives on the maternal side than on the paternal side." In particular, they found homosexuality to be significantly more common among maternal uncles of gay men and among cousins who were sons of maternal aunts than it is among males in the general population.

This implied that, for some male homosexuals at least, the trait is passed through female members of the family. And this in turn gave the researchers an obvious place to start looking for a homosexuality gene: the X chromosome, the only chromosome inherited exclusively from the mother.

To search for such a gene, Hamer recruited 40 pairs of homosexual brothers, took DNA samples from each, and performed a genetic linkage analysis using gene markers. The idea behind the analysis is simple: On average, each pair of brothers will have about half the DNA on their X chromosomes (and other chromosomes) in common. If both brothers are homosexual because they inherited a particular gene on the X chromosome, the gene must lie somewhere in the shared sections of the chromosome, which can be identified by the gene markers. The researcher examines many pairs of brothers, looking for a stretch of DNA that all or most of them have in common. If such a stretch exists, then it probably contains the target gene.

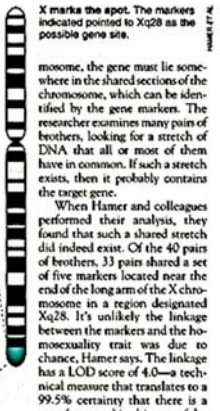
When Hamer and colleagues performed their analysis, they found that such a shared stretch did indeed exist. Of the 40 pairs of brothers, 33 pairs shared a set of five markers located near the end of the long arm of the X chromosome in a region designated Xq28. It's unlikely the linkage between the markers and the homosexuality trait was due to chance, Hamer says. The linkage has a LOD score of 4.0—a technical measure that translates to a 99.5% certainty that there is a gene (or genes) in this area of the X chromosome that predisposes a male to become homosexual.

Hamer warns, however, that this one site cannot explain all male homosexuality. Although his pedigree analysis showed that the homosexuality trait is usually maternally inherited, he did see some families where the trait seemed to be passed paternally. And even among his 40 sets of brothers, chosen so that there was no evidence of the trait passing through male family members, seven sets of brothers did not share the stretch of Xq28 where the gene appears to lie. Instead, Hamer says, it seems likely that homosexuality arises from a variety of causes, genetic and perhaps environmental as well.

Still, researchers can hardly wait to get their hands on the gene in order to study just what it does. "It's very exciting," says Michael Bailey of Northwestern University in Chicago, co-author of a study 2 years ago that found half of the identical twins of gay men to be themselves gay. "If we can find a gene for sexual orientation, we can start to find out what the gene does."

The list of questions to be asked about

Gene team. Dean Hamer, and (from left) Stella Hu, Nan Hu, Angela Pattatucci, and Victoria Magnuson are studying the genetics of sexual orientation.



X marks the spot. The markers indicated pointed to Xq28 as the possible gene site.

Abstract

The role of genetics in male sexual orientation was investigated by pedigree and linkage analyses on 114 families of homosexual men. Increased rates of same-sex orientation were found in the maternal uncles and male cousins of these subjects, but not in their fathers or paternal relatives, suggesting the possibility of sex-linked transmission in a portion of the population. DNA linkage analysis of a selected group of 40 families in which there were two gay brothers and no indication of nonmaternal transmission revealed a correlation between homosexual orientation and the inheritance of polymorphic markers on the X chromosome in approximately 64 percent of the sib-pairs tested. The linkage to markers on Xq28, the subtelomeric region of the long arm of the sex chromosome, had a multipoint lod score of 4.0 (P = 10(-5), indicating a statistical confidence level of more than 99 percent that at least one subtype of male sexual orientation is genetically influenced.

A Genetic Study of Male Sexual Orientation

J. Michael Bailey, PhD; Richard C. Pillard, MD

Arch Gen Psychiatry. 1991;48(12):1089-1096. doi:10.1001/archpsyc.1991.01810360053008.

ABSTRACT

• Homosexual male probands with monozygotic cotwins, dizygotic cotwins, or adoptive brothers were recruited using homophile publications. Sexual orientation of relatives was assessed either by asking relatives directly, or when this was impossible, asking the probands. Of the relatives whose sexual orientation could be rated, 52% (29/56) of monozygotic cotwins, 22% (12/54) of dizygotic cotwins, and 11% (6/57) of adoptive brothers were homosexual. Heritabilities were substantial under a wide range of assumptions about the population base rate of homosexuality and ascertainment bias. However, the rate of homosexuality among nontwin biological siblings, as reported by probands, 9.2% (13/142), was significantly lower than would be predicted by a simple genetic hypothesis and other published reports. A proband's self-reported history of childhood gender nonconformity did not predict homosexuality in relatives in any of the three subsamples. Thus, childhood gender nonconformity does not appear to be an indicator of genetic loading for homosexuality. Cotwins from concordant monozygotic pairs were very similar for childhood gender nonconformity.

Memo

DATE :

MEMBER :

PLACE :

Memo

DATE :

MEMBER :

PLACE :