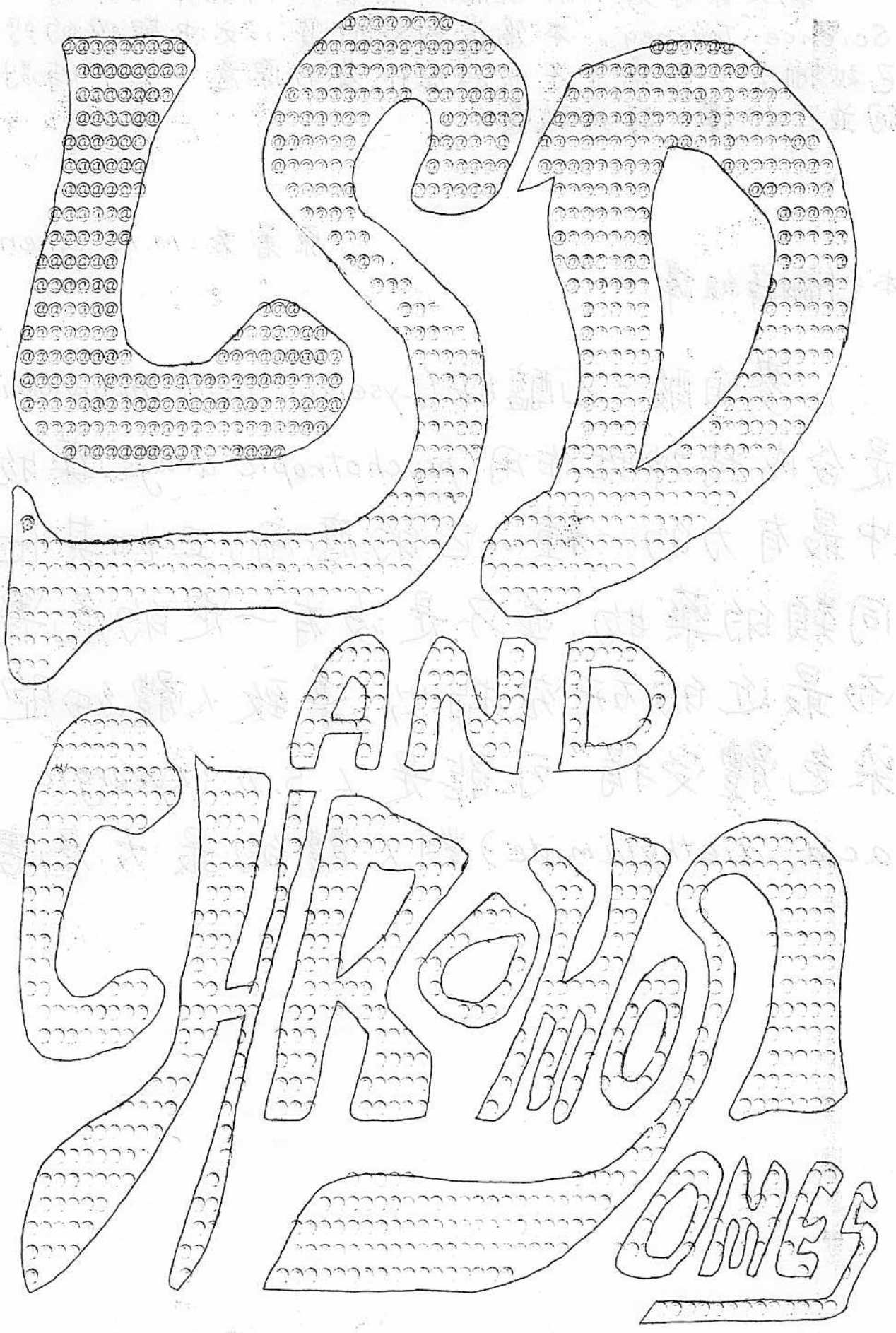

LSD
AND
CHROMOSOMES

本文原作者 M.M. Cohen，發表於 1968年九月的 Science Journey。本篇是節譯性質，文中艱深的段落已被刪去，而為了不曲譯原作者的原意，文中特別名詞並不作譯。謹此致意。

原著者：M.M. Cohen

本刊翻譯組譯

麥角酸二乙醯胺(Lysergic acid diethylamide)是合成精神性作用(psychotropic drugs)藥物中最有力的一種。它的應用，正如其他同類的藥物，並不是沒有一定的危害。而最近的研究指出"導致人體細胞染色體受損"可能是 L.S.D.(Lysergic acid diethylamide)對人體的最大危害。

L.S.D. 的新舊觀

由於不明 L.S.D. 對用者的影響和它們可能的持久性，今日青少年濫復此藥的趨勢就產生一種警号般的意識。美國過去的三年內，不良而甚至惡性的 L.S.D. 反應數字迅速增加。最嚴重的症狀包括持久的精神性反應；停用此藥後的週期自發性歷練(spontaneous recurrent L.S.D. experience)；障礙性反應如過度驚慌(acute panic)，抑鬱(depression)和錯亂(confusion)症狀；和比較少見的反應如自殺、殺人和痙攣。至於會做成中毒(toxicity)和"毒癮"(addiction)的設議則尚未被証實。

以前 L.S.D. 的"mind-expanding"特性被寄望治療酒精中毒(alcoholism)和其他某些精神病症。但今日這種寄望都不復有了，代之而起的是增加應用此藥所產生的嚴重問題。例如其可能做成的不良影響和後果。好久以前，科學機構就對「L.S.D. 的表面心理性影響是否完全有利？」這個問題發生興趣。近期經幾方面的研究，指出 L.S.D. 除了影響和作用於腦部外，還能對其他某些器官有持久的心理和可能的嚴重生理效應。而作者和其他一些實驗室則皆指出 L.S.D. 能導致人類染色體(chromosome)蒙受捐害。換言之，危害人體基本遺傳物質。

幻藥和 L.S.D.

幻藥(Hallucinogenic drugs)的應用在原始社會中只被認為是宗教儀式的附庸品。但在比較進步的社會裏，藥物的用途就不止這些了。例如防止和治療症病。早於 1888年，一個先驅的精神藥物學家——Lewis Lewin，便已呼響其他科學學者注意和研究 peyote 和其他的精神性作用藥物。

幻藥屬於一組重要的化合物——精神性作用藥物。但由於它們有其特別的影響和効應，它們自成一家而成為一個分組(Sub-group)。和其他精神性作用藥物比較，幻藥使用者對自處的環境，所見物品，物品位置和時間變更都起了極端和持久的變異。甚至令人神智喪失。有時更會令人失去其正常人格表現。就算用者仍然清醒，他所歷練的是一個夢般的世界。但這個夢境世界在他而言就是真實的，比其習處的自然世界還真實，而物品和色彩更變得強烈和燦爛而失去其特徵好像隨時會脫離其位置而起。

L.S.D.的合成經過

差不多所有的精神性作用藥物都由植物提取而來。它們有相似的化學結構。其他的是由合成而得來。而L.S.D.就是其中最富効力的一種。此藥在1938年初次由一位化學家——Albert Hoffman 和 Arthur Stoll合成。他們對抽取一種菌類(Claviceps purpurea)中的植物鹼(alkaloid)衍生物(derativies)發生興趣。後者相似一類叫麥角(ergot)的物品。因為Ergot能使血管收縮(vaso constriction)，故能應用於生產時候，使產生強烈引縮和促進正常肌肉狀態(muscle tone)，當Hoffman做完第二十五次實驗後，d-lysergic acid diethylamide就被合成。初時，由於L.S.D.在結構上和一種普遍應用於解痙攣的藥物——coramine(nicotinic acid dimethylamide)有很多的相同，故亦被寄於對治療痙攣有効力。但實驗使人失望，L.S.D.對子宮肌肉之協調(tone)只有輕微影響。雖然預期的影響見不到，但在動物的試驗中，却發現了意外的反應。接受了這種藥物之後的一些動物變得極度興奮，而其餘的則進入一個癡呆狀態(catatonic state)。

L.S.D. 第一次被服用

合成後的第五年，亦即1943年的四月十六日，正在對L.S.D.作其他探討時，Hoffman忽然被一種奇異而不尋常的感覺所騷擾。他在他的實驗筆記上寫了以下一段經歷：在上一個星期五，即是四月十六日，在下午時，一種不能不休息的奇異感覺和些微的暈眩逼使我停止我的實驗工作，回家休息。回到家中之後，我卧着。我沉入了一個並不是不悅人的譫妄中(delerium)。它的特徵是令人極度幻想。雖然我的眼皮蓋着，但我卻感覺到眼前如日光般燦爛光輝，真不可信。當我恍惚地躺着，我腦海中升起了一個個無止際的夢境，清楚的見到無數怪誕的影像。那些燦爛的色彩，更是瞬時變化，像萬花筒中，五光十色。之後，這些現象漸漸消失。

Hoffman估計L.S.D.和這些特別的感覺有一些關連。由於他沒有服食過這藥，一定是由於空氣感染而使他吸入極少分量的藥物。為着進一步觀察此藥所起的反應，Hoffman故意服用0.25mg.的L.S.D.和其他ergot derivitates藥物進食數量比較，0.25mg.這個量是少的。今日L.S.D.通常的服用量是300-600mg.

這樣L.S.D.便第一次被人所服用。所起的感覺和寫在Hoffman筆記上的一樣。經過這次以後，毫無疑問L.S.D.是能對人體精神和物理兩方面產生極不尋常的影響。

L.S.D.化合結構

從化學方面看，L.S.D.是一種胺植物鹼相似一種ergot的成份麦角新碱(ergonovine)，亦能促進子宮引縮。和其他的胺植物鹼，例如ergot的另一成份麦角胺(ergotamine)比較，則L.S.D.就只能引起輕微的血

管引縮(vaso contriction)。L.S.D.所有顯著的影響都是針對中樞神經系統。自主神經和副交感神經系統(automatic and parasympathetia nervous system)亦有反應，但他們作用都是centrally mediated。例如：pupillary dilation瞳孔擴大在服用後是非常尋常的，但直接施於眼睛則得不到這種結果。影響中樞神經的藥很少會對其他系統器官有直接性的同一影響。除了人以外，L.S.D.對其他動物的影響亦被探討和研究。大致上，動物對此藥的抗力較大，而老鼠和rats更能抗拒多量的L.S.D.而不起反應。

1967年三月，在作者的實驗室裏，作者發現微量的L.S.D.能損害人體白血球細胞(leucocytes)的染色體。染色體是細胞核內由DNA(Deoxyribose nucleix acid)和核蛋白(nucleo protein)所構成的一種細胞基體(organelle)。它們帶有代與代之間的遺傳訊息(genetic message)。這些訊息調節和管制生命細胞內的代謝(metabolism)。至於這些節制的過程則非常復雜和精細。生物正常的發育有賴於這些過程的完整和健全。染色體的受損就足以分裂和破壞這些細微的過程而做成種種不同的畸形現象(abnormalities)。在人類臨床細胞遺傳學來說，有足夠的事例証明這種異常(aberations)有自發性的，大約百分之一的兒童會有這症狀。除了這些自發性畸形外，幾種外界的導原，例如：輻射(radiation)、病毒(viruses)和化學藥品(chemicals)亦能做成同樣變化。

L.S.D.對人體染色體的影響是從體內(in vivo)和體外(in vitro)兩方面觀察而得的。體內的研究包括培養六個健康正常的人的白血球。他們都從未服用過L.S.D.，亦未在近期展露(expose)於輻射線，或被病毒所感染。染色體就從這些培養在37°C.的全血中白血

球細胞內取得。在七十二小時的培養中，不同濃度，由 10 - 0.001 μg/ml. 的 L.S.D. 溶液就被注入這些培養物中。體內的一面是研究有服用 L.S.D. 紀錄的人的白血球。這包括十八個同樣下藥和十四個不下藥的對照(control)培養用作比較。這些體內培養和體外一組都在同一情況和同一地方被培養。然後用同一方法去記錄它們染色體的畸形。

L.S.D. 對單性細胞分裂(mitosis 亦作體細胞分裂)有顯著的抑制是體內實驗中的一項最早發現。在對照實驗(control experiments)中，體細胞分裂速度(rate)是 6.2%。而任何濃度的 L.S.D. 溶度皆能對分裂產生顯著的抑壓現象。而抑壓力隨 L.S.D. 逗留的時間加長而加大。在對照實驗的染色體畸形比率是 3.9%，而在其他製治過的培養中，畸形現象少的亦有近兩倍這個數字，即是 7.7%，而多竟達 17.5%，超過四倍對照的數字。這樣就能清楚明顯的指出 L.S.D. 能導致染色體畸形。

染色組體間和其之間切斷[L.S.D. 主要做成染色體的切斷(breaks)]的分佈是不規則的。如果假設 L.S.D. 均勻作用於單位長度的染色體，越長的染色體就有越多的切斷，反之，則越少。但事實上切斷的分佈並不如以上的假設這般均勻地分佈。而事實上，動原體(Centromere)的位置比其他地域易於受損害而致切斷。

體內實驗更証實進一步以上的發現的正確性。同年紀和性別的對照組的切斷比率平均為 3.8%，由 2 - 5.5% 不等。但這十八個有服食 L.S.D. 的用者的細胞染色體的切斷數卻非常驚人。多者在 25% 以上，而少的亦有 5%。切斷的頻度(frequency)與下藥次數，每次下藥數量、每次下藥相隔的時間和提取血液的時間都沒有明顯的關連。由於很多服用 L.S.D. 的人亦是其他某些藥物的用者，故另一項實驗是將其他一些藥物與

L.S.D. 同時試驗。結果指出這些藥物的種類和數目和染色體切斷沒有明顯的關係。實驗中可以見到切斷頻度的增加並不能歸咎於何種藥物的加入。

對孩童的研究

研究一些其母親曾在懷孕時服用 L.S.D. 的兒童時，事實指出此藥能穿過胎盤(placenta)的阻隔而進入胎兒的血液循環(blood circulation)中結果引致胎兒白血球的受損。十二個這般展露於此藥的兒童，和對照(control)組作比較時，八個比較易得到染色體切斷。雖然展露的次數不等，但損害至少作兩倍的增加。

美國許多實驗室亦都証實 L.S.D. 能引致染色體畸形。直到目前為止，大約有220個用者已被發現有染色體畸形的現象。而其中大約 75-85% 的情況更為不利。他們染色體切斷頻度正有增加。柯利昂大學的 S. Irwin 和 J. Egozcu 醫生曾經檢查多過六十個的用者和五個在胎中受染的兒童。其中三個兒童有染色體切斷數增加的表現。但並不是所有被檢查的用者都有染色體切斷的發現。需知，正如其他藥一樣，人與人之間所起的反應是不一樣的。

染色體損毀所做成的危機

染色體切斷可能做成的重要危機一定要從幾方面觀看。首先，對這個展露於這種破壞媒介的體個，染色體異常究竟做成了甚麼顯明或潛伏性危害。對人而言，這方面的資料還不能肯定作出甚麼結果。但需知在某些其他媒介影響之下，例如幅射(radiation)，亦能導致同樣的染色體切斷和互換。從這些有異曲同工之資料，我們可以討論一下。三種遺傳的疾病：Bloom's syndrome, Fanconi's anaemia 和 ataxia

telangiectasia亦能導致同樣的染色體異常。這些病的患者都極傾向患上白血球過多症(leukaemia)和其他腫瘤 neoplasms，例如腫瘤 tumours 和相似的不平常細胞分裂。除此之外，其他能引致相似的異常的媒介，例如輻射之於人及其他種類的生物，和病毒之於實驗室中的動物。這些媒介叫做致癌素(Carcinogens)。雖然染色體毀損與這些 neoplasia 的產生是否息息相關仍不為人所知。但無論如何，癌症和這些染色體畸形是有關連的。基於以上的討論，可以總結講：展露於一些像 L.S.D. 的媒介，最明顯的潛伏性危害就是增加用者對患上白血球過多症和其他一類的癌症的機會。

除此以外亦有一些比較不明顯的危害。在 L.S.D. 能透過胎盤這事來看，最有可能的結果就是先天性缺陷(Congenital disease)。雖然這樣的結論並沒有從人類得到，但在其他實驗已以觀察到，由於展露於 L.S.D. 所做成的先天性畸形和流產(abortions)。早期懷孕的老鼠、腮鼠和田鼠(hamsters)接受了注射之後，其嬰孩呈現範圍極廣濶的先天性缺陷。包括生長的全完阻竭和嚴重的中樞神經系統畸形症狀。早期懷孕服用此藥所得的結果非常顯著和嚴重。但後期注射所得的影響的程度則有限。最近愛我華大學(University of Zowa)的 Dr. Hans Zellweger 和他同伴報告了一宗嬰孩四肢畸形現象。他們認為這是由於這嬰孩的雙親在懷孕期間都曾服用過 L.S.D.。

這些媒介所做成最大的危害或者會做成生殖細胞配子(gamete)染色體的傷毀。由於 L.S.D. 存在於用者的血液中，亦當然能夠到達製做這些生殖細胞的地域。要探討這些細胞染色體的受損過程，就必須直接觀察。

這種研究施於人類當有其困難。但無論如何，從動物着手或是可以解答此一問題。University of Copenhagen 的 Drs. N.E. Shakkeback, J. Philip, 和

O.J. Rafaelson 對雄性老鼠作研究後，証實一次的 L.S.D. 注射就足已做成染色體的損傷。如果白血球染色體遭受的損害同樣出現於這些 spermatogenial cells 的話，L.S.D. 的影響是有遺傳性，事就毫無疑問了。

舉止行為的持久和極度失常是一項公認的 L.S.D. 作用力。在最後服用 L.S.D. 後的一年半內（至少數），自發週期性的神經失常是可以見到的。我們研究一組病者後可以指出行為，藥量，下藥頻率，最後下藥和發現染色體損傷等之間是沒有直接關連的。直到目前，我們亦見不到那些在胎兒時期受染的孩童有甚麼顯著的生心和舉止失常。

結　言舌

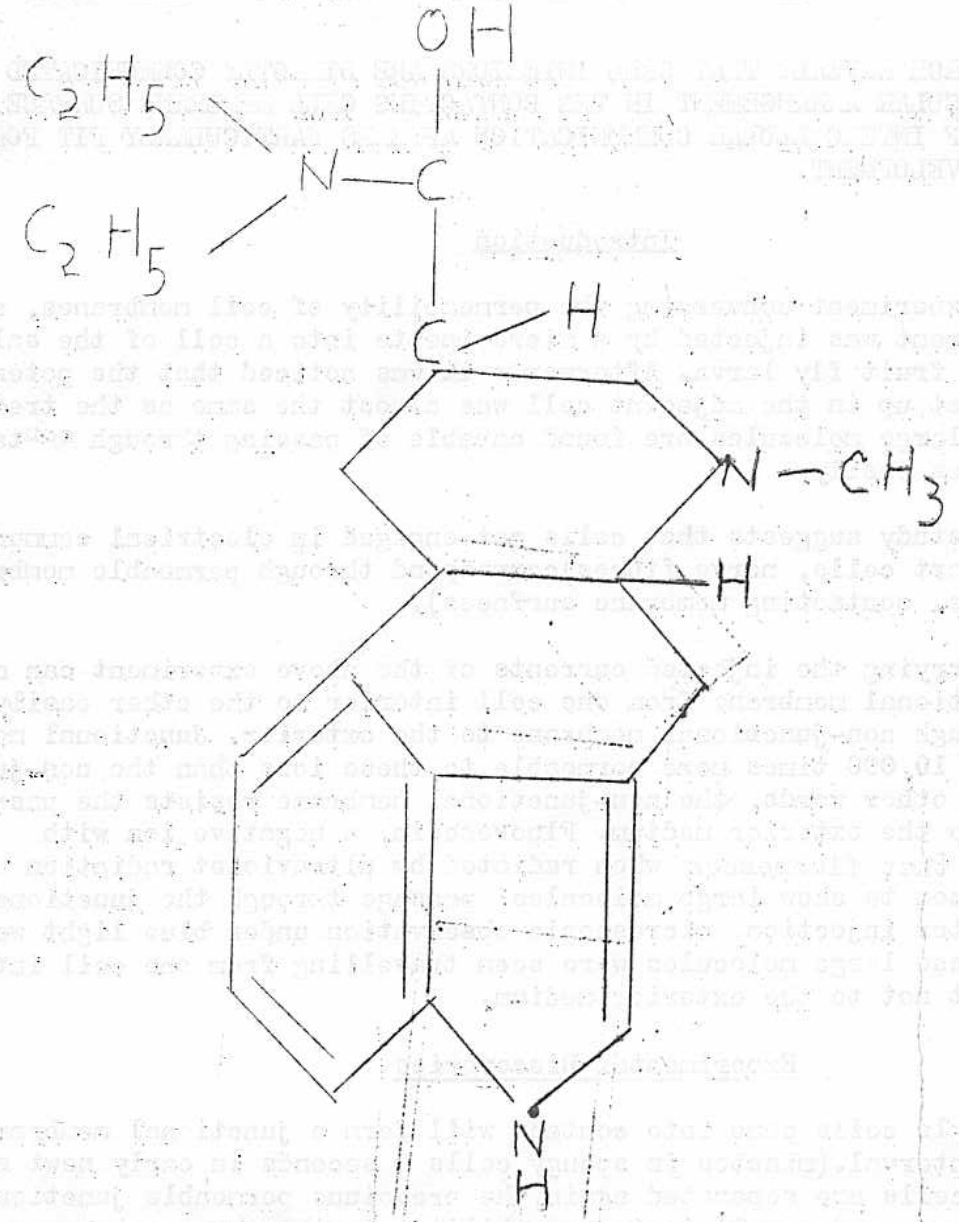
好久以前，精神病學者便已知道 L.S.D. 是一種極有力的 mind-expanding 化合物。但深入研究其生物化學，精神作用和藥理的工作到近年來才進行。依靠今日我們有限的資料和知識，是仍然不敢作出一個肯定的結論。雖然時下對染色體切斷和導致胎兒變化的研究只是開始，但這應該足夠刺激更多和更有規模的研究。這是迫切的需要。對多量人口應該作調查以便找出　真正由 L.S.D 做成的胎死（foetal deaths）和先天性畸形（congenital abnormalities）的數字。

亦用以證清 L.S.D. 的影響是否有遺傳作用，從一代延至一代。幾個實驗室已進行計劃這種探討。但由於現時 L.S.D. 正被廣汎地被服用，全人類的遺傳性和心理性的總損傷的數目是不能在近期內找出。

完

M.M.C/L.C.K./L.S.W.

Pg. 40



Molecular Structure of L.S.D.

JUNCTIONAL MEMBRANE

RECENT RESEARCH REVEALS THAT CELL INTERIORS ARE DIRECTLY COMMUNICATED BY SPECIAL MOLECULAR ARRANGEMENT IN THE CONTACTING CELL MEMBRANE SURFACE. THIS TYPE OF INTERCELLULAR COMMUNICATION APPEARS PARTICULARLY FIT FOR EMBRYONIC DEVELOPMENT.

Introduction

In an experiment concerning the permeability of cell membranes, an electric current was injected by a micropipette into a cell of the salivary gland of the fruit fly larva. Afterwards it was noticed that the potential difference set up in the adjacent cell was almost the same as the treated cell. Later large molecules are found capable of passing through contacting cell membranes easily.

Years' study suggests that cells not engaged in electrical communication (i.e. not heart cells, nerve fibres)correspond through permeable membrane junctions(i.e. contacting membrane surfaces).

Ions carrying the injected currents of the above experiment can move through junctional membrane from one cell interior to the other easily, but not through non-junctional membrane to the exterior. Junctional membranes are 1,000 to 10,000 times more permeable to these ions than the non-junctional membrane. In other words, the non-junctional membrane resists the passing of these ions to the exterior medium. Fluorescein, a negative ion with weight 330, that fluoresces when radiated by ultraviolet radiation or blue light, was used to show large molecules' passage through the junctional membrane. After injection, microscopic observation under blue light was followed. These large molecules were seen travelling from one cell interior to other, but not to the exterior medium.

Experimental Discoveries

Two single cells come into contact will form a junctional membrane short interval.(minutes in spongy cells & seconds in early newt embryo). When such 2 cells are separated again, the prevoiuus permeable junctional membranes change and result in impermeability, in the process known as 'sealing'. If the cells are allowed to contact again, a new junctional membrane is formed at other site. This concludes that A LARGE PART OF THE TOTAL CELL SURFACE MEMBRANE MUST HAVE THE POWER OF FORMING COMMUNICATIVE JUNCTION.

When the calcium ion concentration of the cell interior is raised by some means, the permeability of the junctional membrane decreases obviously. When the calcium content comes to level corresponding to the exterior(e.g. by injecting calcium ion into the cytoplasm of the cell), the junctional membrane becomes impermeable as if the 2 cells are separated. In normal cells, the cytoplasm's calcium content is always many times lower than the exterior.

Permeability is also related to 'Isolation'. Good isolation is maintained if 3 factors are suitably applied. A glyoprotein-containing substance is used at cell surfaces and calcium and magnesium content are supplied to the medium. When the latter 2 factors are above certain values, good insulation is kept and the junctional membrane remains permeable, if these 2 factors are taken away from the medium, the permeability decreases and the junctional membrane may result in sealing.

Hypothesis

With the experimental information, a hypothesis is put forward. It is assumed that the permeability increases with the decrease of calcium content and on the other hand decrease with an increase of calcium. On the surface membrane, there may exist many 'binding sites' that are occupied by calcium in impermeable state. Good communication is achieved when these calcium is removed. When the junctional membrane is formed, i.e. after the existence of a 'junctional seal' around a certain portion of the contacting membrane, the latter becomes incorporated into the intracellular components; whereas they face a calcium ion contentration of several order of magnitude lower than they did to the exterior. In this way, calcium detaches from the 'binding sites on the junctional membrane. Communication is brought about as a result of satisfied permeability The mechanism of this hypothesis requires no special properties of the membranes. The formation of the junctional seals and the maintance of low cellular cytoplasmic calcium concentration are the only requirements. Generally the required amount of calcuim is stored up in mitochromia and other organelles, Thus a low cytoplasm calcium concentration is ensured.

Further Experiment

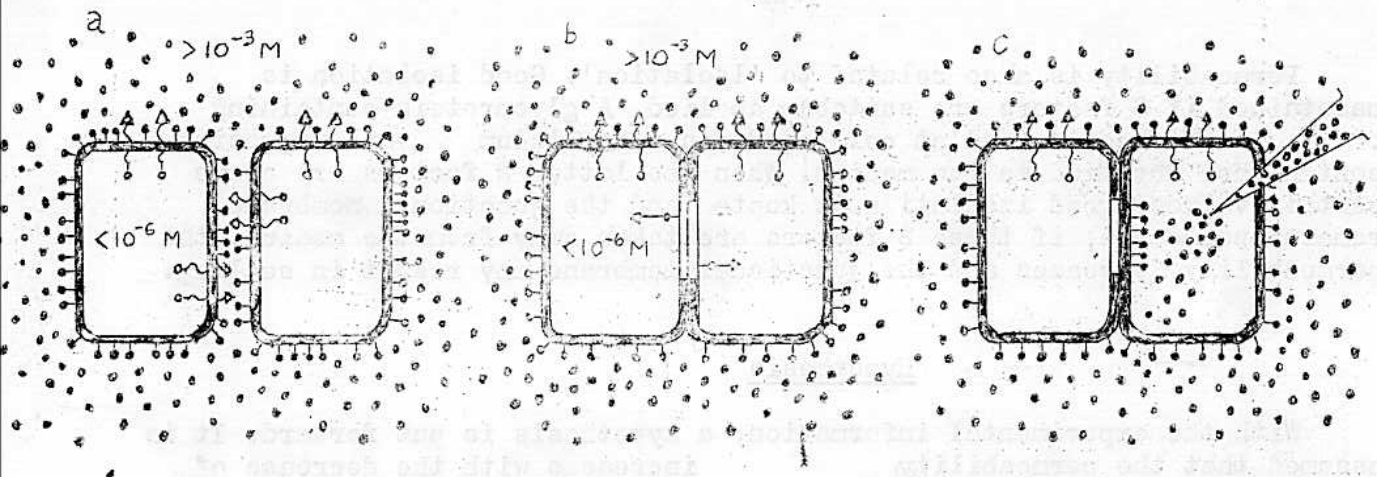
High permeability is ensured by low calcium concentration in the cell Energy-utilizing processes are employed by many types of cells to transport unrequired calcium content to the exterior. The energy for this purpose must be supplied by cellular metabolism and experiments illustruting this assumption prove its reality. In one test, the scientists reduce the rate of formation of ATP of the treated tissues by direct cooling or adding calculated amount of poisons such as cyanide, dinitrophenol(DNF) or oligomycin. This results in a decrease of junctional impermeabitly.

In another test, the metabolism inhibitor and ATP are mixed and injected into the tissue. This produce no obvious reduction in permeability in spite of the presence of the poison.

(See Cellular Respiration)

Effect of Lithium

Uncoupling(the ceasing of intercellular communication) occurs when the sodium part of the extracellular component is replaced by Lithium. This substitute and some other chemicals can increase the cytoplasmic calcium in many tissues. But it is clear that sodium has no effect as no effect is observed when sodium is substituted by choline. It is believed that these substances accelerate the inflow of calcium and lower sown the removal of cellular calcium.

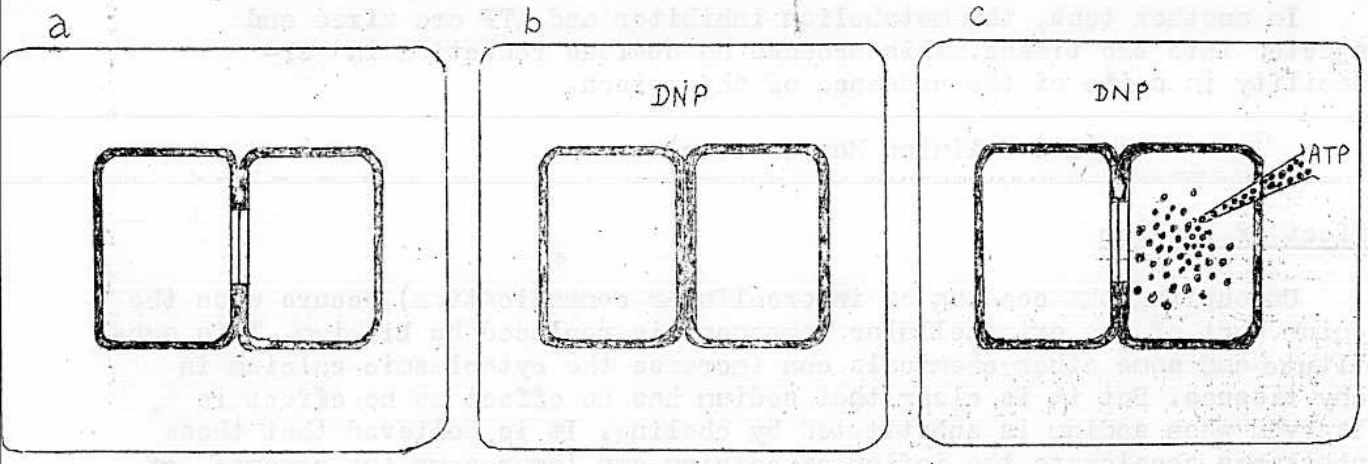


Figures illustrating the CONVERSION HYPOTHESIS (Above)

It is assumed that the membrane permeability is determined by the Ca^{2+} Conc. (dots -Ca^{++}) in the medium on both sides of the surface membrane. Membrane exposing to normal extracellular medium will have its 'binding sits' filledwith calcium while the other side, which faces th e interior that is low in calcium conc.,will have its 'binding sites' sparsely occupied by calcium. When two cells come into contact (c), , the formation of junctional seal furnishes a region(white) of that becomes permeable because of facing a low calcium conc. (c) illustrates the reverse of this process by injecting calcium into one cell interior.

Figures illustrating the the role of metabolic energy. (Below)

Metabolic energy is required to expell the calcium away from the cell interior. (a) DNP, dinitrophenol, is injected into the cell medium and 15 minutes later junctional permeability falls markedly. This is because the poison has broken the manufacture of ATP. The 2 cells no longer communicate by junctional membrane. When one cell (c) is injected with ATP artificially, calcium expelling is continued and junctional communication is observed again.



Information conveyed through junctional membranes

Many large molecules can pass through a junctional membrane. This includes the metabolites and other many activity-regulating agents, e.g. the gene-activity regulating signal molecules. In this way, it is attractive to relate the transportation of substances that govern growth and differentiation of growing tissues with these membranes. Signal molecules may pass from cytoplasm to cytoplasm through this close-range communicative system with little loss . The possible function is that their concentration may furnish the developing cell the information of growth and differe - niation.

However this assumption seems difficult to be proved as no signal molecule can be identified in the developing tissues. Work on the earliest cells of a starfish embryo in 1964 was disappointing as junctional communication ceased after the cleavage was completed. This is contrast to the assumption as the scientists expected junctional communication to be functional in embryonic development. Nevertheless research was carried on and gave to new light. Such embryos, when older, did communicate by junctional membranes, The degree is very satisfied. Embryos from various groups of animals were tested and exciting and encouraging results were obtained. Growing and differentiating tissues did communicate by junctional membranes.

If molecules regulating normal growth and differentiation do require the junctional passage in its conveyance, then the aberrant growth of cancerous cells may be a result of the latter's poor junctional communication. Experiments with mammalian liver back this assumption. No junctional communication is recorded between cancerous cells nor between the cancerous cells and the normal cells. But good communication is observed in normal liver cells. The cancerous cells, though in contact, appear as completely separated individuals. Further investigation by E.M.(Electronic microscope) shows that these abnormal cells lack some structures that link normal cells.

Poor junctional communication is observed in cancerous growth, but not in other growth similar to them. In the regeneration of an adult rat liver, the dividing rate is astonishingly rapid(more rapid than the fastest tumour). However the growth slows down and finally cease when the normal size of the damaged liver is restored. Good junctional communication is present in such regenerating tissues.

Conclusion

Experiments show that contacting cells do form permeable junctions under the above stated circumstances. This furnishes a communicative passage for the growth-regulating substances in embryonic development and differentiation. Though cancerous growth may be explained by poor junctional communication, The lattwe is not the only cause of this type of aberrant growth. The unability to receive or manufacture some growth controlling substances are causes also. Some cancerous cells has junctional communication that allows certain molecules to pass. However this research of intercellular communication gives new light to the study of tissue growth and development.

** This passage was extracted from Werner R. Loe Wenstein's report of "INTERCELLULAR COMMONICATION" which was published in Scientific American May 1970. This passage is arranged and simplified by LO CHI KEUNG.

我們所寫的稿件，加在發刊詞中說明，不敢說是我們自己所創做的。我們只是把它們從多位原著中節錄出來，已引起讀者作進一步探索的興趣。

本文涉及的思想和人物眾多，在短短數千字中當然不能盡其精要。然而希望各位本着「知而求其解」的精神，作進一步的探索和研討。那麼，我們的原意才有了報答和安慰。我想，這亦是編者和各原作者的深意。

文中中英並用，似於雜亂。但內心一想，這亦是編者的深意，雖知「我、你、他」確不只三人已矣。

（志恩序"我你他語錄"）

× × × × × × × × × × × × × × × × × × × ×

俗語說：「青竹蛇見口，黃蜂尾上針」。且談一談前者在香港的概況。

青竹蛇(Bamboo Snake)，學名 Trimeresurus albolabris Gray，是蝮蛇科中的毒蛇，亦是香港唯一的蝮蛇科毒蛇。青竹蛇的毒液的毒性，對人而言，並不算厲害。但它卻有很長的青牙和牙中有很大的中空，能儲備大量毒液。給它咬上倒是極大的麻煩。

如其名一樣，青竹蛇是綠色的。但只是背面。腹部是青黃或微白色的。而尾部的背面則有一部分是褐色的。頭部成極明顯的三角形，與頸部分別致大而一望而知。

青竹蛇有極好的保護色，通常躲藏在綠色的矮叢中和長草叢中。極難為人發現。青竹蛇雖然細小，最長不過三尺，但行動卻不及其他蛇的迅速。當有其他動物走近時，它多是靜寂不動，而當前者走到兩尺左右時才出擊。咬人一口時，才乘機逃走。故所以，剪草的工人時常都被這種蛇所偷襲而受傷。這蛇在赤柱一帶最多。而足跡則遍佈港九和離島。（志恩）

lck/lsw