Complete text

VI LSD No. 251 (§102a)
ACTH (§407f)

M 500 properties
F 17 urine analysis
M 360 schizophrenia

HOAGLAND, H. (Shrewsbury, Mass.)

Some biochemical factors common to schizophrenia and to LSD produced in normal persons. (psychoses)

XXth International Physiological Congress, Brussels July 30th - August 4th 1956. Abstracts of Communications, p. 427.

Biochemical evidence is presented for an hypothesis that schizophrenic patients, may produce an endogenous metabolite which acts after the manner of lysergic acid diethylamide (LSD) when administered to normal persons.

Studies of schizophrenic male patients and normal male controls under similar non-stressful conditions show that the patients excrete only approximately half as much urinary phosphates as do controls of comparable age. Following experimental stresses or the injection of ACTH or of adrenocortical extract the patients show marked increases in phosphate excretion which are not seen in the normals.

A study of normal male subjects given LSD sufficient to produce psychotic symptoms shows that during the time of action of LSD the subjects display a reduced phosphate excretion. But when ACTH was injected during the period of action of the LSD marked enhancement of phosphate excretion occurred. Thus the behavior of the urinary phosphates both at rest and under the impact of corticoids in the LSD-treated normal subject is like that seen in schizophrenic patients without LSD. We have also found that LSD suppresses phosphate excretion in guinea pigs and that ACTH injected after LSD administration produces marked phosphate excretion. Rats do not display these responses to LSD and ACTH. We have suggested (HOAGLAND, H., RINKEL, M. and R.W. HYDE, *Arch. Neur. & Psychiat.* 1955, *73*, 100) that these data together with considerations from the literature indicate that LSD facilitates the formation of phosphorylated substances and that adrenocortical hormones tend to inhibit this excessive phosphorylation.

These results are consistent with the view that schizophrenic patients produce a metabolic substance that acts like administered LSD to produce both psychotic behavior and excessive phosphorylation that is reversible by adrenocorticoids. Data are presented showing that schizopre-

nics are relatively unresponsive to LSD compared to normals and to other psychotics. If enzyme receptors are already occupied by an endogenously produced LSD-like antimetabolite, refractoriness to LSD is to be expected. Certain psychosomimetic metabolites of epinephrine and of 5-hydroxytryptamine are under investigation.

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(Quelques facteurs biochimiques communs à la schizophrénie et aux psychoses provoquées par LSD chez des sujets normaux.)

Chez le sujet sain, des doses de LSD déclenchant une réaction psychosique diminuent l'excrétion de phosphates pendant la durée de la "psychose". L'ACTH, injectée pendant l'effet du LSD, accentue nettement l'excrétion de phosphates. Le comportement des phosphates chez des sujets normaux sous l'influence du LSD, sans ou avec administration d'hormone cortico-surrénalienne, ressemble donc à celui observé chez des malades schizophréniques sans LSD. Chez le cobaye, le LSD inhibe l'excrétion de phosphate; l'injection d'ACTH après LSD l'accentue nettement. Le rat ne montre pas cette réaction au LSD et à l'ACTH. [V. aussi HOAGLAND, LSD No 69, Rapport 39/20.] La réaction au LSD de malades schizophréniques est relativement faible comparée à celle de sujets sains et de malades atteints d'autres psychoses.

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