cæsium hexahalogen complexes of the platinum metals are the least soluble and hence most likely to yield a stable quadrivalent rhodium compound.

When an ice-cold solution of sodium hexachlororhodate (III), saturated with chlorine, was treated with solid cæsium chloride, a fine, pink suspension of cæsium hexachlor-rhodate (III), (Cs_3RhCl_6), was obtained. On adding ceric nitrate in dilute nitric acid this was oxidized instantly to green, insoluble cæsium hexachlor-rhodate (IV), (Cs_2RhCl_6). The identity of the substance was established by analysis. X-ray powder photographs showed the substance to be face-centred cubic and isomorphous with ammonium hexachlor-platinate (IV). The compound decomposed in contact with water, liberating chlorine, and was readily reduced to trivalent rhodium, the colour changing to pink.

Further details will be published in the *Journal of the Royal Society of New South Wales*.

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¹ Alvarez, *C.R.*, **101**, 851 (1885). Wohler and Ewald, *Z. anorg. allg. chemie*, **201**, 151 (1931).

² Grube and Gu, *Z. Electrochem.*, **43**, 397 (1937).

Central Excitant Action of Diisopropylfluorophosphonate

PREVIOUS work from this laboratory has shown that the anticholinesterase group of drugs acts on the central nervous system in cats and man; eserine and the related tertiary compounds are central excitants, prostigmine and the related quaternary compounds are central depressants. Diisopropylfluorophosphonate has an entirely different type of chemical constitution from the hitherto described anti-cholinesterases, and *in vitro* is a more potent inhibitor of cholinesterase than eserine. It was thought of interest to examine its action on the central nervous system; it proved to be a central excitant like eserine sulphate, but larger doses were required to produce similar effects.

Diisopropylfluorophosphonate produces its central excitant effects on intravenous or intrathecal injection in cats under chloralose anaesthesia, in decerebrate animals and in the decerebrate or chloralosed animal made spinal. The reflexes examined were the knee jerk, the crossed extensor reflex, the jar reflex and the flexor reflex. The knee jerk was enhanced, but the most striking effect was on the after-discharge, which became larger and more prolonged; the crossed extensor reflex, which is often absent or is feeble and poorly sustained in the chloralosed animal or in the spinal preparation, made its appearance under the influence of diisopropylfluorophosphonate and became more powerful and better sustained; the jar reflex which is normally absent frequently made its appearance; and the flexor reflex was potentiated though much less regularly than the other reflexes mentioned.

In larger doses diisopropylfluorophosphonate produced convulsions, which were often violent in character and long sustained; in decerebrate animals marked increases or phasic fluctuations in tone occurred. The reflex changes described occurred independently of alterations in blood pressure or respiration or of peripheral potentiation of the nerve-

muscle response; they are independent of the presence of the anaesthetic, or of the cerebrum. The reflex changes are modified by removal of the brain-stem, but the essential features are still shown by the spinal animal. We conclude that diisopropylfluorophosphonate acts on the central nervous system, facilitating excitatory transmission in the spinal cord and brain stem.

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X-Ray Induced Mutations in Dried Bacteria

AGENTS such as ionizing radiations, mustard gas, etc., effective in increasing mutation-rates in higher organisms, have been shown¹ to act likewise in micro-organisms, including several species of bacteria. We can now add a strain of *Bacterium aerogenes* (isolated by one of us—C.H.—from dried milk) to the list of bacteria in which mutations have been induced by X-ray treatment: doses of 35,000 to 50,000 r., which leave 10^{-3} to 10^{-4} survivors, yield 'biochemical' mutants at rates of up to 1 per cent.

A relevant feature of our results lies in the fact that these mutants were obtained by treating bacteria preserved in *the dry state*. This shows that active growth and division at the time of treatment is not an essential condition for the induction of mutations in bacteria. A new similarity has therefore come to light between mutation in bacteria and in higher organisms, since in the latter non-dividing and relatively quiescent cells, such as spores, spermatozoa, etc., are known to mutate readily under the action of mutagenic agents (see, however, Stone *et al.*² for a possible delayed mode of action of irradiation).

The mutants have been isolated by means of Lederberg and Tatum's³ technique; they differ from the parent strain in having one or other new specific nutritional requirement, presumably because a step in the synthesis of an essential metabolite fails to be performed⁴. The identification of these specific requirements is now in progress; we have so far characterized mutants requiring aneurin, cystine, methionine, adenine, uracil, nicotinic acid, threonine, glycine, arginine, asparagine and histidine.

Another relevant feature in our results is the high proportion of mutants which are of the 'adaptable' type. That is, while the original strain grows almost at once on a medium composed of glucose and inorganic salts, a mutant, 'adaptable' or otherwise, can do the same only if a particular nutritive is added to this medium. Without this addition a non-adaptable mutant will never grow, but an 'adaptable' mutant will grow after a 'lag' of, say, twenty-four hours or more. Once it has started growing normally it will continue to do so on successive transfers to the same deficient medium. This 'adaptation', however, is lost on passage through a medium supplying the nutritive.

Clearly these 'adaptable' mutants are of great interest for genetics and biochemistry. They may presumably owe their adaptability to either: (1) an inheritable change causing the production of a changed form of an enzyme; or (2) an inheritable change entailing failure to produce an enzyme and opening the way for an alternative enzyme system to be called into operation; or (3) the inactivation of a gene which, in the normal strain, controls directly