THE THOMAS BAKER, ALICE BAKER, AND ELEANOR SHAW MEDICAL RESEARCH INSTITUTE

TWENTIETH ANNUAL REPORT

The Baker Institute derives its main financial support from the Thomas Baker (Kodak), Alice Baker, and Eleanor Shaw Benefactions. It is also dependent upon grants from The National Health and Medical Research Council, and donations from private sources. The latter are allocated to an Endowment Fund.

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DR. M. C. DAVIS, M.D., B.S.

Historical

The Baker Medical Research Institute originated as a direct outgrowth from the laboratory service of the Alfred Hospital. At November, 1922, this Department was under the control of Mr. Alfred J. Trinca, F.R.C.S. At this time Dr. Mackeddie, a member of the Honorary Medical Staff, outlined a scheme for the establishment of a biochemical laboratory. With this end in view the present Director was sent to England to study under Professor Hugh MacLean, at St. Thomas's Hospital, London. He returned in January, 1924, and was appointed Biochemist to the Hospital.

In 1925, the Edward Wilson Trust provided £3000 for the erection of a biochemical laboratory. Subsequently the late Mr. Thomas Baker, Chairman of Directors of Kodak (A/asia) Pty. Ltd., his wife, and Miss Eleanor Shaw took over the cost of the building and accepted liability for the maintenance of the Institute for five years at a cost of £3500 per annum. This was later supplemented by an additional £1500 per annum for five years. In February of 1927, the late Dr. W. J. Penfold was appointed Director of the Institute. Shortly after Dr. Penfold's appointment, Dr. Corkill was sent to England to study research problems under Sir Henry Dale, at Hampstead. Mr. Baker died in December, 1928, and in his will provided generously for the continued maintenance and support of the Institute as laid down by him in a Deed of Trust.

The policy of the Trust was to provide an efficient routine hospital laboratory service, and to carry out medical research. Accordingly, the biochemical and bacteriological facilities were developed, a technical staff trained, a library collected, and teaching of students in the elements of laboratory medicine commenced. In addition, a research team was built up. The clinicians of the Hospital have been encouraged to come to the Institute and seek the help of the various members of the staff in the elucidation of their problems.

On the 18th May, 1938, the Director, Dr. W. J. Penfold, resigned on the grounds of ill-health, and Dr. Corkill was appointed as his successor. In Dr. Penfold's time the Hospital Pathology Department and the Institute were under his unified administration, but this ceased when the Hospital erected a new Pathological Department, under the direction of Dr. R. H. Willis. With this new departure, the routine bacteriological service, formerly under the administration of Dr. Penfold, was transferred to the Hospital.

At the present time the Baker Institute provides a routine biochemical service for the Hospital, and also prepares bacteriological media and supplies animals for the use of the Hospital laboratory services. The research work of the Baker Institute is now devoted chiefly to physiological and biochemical problems, and apart from fundamental research on these subjects, has contributed considerably in the application of research to clinical problems.

EDGAR ROUSE,
Chairman of Trustees.

The Director's Twentieth Annual Report

TO THE TRUSTEES

of the

THOMAS BAKER, ALICE BAKER, AND ELEANOR SHAW MEDICAL RESEARCH INSTITUTE

Gentlemen,—

During the year important discussions were held concerning the research policy of the Institute, and also the relationship of the Institute to the routine work of the Hospital. In respect of research work, you decided, after due consideration, that in view of the particular qualifications of the present staff, the interests of the Institute would be best served by concentrating on physiological and biochemical research. This has been done and under the appropriate sections will be found descriptions of the various research problems that have been investigated.

The most important work carried out during the year has been in connection with problems dealing with the mechanism of the clotting of blood and related blood diseases. In addition, problems related to carbohydrate metabolism with particular reference to diabetes have been investigated.

In previous reports I have referred to the difficulty of preventing the routine hospital work from making too great demands on the time of the research workers. Latterly the position has become still more acute, and recently a joint meeting of representatives of the Baker Institute, Board of Management of the Hospital, and the Honorary Medical Staff, met to consider the question of routine work. The present and future requirements of the Hospital were discussed, and the latter agreed to provide extra staff and equipment to meet the increased demands. Whilst this is definitely a step in the right direction, I still feel, as intimated in my last report, that if the Hospital is to make the best use of the trained staff of the Institute, it will be necessary to establish a clinical research unit. The Director of such a unit would control all routine services, and also work in close liaison with the Baker Institute.

It is possible that in the future the Hospital will control all the routine laboratory services, but will have them housed in

close proximity to the Baker Institute, with the object of obtaining the closest co-operation between the two departments. In this connection there has been, during the year, a very close liaison between the laboratory work of the Institute and problems concerning treatment and diagnosis in the wards. The practical application of 3,3'ethylidene-bis (4-hydroxycoumarin), the blood anti-coagulant synthesised by Dr. Fantl, has been greatly extended. Many applications for its supply have been made by physicians outside the Hospital, but since its administration must be rigorously controlled by laboratory tests, the Institute has been hesitant to issue it for general use. Apart from 3,3'ethylidene-bis (4-hydroxycoumarin), new biochemical investigations have been introduced, and in general the Institute has done its best to carry out pure research and yet give clinical co-operation.

It is with regret that I record the death of our Honorary Solicitor, Mr. John Turnbull. During his association with the Institute he rendered willing and valuable service. Mr. Hubert Black, partner of the firm of Blake and Riggall, has kindly consented to act as Honorary Solicitor.

Prior to the war, various Honoraries spent some time in the Institute on experimental research. With the ending of the war, and the return of medical men from the Forces, it is hoped that clinical research will recommence. Already Mr. Trinca, Mr. Officer-Brown, and Mr. Devine are investigating various problems.

The new animal house is now fully occupied, and a marked improvement in breeding and general health of the stock can be observed.

The Institute is again indebted to The National Health and Medical Research Council for financial support for the study of diseases of blood and bone.

My thanks are also due to the Department of Biochemistry at the Melbourne University for materials necessary for the work of the Institute, and in this connection Messrs. W. S. Kimpton and Sons must also be thanked.

Finally, I should like to thank the Will Trustees (Thomas Baker (Kodak), Alice Baker and Eleanor Shaw) for their continued generous support, which has served as a great stimulus to the scientific personnel of the Institute.

The Library.

Our thanks are due to the following for gifts of literature during the year: Bayer Products Ltd.; Commonwealth Serum Laboratories; The Connaught Laboratories; Council for Scienti-

tic and Industrial Research; Eastman Kodak Commpany; Mr. Robert Fowler, F.R.C.S.; Graham Research Department, University of London; Imperial Chemical Industries of Australia and New Zealand Ltd.; Institute of Medical and Veterinary Science; The Editor, "Laboratory Journal of Australasia"; Lister Institute of Preventive Medicine; The Mayo Clinic; Eli Lilly Laboratories; Medical Research Council, London; Middlesex Hospital Medical School; Munitions Supply Laboratories; New York State Department of Health; Rockefeller Foundation; Royal College of Physicians, Edinburgh; South African Institute for Medical Research; State Serum Institute, Denmark; Mr. A. J. Trinca, F.R.C.S.

We are very grateful to the Melbourne University and other Libraries for lending many Journals throughout the year.

PHYSIOLOGICAL AND BIOCHEMICAL RESEARCH

Studies on Blood Coagulation.

The administration of the drug 3,3'ethylidene-bis-(4-hydroxycoumarin) (E.D.C.) has been continued in the treatment of post-operative thrombosis, and its use extended to cases of coronary thrombosis. Up to the present, sixty cases have been treated, and according to clinical reports, the results were satisfactory. The routine procedure is a three-day course of treatment with 0.5 gm. on two successive days and then 0.2 gm. on the third day, with patients whose initial prothrombin was 70-100 per cent. of the normal. A prothrombin estimation is carried out on the fourth day, and from then on the E.D.C. dosage is determined by the reactivity of the patient, and varies between 0.1 and 0.3 gm. daily. In most cases it was found sufficient to estimate prothrombin twice weekly.

Although no bleeding tendencies have been encountered as a result of over-dosage of E.D.C., it was felt necessary to have counter measures available. Transfusion of fresh blood will restore the prothrombin concentration at least temporarily, and administration of Vitamin K preparations in large doses has been found useful in the case of hypoprothrombinaemia induced by dicoumarol. Our own experiments were carried out with a water soluble sodium salt of 2-methyl 1,4 naphthohydroquinone monophosphate on rabbits. Treatment of the animals with 50 mgms. of this Vitamin K preparation* was of value in counteracting the effect of 25-30 mgms. of E.D.C. administered orally half an hour later.

In collaboration with Dr. L. Cox, the treatment of disseminated sclerosis with E.D.C. is being tested following a suggestion by Putnam et al. that blood anticoagulants be used in this disease (Arch. Neurol. and Psych., 57, 1, 1947).

Influence of Theobromine on the Coagulation Mechanism.

Link and co-workers (J. Biol. Chem., 156, 725, 1944) reported that methylated xanthines administered to experimental animals produced a hyperprothrombinaemia and they found that these compounds are able to counteract substances capable of producing hypoprothrombinaemia. From their results it would appear that the administration of theobromine and related compounds in the treatment of coronary disease should be discouraged because of the danger of producing intravascular thrombosis due to hyperprothrombinaemia. In view of the practical importance of these statements the problem was investigated, using the quantitative chemical estimation for prothrombin outlined in the

* The Vitamin K preparation was supplied by Nicholas Pty. Ltd.

previous annual report. Suitable cases were selected from Dr. M. C. Davis's Clinic at the Alfred Hospital, and a suspension of 0.5 gm. of theobromine was administered orally three times daily for ten days. Estimations of total protein, fibrinogen, and prothrombin by Quick's procedure, in whole and 12½% diluted plasma, as well as by the adsorption technique, were carried out. No significant influence was detected on prothrombin concentration, and some fluctuations in the fibrinogen concentration were observed. In order to find out whether theobromine has a protective action against induced hypoprothrombinaemia, rabbits were injected intraperitoneally with 60 mgms. of theobromine for three days. Two hours later 20 mgms. of E.D.C. were given orally. In this series the theobromine treated animals showed a more severe hypoprothrombinaemia, though the recovery period was the same as in the control series. However, animals which obtained theobromine in addition to E.D.C. showed a greater increase in fibrinogen concentration than the E.D.C. treated animals.

B.A.L.

The studies on the mechanism of blood coagulation have been continued by Dr. Fantl, Miss Nance and Mrs. Simon. The most interesting finding was that 2,3 Dimercaptopropanol (BAL) which was discovered by Peters, Stocken and Thompson ("Nature," 156, 616, 1945), as an antidote against Lewisite and metal poisoning, had a marked inhibitory influence on plasma coagulation. A detailed study indicated that it has a very pronounced effect on prothrombin, and possibly on thrombin, but no influence on the other components essential to blood coagulation could be detected. BAL is superior to other -SH compounds such as cysteine and glutathione, in its inhibitory action. It would seem that the inactivation of prothrombin by BAL is due to its ability to attack the active centre of the prothrombin molecule possibly by converting -S-S-groups to -SH radicals.

Further evidence that prothrombin is a single compound has been obtained by isolating prothrombin by a new technique, which consists of its adsorption on barium sulphate and fractional elution with phosphate buffer. This procedure allows the separation of prothrombin from the other plasma constituents in about one hour and preparations of great potency are obtained which are fully active when incubated with thromboplastin, calcium ions, and tested on purified fibrinogen.

Citric Acid and Calcium Metabolism.

The value of blood citric acid estimations in the diagnosis of parathyroid tumours was mentioned in the last report. In further investigations relating to calcium and citric acid meta-

bolism to bone diseases, studies have been extended to cases of chronic osteoarthritis. W. Grant Waugh (Brit. Med. J., 1, 873, 1945) found that the injection of lactic acid was beneficial and Warren Crowe (Lancet 1, 563, 1944), suggested the injection of 1 per cent. potassium acid phosphate into the affected joint. Although good clinical results were observed using such a solution, it was felt that repeated injection of potassium ions would lead to undesirable side-effects; Dr. Fantl suggested the use of a M/15 mixture of primary and secondary sodium acid phosphates having a pH 5.8, and Dr. Bean, working in Dr. Clark's Out-patients' Clinic, Alfred Hospital, has found that this solution gives satisfactory results. Estimations of calcium, inorganic phosphatase, alkaline phosphatase, and citric acid in blood serum have been carried out prior to and after a series of injections. Apart from an occasional rise in alkaline phosphatase activity shortly after the injection, no other changes were found. Whether this phenomenon can be explained as a stimulation of osteoblastic activity is now under investigation.

Publications.

P. FANTL AND MARGARET H. NANCE:

"Activation of Prothrombin." The Australian Journal of Science, Vol. IX, 21st December, 1946, p. 117.

P. FANTL AND MARGARET H. NANCE:

"Acceleration of thrombin formation by a plasma component." "Nature," Vol. 158, 16th November, 1946, p. 708.

P. FANTL AND MARGARET H. NANCE:

"Influence of 2,3-Dimercaptopropanol (BAL) on Blood Coagulation." "Nature," Vol. 159, 7th June, 1947, p. 777.

Fat Metabolism.

As previously reported, Mr. Lincoln has been studying the enzymatic dehydrogenation of saturated fatty acids with the idea of determining whether dehydrogenation is a prerequisite for oxidation.

As examples of naturally occurring fatty acids, palmitic and myristic have been investigated, and margaric acid, a compound not found in nature, has been included. The reaction products following the action of dehydrogenase prepared from rat liver, and using methylene blue as a hydrogen acceptor, have been isolated. The experiments indicate that one double bond is introduced into the molecule; ozonolysis of the resulting unsaturated acids was carried out, and the reaction products

identified either as free acids or their silver salts. In some cases the aldehydes obtained from decomposition of the ozonides were isolated as 2:4 dinitrophenylhydra zones.

It is known that for fatty acid oxidation, adenylpyrophosphate is required, which transfers one of its labile phosphate groups to the fatty acid molecule forming a fatty acid-phosphoric acid anhydride, which is therefore the first reaction product.

Since experiments with the fatty acid dehydrogenase are usually carried out in a phosphate medium, it might be possible that adenylic acid is phosphorylated to adenylpolyphosphate, which can serve as phosphate donor. This possibility was tested by the use of dialysed extracts. Such extracts are inactive without adenylic acid, but after addition of the latter, full activity could be restored in a phosphate-free buffer solution. Thus adenosine triphosphate is not essential for the above dehydrogenase system.

From these experiments the following conclusions can be drawn. The dehydrogenation of higher fatty acids gives characteristic reaction products in each case, and is a process apparently independent of fatty acid oxidation, which requires adenosine triphosphate.

Carbohydrate Metabolism.

In earlier experiments by the writer and Mr. Nelson, it was shown that fructose markedly increases the utilisation of glucose by muscle tissue. In agreement with other workers, it was confirmed that fructose is not utilised by muscle tissue. These experiments were carried out in the spinal eviscerated animal receiving a continuous intravenous infusion of glucose. In control tests the decrease in blood glucose after the injection of insulin, was determined over a stated period. Under the experimental conditions it is usually accepted that the major portion of the glucose that is removed from the circulation is deposited as muscle glycogen. When fructose was added to the perfusion fluid, there was approximately a 100 per cent. increase in the glucose disappearance.

These experiments were repeated and extended on the isolated rat diaphragm muscle by Gemmill's technique. It was found that the amount of glucose disappearing in the absence of insulin was not altered by the addition of fructose. When insulin was present fructose greatly increased the rate of glucose disappearance, and the glucose lost could be fully accounted for as muscle glycogen. Further, fructose had no influence on the oxygen consumption of the isolated tissue.

In a previous report mention was made of studies on alloxan diabetes. It was shown that the severe hypoglycaemia which follows the injection of alloxan could be avoided if alloxan was given in increasing doses on successive days.

The investigation of the mechanism of alloxan diabetes has been extended.

The first effect of intravenously injected alloxan is hyperglycaemia lasting some three to four hours, and this is followed by profound hypoglycaemia which, if unrelieved by the administration of glucose, ends fatally.

The mechanism of the initial hyperglycaemia is by no means settled. Several workers consider that it is due to adrenal stimulation, whilst others believe that alloxan acts directly on the liver.

Amongst the former there is still further difference of opinion, some holding that the adrenal cortex is responsible, and others that the medulla is responsible.

In a series of experiments with adrenalectomised rabbits, the writer and Mr. Nelson have shown that the initial hyperglycaemia is completely abolished. These adrenalectomised rabbits show a precipitous hypoglycaemia with convulsions within one to one and a half hours. That the hyperglycaemia is due to the adrenal medulla, and not to cortex, was shown in a series of experiments with rabbits previously injected with ergotoxine ethanesulphonate. These animals showed a hypoglycaemia of similar rapid onset to those completely adrenalectomised. The hyperglycaemia produced after alloxan injection is apparent after a very short period. It is generally present after 15 minutes and always after 30 minutes. This is very suggestive that adrenalin is responsible, since there is no evidence to show that the hyperglycaemia due to the adrenal cortical hormones occurs in less than four hours, which is the maximum period for which alloxan hyperglycaemia lasts.

The hypoglycaemic action of alloxan is probably exerted through the pancreas, either by stimulating the pancreas to secrete insulin, or by damaging the islets and liberating preformed insulin. No satisfactory evidence has been presented in favour of the former theory, but there is histological evidence in favour of the latter view.

Houssay et al, however, deny that alloxan hypoglycaemia is mediated by the pancreas, and conclude that the effect is probably due to lack of glucose production by the liver. They do not present any experimental evidence in support of this theory, which is contrary to the results found from "in vitro" experiments.

Publications.

A. B. CORKILL:
"Some Clinical Aspects of Carbon-monoxide Poisoning" (In Press). The Medical Journal of Australia.

A. B. CORKILL:
"Carbohydrate Metabolism" (In Press). Proceedings of the Royal Australasian College of Physicians.

A. B. CORKILL AND J. F. NELSON:
"The Pituitary Gland and Carbohydrate Metabolism. Part I. The Posterior Lobe." The Medical Journal of Australia, 1st February, 1947, p. 130.

A. B. CORKILL AND J. F. NELSON:
"The Pituitary Gland and Carbohydrate Metabolism. Part II. The Anterior Lobe." The Medical Journal of Australia, 8th February, 1946, p. 172.

A. B. CORKILL AND J. F. NELSON:
"The Influence of Fructose on the Utilisation of Glucose by Isolated Muscle" (In Press). The Australian Journal of Experimental Biology and Medical Science.

CLINICAL RELATIONSHIPS

The Institute has again closely collaborated with the Honorary Medical Staff in the study of various diseases. During the year a Hartridge Reversion Spectroscope was purchased, and this has been of considerable value in the diagnosis of carbon-monoxide poisoning. In this connection, three interesting cases were studied. Various authorities in America have recommended the therapeutic use of hypertonic saline. Previously this measure had not been used in this Hospital, but in three cases in which it was used, there was an immediate and markedly beneficial effect on the restlessness and headache that persisted after consciousness had been regained.

Laboratory facilities have been granted to the Honorary Pathologist to the Institute, Mr. A. J. Trinca. Mr. Trinca, at the request of the Central Cancer Registry, is carrying out research on the histology of breast cancer.

Mr. C. J. O. Brown and the Thoracic Surgery Unit have been carrying out experiments in connection with Fallot's Tetralogy. At present they are concerned with the development of a technique for making systemic pulmonary shunts.

Mr. John Devine is concerned with various surgical problems. One of these is the reconstruction of the common bile duct employing peritoneal grafts and tubes of fibrin, the latter having been prepared by Dr. Fantl. Progress of this work will be described in the next report.

ROUTINE BIOCHEMISTRY DEPARTMENT

The work of this Department has been maintained at its usual steady level. With the introduction of new methods of diagnosis and treatment, additional tests have had to be developed; this applies particularly to prothrombin estimations. The anti-coagulant 3,3'ethylidene-bis (4-hydroxycoumarin) (E.D.C.), previously referred to, is now being used in this Hospital for the treatment of thrombotic states, and this has necessitated the introduction of prothrombin estimations as a routine procedure. There has also been an increase in serum phosphatase determinations, and liver function tests, particularly Hanger's flocculation reaction. Citric acid determinations have become a routine method for the investigation of diseases of bone.

We are again indebted to Mont Park Mental Hospital for the supply of control specimens of cerebro-spinal fluid.

During the year the following tests have been carried out:

Blood Urea Estimations	587
Urinary Protein	266
Urea Concentration	275
Urea Clearance	254
Blood Sugar Estimations	494
Blood Sugar Curves	174
Benedict Tests	30)
Acetone Bodies	307
Cerebro-spinal Fluid Examinations	438
Lange's Coloidal Gold Curves	27
Basal Metabolic Rate Determinations	204
Fouchet Tests	40
Van den Bergh Tests	89
Benzidine Tests	58
Pyramidone Tests	35
Urinary Diastase	31
Serum Calcium	32
Test Meals	341
Blood Chloride Estimations	10
Urine Examination for Bilirubin, Urobilin, Etc.	60
Prothrombin Estimations	77
Miscellaneous	431
Hanger's Cephalin Flocculation Test	36
	4 575
Electro-cardiographs	686
	5,261

Financial statements for the year are appended.

A. B. CORKILL, Director.

THE THOMAS BAKER, ALICE BAKER AND ELEANOR SHAW MEDICAL RESEARCH INSTITUTE.

Revenue Account for Year Ended 31st December, 1946.

EXPENDITURE				INCOME					
Medical Salaries	£1,373 11 1			Thomas Baker (Kodak), Alice Baker and Eleanor Shaw Benefactions				£7,200	0 0
Other Salaries and Wages	6,857 2 11			Alfred Hospital—Sale of Media				533	5 9
		£8,230	14 0	Grant—Department of Health				930	0 0
Drugs, Etc.		249	6 11	Interest on—					
Instruments and Glassware		333	17 10	Investments held by Trustees Baker Benefactions—					
Fodder for Animals		550	9 2	Australian Commonwealth Inscribed Stock	£552	10	0		
Fuel and Lighting		93	17 6	Endowment Investments—					
Insurance		40	14 10	Australian Commonwealth Inscribed Stock	162	10	0		
Repairs and Renewals		44	19 10	Grain Elevator Board	93	15	0		
Library—Maintenance		88	1 4	Inscribed Stock Australian Commonwealth Bonds	15	5	10		
Printing, Stationery and Postage		95	13 4					824	0 10
Travelling		36	5 10	Proceeds from Sale of—					
Telephone		59	19 11	Monograph	10	1	6		
Sundries		164	15 0	Vaccine, Etc.	80	6	1		
								40	7 7
				Biochemistry Fees				409	9 8
				Excess Expenditure for Year				51	11 8
		£9,988	15 6					£9,988	15 6

THE THOMAS BAKER, ALICE BAKER AND ELEANOR SHAW MEDICAL RESEARCH INSTITUTE.

Balance Sheet at 31st December, 1946.

LIABILITIES.						ASSETS.					
Endowment Funds				£8,000	0 0	Cash at Commercial Bank of Australia Ltd.				£549	12 3
Capital	£1,092	0	9			Investments—					
Revenue Account to 31st December, 1945	1,609	3	2			Grain Elevator Board Inscribed Stock	£2,500	0	0		
	£2,701	3	11			Commonwealth Inscribed Stock 3¼%, due 15/10/1960	5,000	0	0		
Less Revenue Account for Year 1946	51	11	8			Commonwealth Bonds, 3¼%, due 15/9/1961	500	0	0		
				2,649	12 3					8,000	0 0
						Furniture and Fittings				2,100	0 0
				£10,649	12 3					£10,649	12 3

Note: £17,000 Australian Commonwealth Inscribed Stock held by Trustees Baker Benefactions.

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