electrophoretic pattern of normal human plasma. Svensson's arrangement is apparently missing in the book.

In spite of such errors and oversights, Abramson, Moyer and Gorin's book on the electrophoresis of proteins represents a compilation of data on the electrophoretic analysis of proteins useful to all investigators of proteins.

B. F. CHOW

THE SQUIBB INSTITUTE FOR MEDICAL RESEARCH,
NEW BRUNSWICK, N. J.

CHEMISTRY OF DENTAL MATERIALS

Outline of the Chemistry of Dental Materials. By LAURENCE G. WESSON, research biochemist, Forsyth Dental Infirmary for Children, Boston, Massachusetts. 106 pp. 5×7.5. St. Louis: The C. V. Mosby Company. 1942. $1.50.

THIS book contains a concise review of some of the properties and uses of materials employed in dental practice. The chemical changes which many of these materials undergo and their effects on the oral tissues are clearly, although briefly, described. Such topics as the chemistry of vulcanization, and the formation of polymethyl methacrylate resin, which is used as a substitute for vulcanite in artificial dentures, will be of interest to the dentist. The sections on dentifrices, dental cements, the action of ammoniacal silver solution and photography should prove of value.

The dental section of the book is preceded by an elementary review of some of the principles of chemistry. Although brief, this material should be helpful to the dental practitioner. The descriptions of such topics as the nitrogen cycle and the potential acidity and alkalinity of food, although of general interest, could well have been omitted in a book of this type.

This outline should also prove useful as a supplementary text in courses for dental hygienists.

MAXWELL KARSHAN

COLUMBIA UNIVERSITY

SPECIAL ARTICLES

THE EFFECT OF TRYPTOPHANE DEFICIENCY ON REPRODUCTION[1]

PREVIOUS reports from this and other laboratories[2-7] have shown that tryptophane deficiency induces loss of weight, alopecia, cataract formation, corneal vascularization, defective dentition, testicular atrophy, hypoproteinemia and hypochromic anemia.[8] The present report describes observations we have made on the effect of a tryptophane deficient diet on the reproductive function in female rats.

Normal adult male and female rats from a hybrid albino and hooded Norwegian rat colony were maintained on stock diet and mated. As soon as vaginal smears showed the presence of sperm the females were segregated in individual cages and were fed a tryptophane deficient diet[7, 8] *ad libitum*. The data given in Table 1 show that all the rats on the deficient diet failed to cast a litter in contrast to a group of animals continued on the control diet all of which reproduced normally. The animals on the deficient diet were kept on it for 35 to 40 days. All of them lost weight and it was notable that symptoms of tryptophane deficiency developed earlier in these animals than in unmated rats on a tryptophane deficient diet. For example, alopecia, which in our experience is rarely evident before 60 days on the deficient diet, was very evident within 30 days in the present group of animals. Corneal vascularization was likewise well developed early in the deficiency period.

TABLE 1

THE EFFECT OF TRYPTOPHANE DEFICIENCY ON THE WEIGHT AND SIZE OF LITTER OF THE PREGNANT RAT

Group	Animal number	Initial body weight	Weight change for gestation period	Average daily food intake	Gestation period	Size of litter
		gms.	*gms.*	*gms.*	*days*	
Control	PCTH–3	201	+29	9.3	22	9
	PCTH–4	168	+43	7.6	23	6
	PCTH–5	209		7.7	22	7
	PCTH–6	221	+30	6.9	24	6
	PCTH–7	187	+41	9.2	22	8
	PCTH–8	208	+37	10.0	22	10
	PCTH–9	263	+27	10.0	22	10
	PCTH–10	232	+11	9.9	30	4
Deficient*	PTH–2	211	−40	9.1		0
	PTH–3	208	−13	8.0		0
	PTH–6	193	−63	8.0		0
	PTH–7	219	−24	7.6		0
	PTH–8	199	−29	8.9		0
	PTH–11	242	−43	6.9		0
	PTH–12	220	−40	7.0		0
	PTH–13	219	−22	9.7		0
	PTH–15	228	−35	9.6		0

* The weight change determined as of the 22nd day after insemination.

In order to determine the fate of the fetus in these deficient animals a second experiment was carried out similar to the above in which female litter-mates were

[1] This investigation was aided by grants from the Rockefeller Foundation, Merck and Company and E. R. Squibb and Sons.

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[6] J. R. Totter and P. L. Day, *Jour. Nutr.*, 24: 159, 1942.

[7] A. A. Albanese and W. H. Buschke, SCIENCE, 95: 584, 1942.

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placed on the deficient diet after mating. These animals were then sacrificed on the 9th, 11th, 13th and 14th days after insemination, and the uteri examined. It was found that embryos of normal size and appearance were present on the 9th day, whereas by the 14th day almost complete resorption had occurred, the embryonic masses being then scarcely discernible. A similarly treated control animal sacrificed on the 14th day of gestation showed twelve normal sized embryos.

It would appear from the above experiments that tryptophane is a most important dietary essential for normal gestation and that the available stores of the adult female rat are depleted in the course of ten days of a tryptophane deficient regimen.

We have found no reports in the literature dealing with the effect of tryptophane deficiency on the female reproductive function. However, numerous studies are to be found[9] on the deleterious effects of low protein diets on the reproduction of farm animals, and Guilbert and Goss[10] have shown that feeding a low protein diet to female rats results in reproductive failure. In the light of our findings it is pertinent to raise the question whether these effects of a low protein diet may be attributed to a low tryptophane intake.

ANTHONY A. ALBANESE
ROMAINE McI. RANDALL
L. EMMETT HOLT, JR.

DEPARTMENT OF PEDIATRICS,
JOHNS HOPKINS UNIVERSITY

INFLUENCE OF FEVER UPON THE ACTION OF 3,3′-METHYLENE-BIS-(4-HYDROXY-COUMARIN) (DICUMAROL)

FOLLOWING the isolation and synthesis of the anticoagulant Dicumarol (3,3′-methylene-bis-(4-hydroxycoumarin) by K. P. Link and collaborators,[1,2] this drug has rapidly gained the attention of scientists and clinicians alike. Since this drug will frequently be used in conditions accompanied by fever, it appeared desirable to investigate the influence of higher body temperatures upon its action. It is well known that biochemical reactions are accelerated by increase in temperature, but the degree of this acceleration varies greatly from instance to instance, depending on the type, and may show unexplained abnormalities.[3]

Fever was induced in rats by the injection of a yeast suspension or typhoid vaccine which resulted in a marked rise of temperature 5 to 15 hours later. If necessary, a second injection was made. Twelve hours after the administration of the pyrogenic agent, the rats were fed 2.5 mg Dicumarol orally. Prothrombin time was determined in 12.5 per cent. plasma 24 hours after Dicumarol administration, using the method described by Link and associates.[4] In normal rats this dose of Dicumarol raises the prothrombin time from about 45 seconds to 1 minute, 47 seconds. Fever alone causes no or only a very slight increase in prothrombin time. However, animals having received Dicumarol during fever showed a tremendous increase of the prothrombin time as compared with the rats having a normal temperature.

TABLE 1

EFFECT OF 2.5 MG DICUMAROL IN RATS (PROTHROMBIN TIME OF 12.5 PER CENT. PLASMA 24 HOURS AFTER ADMINISTRATION)

	Group				
	A	B	C	D	E
Maximal rectal temperature (°F)	99.2 ± .3	100.3 101.0	101.2 102.0	102.2 103.0	103.2 104.0
Number of animals..	9	7	10	7	11
Prothrombin time ...	1′47″	4′06″	5′55″	6′10″	8′55″
Standard error	0′10″	0′25″	0′50″	1′04″	0′56″
Group A : Normal rats		Group B–E : Rats in fever			

The degree of this increase of the prothrombin time varied considerably but tended to become more pronounced with higher temperatures. A response to Dicumarol falling within the limits of normal variation occurred only three times among the rats in the stage of fever. To facilitate tabulation the value of 12 minutes was deliberately attributed to experiments in which clotting was delayed for an indefinite period in excess of 12 minutes. This occurred once in Group C and D and five times in Group E. For classification we used the maximal temperature attained during the experiment. Due to the variation of the response and the necessary deliberate form of classification, the standard error of the single groups is considerable, but statistical evaluation has shown that the differences between normal and pyretic rats are significant.

Details and further work in which changes in the metabolic rate were produced by increasing the environmental temperature or by other methods will be reported later. As far as we know, no definite observations of this type have been published so far. Our observations suggest that the administration of this drug should be particularly watched in patients having an increased body temperature, to avoid a dangerous depression of the prothrombin level.

R. K. RICHARDS

DEPARTMENT OF PHARMACOLOGY,
ABBOTT LABORATORIES,
NORTH CHICAGO, ILL.

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