Results of an experiment of this type are shown in Fig. 1. Tube 1 contains contractile protein incubated with platelet extract in the absence of ATP, tube 2 contractile protein and ATP without platelet extract, tube 3 contractile protein with ATP and platelets extract, tube 4 contractile protein with ATP and platelet extract to which calcium was added to a calculated final concentration of 0·1 mM Ca^{++}.

Comparison of tubes 1 and 2 shows the degree of super-precipitation obtained in the presence of ATP. Tube 4 shows the same degree of super-precipitation, indicating that the platelet extract is inactive in the presence of free calcium ions. In contrast, tube 3 shows complete solution of the contractile protein by the combined effect of ATP and the platelet extract. These results are identical with observations previously made on the behaviour of actomyosin in the presence of ATP and relaxing factor from muscle[8]. Partial inhibition of the ATPase activity of platelet contractile protein by the platelet extract was also found to occur.

It may be concluded that an activity having the same fundamental characteristics as the relaxing factor of striated muscle is present in pig blood platelets. So far as is known this represents the first demonstration of relaxing factor activity in cells other than those of heart and skeletal muscle. The presence of this factor—the activity of which is destroyed by calcium ions—in platelets is clearly in agreement with the reaction mechanism proposed for the 'release reaction' in my earlier report[2] and explains the cardinal role of calcium ions in the process. Simultaneously, the part played by calcium in the physiologically important alterations of viscous metamorphosis in platelets is thus explained, since according to my observations[2] viscous metamorphosis represents a parallel effect of the mechanism which is responsible for the release phenomenon.

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PHYSIOLOGY

Extra-uterine Re-circulation of Iodine-131 from the Young to Mother in Rats

Suckling rats or puppies, separated from their mother during the first two weeks of life, cannot urinate spontaneously even though the bladder may be filled with urine[1]. Although these findings have indicated the dominant role of the mother in the regulation of excretion of urine in such immature mammals, no attention has been paid to the possibility that mothers may also ingest the urine of their young. To examine this problem the transfer of iodine-131 from young rats through the lactating mothers into the body of the intact young was investigated.

The experiment involved 10-day-old rats of the Wistar strain from three different litters. Eight animals from each litter were divided into two equal groups, *A* and *B*. Group *A* received intraperitoneally 2 μc. of iodine-131 and were then reunited with their mother for 4 h. During this time the animals of group *B* were isolated in another cage. 4 h later the animals of group *A* were killed and the mother was transferred to group *B*. At the end of a 4-h period during which the animals were able to suckle, they and the mothers were killed by bleeding from the aorta abdominalis under ether anæsthesia. The radioactivity in the serum, stomach and thyroid gland was determined in a well-type scintillation counter.

Table 1

	Litter	Group *A* ^{131}I injection 4 h with mother	Mother	Group *B* 4 h isolation followed by 4 h with mother
Stomach	1	333,300*	5,910	24,130*
	2	340,110	2,662	14,054
	3	311,114	3,970	32,774
Thyroid gland	1	92,170	56,510	1,577
	2	146,595	22,050	2,647
	3	153,470	72,130	1,028
Serum	1	46,740	485	6,000
	2	46,595	121	986
	3	47,770	408	1,257
Urinary bladder	1	7,640		1,722
	2	40,373		1,310
	3	101,280		353

* Values represent means of c.p.m. per total organ or 1 ml. of serum in groups of four animals.

The results (Table 1) indicate that a considerable quantity of iodine-131 administered to 10-day-old rats of group *A* re-appears in the organism of intact nurslings of group *B*, after passing through the organism of the mother. This presence of iodine-131 in the stomach, serum and thyroid gland of uninjected sucklings demonstrates a rapid re-circulation of iodine between immature rats and their mother in both directions. The observed ingestion of urine excreted by immature animals may represent a saving mechanism which could include several other substances in order to compensate for their loss via the milk. This suggests the possibility that in species with immature new-born animals the mother may be responsible for the maintenance of their normal internal environment in a manner very similar to that occurring during the intra-uterine life.

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Water-induced Cerebral Over-hydration in the Maturing Rat Brain

Cerebral injury due to anoxia and ischæmia in the adult rat is accompanied by œdema of the brain[1–3]. Recent work has shown that swelling of the brain in these circumstances does not occur in rats aged less than ten days[4]. Although this may be a consequence of the increased resistance of the young mammal to anoxia[5,6], there could be primary inability of the brain to develop œdema before a certain stage of development is reached. In order to test the latter possibility, the propensity to develop cerebral œdema following administration of water was investigated in rats of different ages.

Colony bred, Wistar rats aged 1–55 days were used. Animals were given 50 ml./kg of distilled water intravenously under ether anæsthesia. Litter-mates acted as untreated controls. In the first week of life, intravenous infusions were made into the facial vein, using a *G* 27 gauge luer hypodermic needle connected by a polythene tube to a tuberculin syringe. In older animals, the water was injected into the external jugular vein via a polythene cannula. One hour after administration of water both the test and control animals were killed by light ether anæsthesia followed by exsanguination. The forebrains were rapidly removed and the dry weights were estimated by weighing before and after drying to constant weight at 110° C.