present exporiments challonge the discussion in many other directions, only the aspects pertaining to problems implied in the aforementioned hypothesis will be briefly commented. Despite the high degree of differentiation, various portions of the grey matter of the central nervous system could still be considered morphologically similar in that they are all composed of neurones, glia cells, unmyelinated and myelinated fibres, blood vessels, elements of the supposedly common ground substance, etc. Therefore, in view of the high specificity of serological reactions, so amply attested by Landsteiner[7], it can be assumed that immune γ-globulins used in our experiments contained a bulk of antibodies against the number of various components present in homogenates of given brain regions, including non-specific ones, common to the brain as a whole. Yet, as could be judged on the basis of variations in contents of histamine-like substance found in the present experiments, anti-caudate and anti-hippocampal γ-globulins were still able to 'recognize' the corresponding nervous structures. It could be inferred from these considerations that the predominant (if not selective) increase in the histamine-like substance content in the part of the brain attacked by the corresponding antibody indicates that in the antigen–antibody reaction are involved primarily those elements (most likely cellular, whatever they might be: neurones, glia or both) which determine the antigenic specificity of the given nervous structure.

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Possible Role of a Phospholipid in the Development of the Slow Muscle Contracting Activity of Human Plasma

The presence of a substance in the *G*.2 fraction of human blood plasma with a slow contracting activity on plain muscle (S.M.C.) was established and the active principle has been isolated from the *G*.2 fraction and designated as *G* acid[1]. It is important to note that the same effect has been obtained from citrated human plasma and is best observed with dialysed plasma, since dialysis eliminates the quick contracting agents[2]. The chemical nature of *G* acid prompted a search for a possible precursor of this lipid-soluble unsaturated fatty acid (3-octadecenoic acid)[1].

Although the *G*.2 fraction stimulates the isolated guinea pig ileum, the γ-globulin and the albumin fractions are much less active on the same muscle preparation. The S.M.C. effect of human plasma and of *G*.2 fraction is reduced to about 50 per cent of the initial effect after the addition of calcium ions to a final concentration of M/40. The serum prepared by recalcification of the plasma is much less active on smooth muscle than the serum obtained after clotting the same plasma with human thrombin.

The formation of a slow muscle-contracting substance by alkaline hydrolysis of lecithin or by the action of phospholipase *A* present in cobra venom on lecithin[3], and the recognition of another variety of phospholipid in human serum identified as lysolecithin[4], suggested the investigation of the effect of human plasma on egg lecithin *in vitro*. The results obtained show that the addition of egg lecithin (0·25 per cent) to plasma causes a marked increase in its S.M.C. activity which is neutralized by the addition of calcium ions to a final concentration of M/40. Lecithin alone is inactive on the isolated ileum of a guinea pig at the same level of dosage. Incubation of the plasma with lecithin at 37° C or raising the *p*H of the plasma to 8 increases the slow contraction of smooth muscle produced by lecithin-plasma. Olive oil, cotton-seed oil and butter fat do not produce similar effects on smooth muscle when emulsified with human plasma and incubated at *p*H 8.

It may be interesting to note that the amount of egg lecithin which doubles the S.M.C. effect of human plasma is about 250 mg per cent of the plasma, a concentration which roughly corresponds to the concentration of the naturally occurring choline-containing phospholipids in human plasma. Parallel results were not obtained after incubation of egg lecithin with 5 per cent solution of human serum albumin in saline.

The S.M.C. effect produced by lecithin added to human plasma is not antagonized by atropine or by antazoline. Similar to the S.M.C. effect of the original plasma, the S.M.C. effect produced by added egg lecithin is not destroyed by an esterase-rich fraction of blood which destroys acetylcholine under the same conditions. The esterase-rich fraction is produced during the purification of human serum albumin by the method of Kekwick and Mackay[5].

Processing of human plasma with kaolin considerably decreases its effect on the isolated ileum of a guinea pig[6,7]. The S.M.C. effect produced by lecithin added to human plasma is completely abolished by the same treatment. The S.M.C. effect of such plasma is indistinguishable from the S.M.C. effect of kaolin-treated plasma without added lecithin.

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Brown Adipose Tissue and Thermoregulatory Heat Production in the Rat

The role of brown adipose tissue in thermoregulatory heat production of the cold-adapted rat was inferred by R. E. Smith from the thermogenic activity of the interscapular fat *in vitro*[1] and the anatomical distribution of brown adipose tissue in the body[2]. The thermogenic role of brown fat has been directly demonstrated in the hibernating hamster by measuring temperatures at various sites in the body[3], and by measuring simultaneously the oxygen consumption of the whole animal, in the new-born rabbit[4,5].

In our experiments, copper-constantan thermocouples were inserted under light urethane anæsthesia (0·8 g/kg body-wt.) into adult rats adapted to room temperature at the following sites: (1) midbrain, 7 mm deep; (2) colon, 10 cm from the anus; (3) interscapular fat; (4) muscle,