trouble: however, the results were confirmed by absorption tests.

How alike the gibbon Xg^a antigen is to that of human beings may ultimately be seen from chemical analysis, but at present it is indistinguishable: absorption of the two available examples of anti-Xg^a by Xg(a+) gibbon cells removed the antibody for human and for gibbon Xg(a+) cells, as did absorption by human Xg(a+) cells. Absorption by gibbon or human Xg(a−) cells did not remove the antibody for Xg(a+) members of either species.

The distribution of the antigen in the two sexes suggests that it may be X-linked in the gibbon as it is in man. If the antigen were an autosomal character in the gibbon the chance of observing such a distribution would be 1 in 3. It is hoped that more gibbons will be available in 1965 and that the question of X-linkage will then be settled.

That the gibbon Xg^a antigen may be X-linked is perhaps the less surprising since hæmophilia is X-linked in the dog[8] as it is in man, and the clotting factor deficiency appears to be the same[9]. The presence of Xg^a in the gibbon can scarcely be considered taxonomically important, though X-linkage, if confirmed, would suggest that there is some powerful evolutionary reason why the gene for this antigen should be located on the X chromosome.

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PATHOLOGY

Serum Enzymes in the Jaundiced Homozygous Gunn Rat

In the course of investigations using jaundiced Gunn[1] rats, serum enzyme examinations were performed on these rats and on those of a Wistar strain. Jaundiced homozygous Gunn rats are lacking in hepatic glucuronyl transferase using bilirubin as a substrate. Many workers have established that hepatic cellular damage is associated with elevation of certain serum enzymes, and an investigation was undertaken to compare the serum enzyme levels in jaundiced Gunn rats and those of a Wistar strain to determine whether the jaundice was associated with raised serum enzyme activities.

Blood specimens were obtained from the tail vein of male rats and the serum was analysed for glutamic-oxalacetic and glutamic-pyruvic transaminase[2], lactic dehydrogenase[3], cholinesterase[4] and aldolase[5]. Normal values for the activities of the five serum enzymes were established by analysing random blood samples in 25 Wistar strain rats. The serum enzyme activities in jaundiced Gunn rats were established by analysing random blood samples in 16 rats.

Table 1. Serum Enzyme Activities in 16 Jaundiced Gunn Rats and 25 Rats of a Wistar Strain

Results (units/ml.) given as mean and standard deviations

	Gunn rats	Wistar rats
Glutamic oxalacetic transaminase	117 ± 24	124 ± 53
Glutamic pyruvic transaminase	36 ± 9	41 ± 13
Lactic dehydrogenase	2,540 ± 560	2,408 ± 662
Aldolase	133 ± 34	128 ± 36
Cholinesterase	51 ± 17	55 ± 24

There was no significant difference between the enzyme levels in jaundiced Gunn rats and those of the Wistar strain (Table 1). The transaminase levels in these rats were lower than those found in male Sprague–Dawley rats by Altland and Highman[6]. They found levels of 261 ± 25 units and 63 ± 3 units for glutamic-oxalacetic and glutamic-pyruvic transaminase, respectively. Lactic dehydrogenase and aldolase activity was greater than that found by Altland and Highman, who found levels of 1,082 ± 72 units and 68 ± 3 units for lactic dehydrogenase and aldolase, respectively. Sibley and Lehninger[5] found a range of aldolase activity of 30–100 units/ml. in male Sprague–Dawley rats.

The results show that lack of glucuronyl transferase in jaundiced Gunn rats is not associated with increased serum enzyme activity for those enzymes tested.

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Demonstration of Hydroxyindole-O-methyl Transferase, Melatonin, and Serotonin in a Metastatic Parenchymatous Pinealoma

Pinealomas have been associated with two endocrine syndromes: destructive, non-parenchymal lesions frequently produce precocious puberty in young boys; true parenchymatous tumours have been found in children with delayed pubescence[1]. Little is known about the biochemistry of these tumours, or about possible mechanisms by which they exert their pathological effects on gonad function.