metacentric chromosomes is to be considered more 'specialized' and diverged from the ancestral type, while the slender loris, *L. t. lydekkarianus* with 62 chromosomes comprising both acro- and metacentrics, is regarded as 'generalized' and nearer to the ancestral type.

The sex chromosomes in pachytene are confined to the sex vesicle as in man, mouse, and field vole. The exact nature of association within the vesicle is not clear. At later stages, however, the 2 segments, the euchromatic and heterochromatic, are recognisable. Homology between the sex chromosomes can be accounted for only in this short euchromatic segment. Chiasma formed in this segment, rapidly terminalises to form typical end-to-end type of association. Similar association has also been observed in the hamster sex chromosomes[6] and is considered to be due to the crossing over in a minute euchromatic segment which terminalizes before they become visible as separate units.

Failure to demonstrate a side-to-side paring between *X* and *Y* in many mammals has raised doubts regarding chiasma formation between them[7-9]. However, recent electron microscope studies[10] have clearly demonstrated a side-to-side pairing in the sex vesicle of hamster and mouse[11].

Zusammenfassung. Die diploide Chromosomenzahl bei *Loris tardigradus lydekkarianus* beträgt 62, und der Karyotyp besteht sowohl aus akro- wie aus metazentrischen Chromosomen. Beobachtungen über die meiotischen Chromosomen des Männchens ergeben, dass sich die Chiasmaformation zwischen *X* und *Y* befindet.

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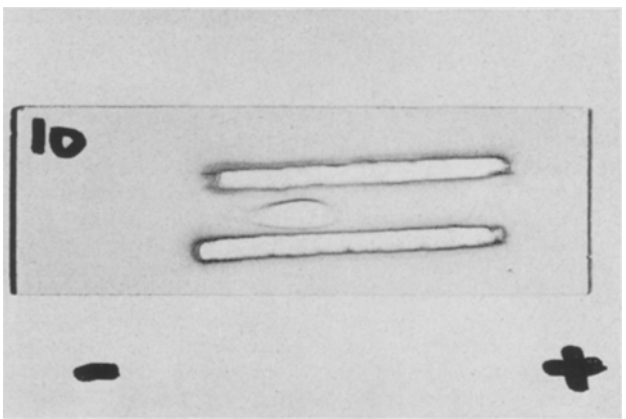
A Serum Antigen Detected in Cattle by Micro-Immuno-Diffusion

Interest in inherited differences in serum antigens (allotypes) has been increasing in recent years in view of their importance in genetics and in plasma protein chemistry. Intraspecific genetic differences in serum proteins have been described for a number of mammalian species[1-5]. Very recently they have been detected in cattle[6] also. This communication reports a serum antigen detected in cattle by micro-immuno-diffusion. The antiserum used for these studies was obtained by iso-immunization (one injection s.c. and two i.m., each at an interval of 10 days; and 6 months later, as a booster dose, one more injection s.c.) with pooled bovine sera diluted 1:1 with hemagglutination buffer (Difco Laboratories, Detroit, USA) and then emulsified with an equal volume of Freund's complete adjuvant. Before being diluted, the pool of bovine sera was frozen and thawed 8 times to produce some slight denaturation or alteration of the proteins and in this way to increase their antigenicity. Serum samples were tested on 26×76 mm microscope slides using 1% Special agar-Noble (Difco Laboratories, Detroit, USA) as supporting medium. The diameter of the wells was 1.7 mm, the distance between the central well and the 4 peripheral wells was 2 mm.

Each serum sample was tested undiluted as well as diluted by two-fold dilutions up to 1/8. The antiserum was used undiluted. The slides were read after 12 h. At this time all the precipitation lines had developed and further incubation did not reveal additional lines.

The mobility of the Ci(a+) antigen (Figure) was studied by immuno-electrophoresis using the micromethod described by HIRSCHFELD[7] (barbiturate buffer, pH 8.6, 7 V/cm for 90 min).

The data of the Table correspond well with the results expected if the antigen follows mendelian segregation with negative subjects homozygous for a recessive gene Ci and positive subjects homozygous or heterozygous for the allelic gene Ci^A (named provisionally using a two letter abbreviation derived from the name of the animal that produced the antiserum). As expected, matings of negative parents gave only negative offspring. Matings of positive parents gave at least 75% positive offspring



Immuno-electrophoresis mobility of the Ci(a+) antigen. Direction of migration: from left (cathode) to right (anode). Amidoschwartz staining. Upper trough: antiserum collected after the booster dose (same antiserum used for the immuno-diffusion studies). Lower trough: antiserum collected after the third injection.

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and matings between positive and negative parents gave at least 50% positive offspring. The frequency of the gene Ci^A, estimated from the frequency of the phenotypes in the 277 offspring of the Table, was 0.43.

To study the constancy of the Ci(a+) antigen, from 4–6 serum samples, collected from 10 animals (6 positive and 4 negative) over almost 1 year time, were tested all at once and found to give consistent results. An animal, tested 12 h after birth, was positive. His mother was negative. This shows that the antigen Ci(a+) expresses itself at birth or soon after.

Whether this antiserum and the one described by RAPACZ et al.[6] are related, remains to be ascertained. At present it is known that they have been obtained using different antigen preparations: whole serum for the one described here and an ammonium sulphate fraction of serum for the one described by RAPACZ et al.[8,9].

Inheritance of the antigen

Type of mating	No. of matings	Offspring positive	negative	total
+ × +	26	20	6	26
+ × −	162	93	69	162
− × −	89	0	89	89

Riassunto. È descritto un antigene identificato nel siero dei bovini mediante microimmunodiffusione. I dati familiari mostrano che esso è sotto controllo genetico. L'antisiero è stato ottenuto mediante isoimmunizzazione con pool di siero bovino diluito 1:1 con soluzione tampone ed emulsionato con un egual volume di adiuvante completo tipo Freund.

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Identification of Ceruloplasmin in Human Liver Cells by Fluorescent Antibodies and Absence of this Protein in Wilson Disease

Ceruloplasmin is a copper-containing blood plasma protein regulating copper metabolism in the human organism. There are data indicating that ceruloplasmin synthesis occurs in liver[1,2]. There is some evidence that in Wilson disease the patient's ceruloplasmin does not differ in general physico-chemical characteristics from that in normal subjects[3,4]. The decrease of ceruloplasmin concentration in the patient's blood is due to genetically determined inhibition of the protein synthesis. However, there has been no direct evidence so far to support this hypothesis.

The present paper reports some direct evidence of ceruloplasmin synthesis in normal human liver cells and almost entire absence of liver ceruloplasmin in Wilson disease.

Materials and methods. Slices of liver, brain, spleen, heart and kidney from a practically normal male, aged 37, who died of a sudden chest compression, were studied. The tissues of these organs were taken 2 h after registration of clinical death. Liver tissues taken in biopsy by liver puncture according to MENGINI's technique[5] were obtained from 8 patients with liver disorders of non-congenital ethiology (concentration of ceruloplasmin 45–60 mg 100 ml) as well as 3 with Wilson disease (concentration of ceruloplasmin 3–6 mg 100 ml). To obtain anticeruloplasmin serum, rabbits were immunized with human homogenic ceruloplasmin by the method described earlier[6]. For identification of ceruloplasmin in slices of various human organs COONS's indirect method was used[7]. Anticeruloplasmin serum adsorbed to remove antibodies to other plasma proteins by diazotized protein complex from a patient with Wilson disease was used as an intermediant. The adsorbtion of plasma protein antibodies of intermediant serum was complete enough since precipitation on agar gel by OUCHTERLONY's technique[8] with human ceruloplasmin, blood serum from normal subjects and those with Wilson disease showed only one arc (Figure 1). Preparations from organs obtained in autopsy and biopsy were immediately frozen and treated as described earlier[2]. The fluorescence observed was considered specific only if no fluorescence was seen in control preparations[2]. The blood serum ceruloplasmin concentration was determined by RAVIN's method[9].

Results. When studying slices of human kidney, spleen, brain and heart obtained in autopsy, no localized specific fluorescence was observed in the cells of these organs. Fluorescence was of diffuse character and slight intensity. In liver preparations, where anoxia is extremely high, specific fluorescence was seen extracellularly, i.e. in the intercellular space (Figure 2). However, certain parenchimatous cells showed sites of specific fluorescence

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