Lectin	Human blood group specificity	mol.wt	Subunits	Carbohydrates	Cys/2	Stability
A. edulis I	no	60,000	4	18%	0	75 °C
A. edulis II	no	32,000	2	2%	0	85 °C
A. bisporus	no	64,000	4	4%	–	–
A. campestris	no	64,000	4	4%	4	85 °C

the footpads with a mixture of 10 mg lectin in 2 ml Freund's complete adjuvant once a week for 4 weeks. The animals were bled 14 days after the final injection. The fraction of immunoglobulins was isolated by precipitation with ammonium sulphate (degree of saturation 0.33). Double diffusion in agar was done using 1% agarose in 0.05 M sodium barbital buffer pH 7.4.

Results and discussion. Heating of the crude extract of *A. edulis* at 75 °C results in the precipitation of most of the proteins but not of the lectins. Gel chromatography of the heated lectin extract on Sephadex G_{75} results in a fraction of high molecular polysaccharide and 2 lectin-containing fractions. Rechromatography of the lectin fractions reveals 2 lectins (figure). The lectins are homogenous in disc electrophoresis and each of them shows a single band. The amount of lectins obtained from 100 g dried plant material was about 12 mg of lectin 1 and 280 mg lectin 2, respectively. Mol.wt estimated by thin-layer gel filtration was about 60,000 for lectin 1 and 32,000 for lectin 2. Mol.wt of the subunits of both lectins estimated by disc electrophoresis in the presence of sodium dodecylsulphate was about 14,000. The values for carbohydrate content obtained by the phenol-H_2SO_4 method were 18% for lectin 1 and 2% for lectin 2 with D-glucose as standard. The carbohydrates were not identified further. Neither cystein nor cystine was found in either of the lectins. We suggest that lectin 1 consists of 4 subunits and lectin 2 of 2 identical subunits which are noncovalently bounded. Only L-valine was found as N-terminal amino acid of lectin 2 (that of lectin 1 has not yet been determined). The hemagglutination of lectin 2 was studied in the presence of the following carbohydrates: D-glucose, D-mannose, D-galactose, L-fucose, L-rhamnose, D-glucosamine, D-galactosamine, N-acetyl-D-glucosamine, N-acetyl-D-galactosamine and N-acetyl-neuraminic acid. None of the sugars tested inhibited the hemagglutination of human erythrocytes in a final concentration of 0.1 M. 5% solutions of dextran, mannan (from yeast) and galactan (from *Lupinus albus*) showed no inhibition activity. The purified lectins showed no specificity for one of the human erythrocyte types. Erythrocytes of the types A, B and 0 were agglutinated. Blood group substances A and B were precipitated by the lectins. The activity of lectin 1 and 2 was unaffected by heating for 10 min at 75 °C and 85 °C, respectively. Also, incubation of the lectins in a solution of 6 M urea at room temperature for 60 min and in buffers from pH 2–11 had no influence of the hemagglutinating activity. When tested in immunodiffusion against the immunoglobulins IgA, IgG and IgM the lectins from *A. edulis* showed single bands against the immunoglobulins. That means the lectins are able to react with carbohydrate moities of the immunoglobulins. Also the hemagglutination of human erythrocytes by 1 mg lectin II per ml phosphate-buffered saline (titer 1:1024) was inhibited by equal parts of 0.2% IgA (titer 1:2) and 0.2% acid α_1-glycoprotein (titer 1:4). When tested in immundiffusion against the antiserum to lectin 1 and 2 a crude extract of *A. bisporus* (titer 1:128) revealed cross reaction of the lectins, showing relationships between the lectins. Some properties of the lectins from *Agaricus* are summarized in the table. Further studies of the structure and nature of the lectins from *A. edulis* are under way in our institute.

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Abnormal limbs *(abl)*, a recessive mutation affecting the tadpoles of *Xenopus l. laevis*

Anne Droin and M. Fischberg[1]

Station de Zoologie expérimentale, Université de Genève, 154 route de Malagnou, CH-1224 Chêne-Bougeries (Switzerland), 18 February 1980

Summary. 'Abnormal limbs' *(abl)* is a recessive and semi-lethal mutation introduced by a ♂ from the laboratory stock. Brachymely, syndactyly, polydactyly and brachydactyly are the main abnormalities encountered. They occur more frequently in the forelimbs than in the hindlimbs.

This mutation, provisionally called M_5, was found during the genetic analysis of somatic nuclei undertaken in our laboratory after nuclear transplantation experiments; it was introduced by a male from the stock. The heredity of the mutation has already been described[2].

10 matings were effected between heterozygous individuals of different generations; they have yielded 230 homozygous tadpoles out of 956, i.e. a percentage of 24.1, characteristic of a Mendelian recessive gene.

Description of the phenotype. The phenotype of the mutant tadpole is recognizable at about 22 days of development, i.e. at stage 52 of the normal table of *Xenopus l. laevis*[3]. The proximal parts of the most abnormal limb buds are usually wider and thicker in the *abl* tadpoles than in the wild-type

Abnormalities of the limbs in the mutant *abl* of *Xenopus l. laevis*

Matings	Number of tadpoles*)	Brachymely		Syndactyly		Polydactyly		Brachydactyly		Clinodactyly		Supernumerary claws
		f	h	f	h	f	h	f	h	f	h	h
3×13	19	14	6	19	2	11	1	10	–	4	1	4
10× 6	17	17	13	13	3	9	5	6	2	6	2	5
11×14	16	16	12	15	7	16	7	15	4	5	1	8
Total	52	47	31	47	12	36	13	31	6	15	4	17

* As asymmetry was very rare (6/52 cases), only 1 forelimb (f) and 1 hindlimb (h) have been considered per tadpole.

ones whereas the distal parts are shaped normally. As the buds elongate their orientation becomes abnormal; instead of growing along the body, they can be oriented either ventrally or completely turned round, pointing rostrally. At metamorphic climax (stage 58) limb development is well advanced and the abnormalities very distinct (figure 1). Except for the limbs, general development proceeds normally but the *abl* tadpoles reach stage 58 slightly later than the wild-type ones.

During metamorphosis, usually from stage 61 onwards, the froglets with the most abnormal limbs develop oedemata, most frequently of the dorsal lymph sacs and of those of the hindlimbs (figure 1, d). At the end of metamorphosis, most of the mutants die; 4 tadpoles out of 39 homozygotes, belonging to 3 matings, passed metamorphosis and gave rise to adults. This mutation can thus be considered as semi-lethal.

The main abnormalities are summarized in the table. The total numbers indicate that brachymely or shortening of the limbs is the most frequent malformation encountered followed by syndactyly or fusion of the digits, then by polydactyly (too many digits) and brachydactyly (shortening of the digits) of the forelimbs; brachydactyly of the hindlimbs and clinodactyly or bent digits are more rare, while a relatively frequent anomaly of the hindlimbs is the occurrence of supernumerary claws; nearly 1 for every digit instead of the 3 present in the normal hindlimb (figure 1, b and c). In addition, 3 other abnormalities were found; 1 case of ectrodactyly (4 digits instead of 5 in a hindlimb), 1 case of phocomely (the hand directly attached to the body) and 1 case of diplopody or duplication of the autopodium of the hindlimb.

When the matings are considered separately, one observes a great variability in the expressivity of the mutation (table). Mating 3×13 shows a weak expression, whereas it is strong in the offspring of mating 11×14 in which the forelimbs are severely affected by the 4 main malformations and 75% of the hindlimbs are affected by brachymely.

Fig. 1. *Xenopus laevis* froglets at metamorphosing stages; *a* Normal; *b–d* samples of *abl* showing different types of abnormalities; in *b* and *c* inserts, the supernumerary claws are visible (arrows); in *d* the oedemata of the lymph sacs are conspicuous.

In between these extremes one finds great variation, for example a medium expression presented by the tadpoles of mating 10×6.

A succinct anatomical analysis of whole limbs submitted to the transparency technique after staining with Victoria blue[4] showed conspicuous malformations of the cartilages, more pronounced in the forelimbs than in the hindlimbs. At all levels of the limbs the cartilages may be missing, short and wide, fused or duplicated, and bifurcated or distorted (figure 2).

This is the 2nd mutation affecting the limbs found in our stock of *Xenopus l. laevis,* the other one being 'polydactyly' *(pd)*[5]. The *pd* phenotype presents some similarity with *abl* but matings between frogs heterozygous for these 2 mutant genes gave only normal offspring, showing that these mutations are not allelic.

1 Acknowledgment. The authors are indebted to Prof. A.W. Blackler, Dr I. Hadji-Azimi and Mrs J. Gazel-Stock for their valuable collaboration. This work was supported by the Fonds national de la Recherche scientifique (requêtes No 3.60.68 et 3.221.73).

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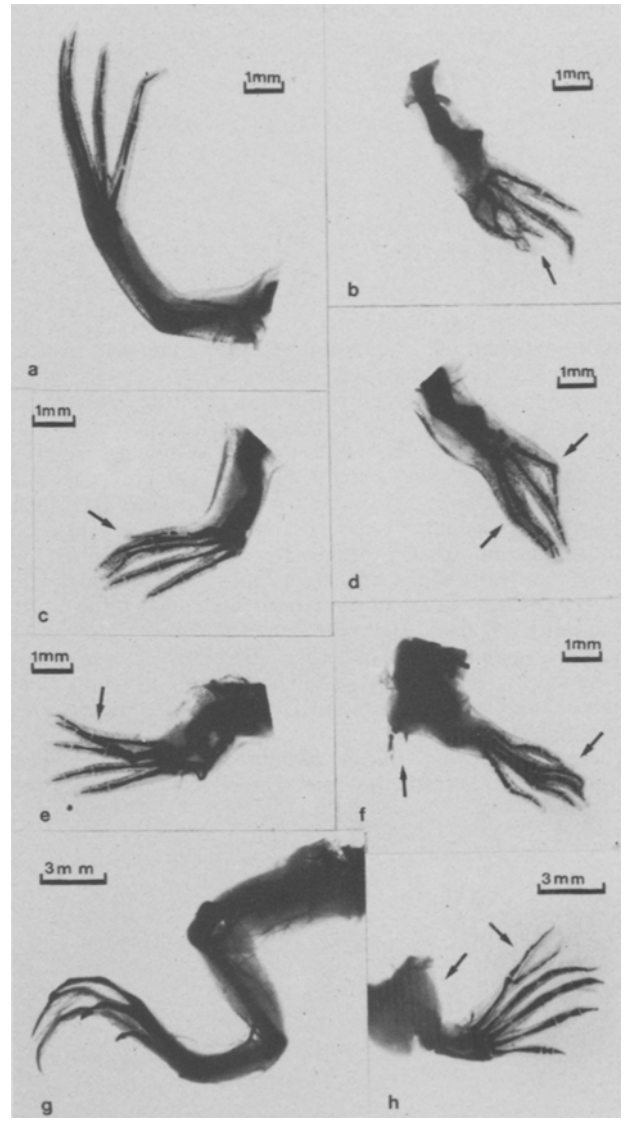

Fig. 2. Samples of limbs submitted to the transparency technique; *a* normal forelimb; *b–f* abnormal forelimbs, all showing brachymely. Arrows point to the main abnormalities observed: in *b,* brachydactyly; in *c,* polydactyly; in *d,* syndactyly and clinodactyly; in *e,* syndactyly and polydactyly; in *f,* syndactyly, polydactyly and abnormally situated supernumerary digits; *g* normal hindlimb; *h* abnormal hindlimb showing brachymely and polydactyly (arrows).

Distribution of constitutive heterochromatin (C-bands) in the somatic chromosomes of an Indian bird, *Chrysomma sinense* (Gmelin)

T. Sultana and S.P. Bhunya[1]

Post Graduate Department of Zoology, Utkal University, Vani Vihar, Bhubaneswar-751004 (Orissa, India), 23 November 1979

Summary. The localization of constitutive heterochromatin has been studied in a passerine bird, *Chrysomma sinense* ($2n = \pm 70$). In all the 7 pairs of macrochromosomes pericentric heterochromatin has been observed as usual except in pairs Nos 2 and 4, in which both pericentric and non-centromeric heterochromatin have been recorded.

Though the function of constitutive heterochromatin is largely unknown[2], several possibilities have been proposed[3–7]. In birds, studies on the amount and distribution of constitutive heterochromatin are very scanty. Usually constitutive heterochromatin has been recorded in W-chromosomes[8] and the centromeric regions of Z and autosomes. In most cases, most of the microchromosomes have been reported to be entirely heterochromatic[9,10]. There are insufficient data concerning the interstitial and terminal localization of heterochromatin in birds[11]. Such non-centromeric heterochromatin, however, has been reported to play an important role in intrapopulational variation in mammals[3]. Miklos and Nankivell[5] suggested that the presence of interstitial heterochromatin may function in the regulation of recombination. In view of all these facts a study of the distribution of heterochromatin is considered important. In