spectively in control, phlebotomized and hypertransfused groups. Compared to control group, the p-value was > 0.001 in both experimental groups. Also shown in figure 2 is the mean ($\pm$ SD) of hematocrits and reticulocyte counts obtained immediately before the animals were killed.

Histological examination of implants and long bones revealed erythroid hyperplasia with almost total absence of fat cells in phlebotomized group; whereas fat cells were present in an appreciable number in the control (figure 1) and hypertransfused groups, with clusters of erythroid cells being almost non-existant in the latter group.

These findings indicate that like other parts of marrow, the ectopic marrow nodules are responsive to erythropoietic modulation and must be considered as part of total erythropoietic mass. The appropriate response of these nodules to erythropoietic stimulation and suppression, makes it probable that they also partake in other functions of the marrow, including granulopoiesis and megakaryapoiesis.

Previous studies have indicated that the marrow tissue consists of 2 distinct elements: proliferating hemopoietic cells and its supporting stromal meshwork[4]. Radiobilogic and cytogenetic studies[5,6] in cross-transplanted animals, have suggested that these nodules are chimeric structures with the donor tissue providing the stromal network upon which circulating hemopoietic stem cells of recipient origin proliferate. Ectopic implants of marrow tissue, therefore, should be considered as transplants of marrow stroma rather than those of hemopoietic stem cell. Our present findings are in agreement with this concept suggesting that erythropoietic cells in these marrow nodules are a part of the recipient's total erythroid pool and should appropriately respond to factors modulating erythropoiesis.

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Enhancement of humoral immune response against human lung elastin peptides[1]

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Summary. When guinea pigs instead of rabbits were used as the host animals, 8–16 times higher antibody titers against human lung elastin peptides were produced with only 1/20 the amount of antigen per unit body weight. This corresponds to a 200-fold enhancement of the immune response.

Elastin is only weakly antigenic and the few reports of antibodies to this protein[2–4] or its soluble precursor[5,6] indicated that only low titers could be attained. Soluble breakdown products prepared by elastase[7–9] or alcoholic KOH[3,10] degradation of elastin from ligamentum nuchae[3,7,9], aorta[10] or lung[8] also showed weak immunogenicity in rabbits. Studies by Timpl and others[11] demonstrated that rabbits, though traditionally used for the production of most antibodies, are not always the host animals with the best response to a given antigen. In an attempt to enhance the antigenicity of human lung elastin peptides and to obtain higher titer antibodies as a probe for the determination of lung elastin degradation in vivo, the present study was undertaken to investigate the response of other potential host animals to these immunogens.

Materials and methods. Isolation and characterization of human lung elastin peptides have been described in detail elsewhere[8]. The different host species and the immunization procedures used are listed in table 1. Healthy New Zealand rabbits of both sexes, weighing 3–3.2 kg, female Hartley strain guinea-pigs weighing 300–350 g, female Sprague-Dawley rats weighing 250–300 g and female Swiss mice weighing 18–20 g were immunized s.c. or i.p. with complete or incomplete Freund's adjuvant. Sera were tested for the presence of precipitating antibodies by double diffusion in agar gel as described previously[8]. Titers were determined against human lung peptides using serial dilutions of the antibodies. To assess species and organ specificity the sera were also tested against peptides derived from lung elastin of different species and from human aorta elastin.

Results and discussion. Comparison of rabbits, guinea-pigs, rats and mice as potential host animals showed that with only 1/20 the amount of antigen per unit body weight, guinea-pigs produced 8–16 times higher anti-elastin peptide titers than rabbits, a 200-fold enhancement of the antibody titer (see table I). Rats and mice did not produce detectable antibodies under the same conditions or with a different

Table 1. Host species and immunization procedures

Animals used Species	Number	Weight (g)	mg injected per animal per injection	Adjuvant	Route	Total number of injections	Antibody titer
Rabbits	2	3000–3200	100	CFA	s.c.	3	1:2
Rabbits	2	3000–3200	100	IFA	s.c.	3	1:2
Guinea-pigs	6	300–350	5	CFA	s.c.	3	1:32
Guinea-pigs	6	300–350	0.5	CFA	s.c.	3	1:16–1:32
Rats	5	250–300	4	CFA	s.c.	3	No reaction
Rats	5	250–300	4	CFA	i.p.	3	No reaction
Mice	6	18–20	0.25	CFA	s.c.	3	No reaction

s.c., subcutaneous; i.p. = intraperitoneal; CFA = complete Freund's adjuvant; IFA = incomplete Freund's adjuvant.

Table 2. Reactivity of anti-human lung elastin peptide sera with enzymatic digests (20 mg/ml) of different elastins

Elastin source	Guinea-pig antiserum (1:8)	Rabbit antiserum (undiluted)
Human lung	4+	2+
Human aorta	4+	±
Rabbit lung	4+	–
Rabbit skin	+	–
Rat lung	4+	–
Dog lung	4+	–
Dog skin	+	–
Ox lung	4+	–
Sheep lung	4+	–

route of injection. These findings are consistent with the results of Plescia et al.[12] that rabbits produced antibodies to both DNA and methylated BSA, whereas mice did not produce antibodies against either. Similarly, different patterns of specificities in different animal species were described for collagen, another connective tissue protein. In this case rabbits, guinea-pigs, rats, mice and chickens elicited antibodies to different antigenic sites[11].

Anti-elastin peptide guinea-pig serum showed a faint precipitin reaction with 2.5 mg/ml immunogen, whereas the rabbit antiserum did not give a detectable reaction with 10 mg/ml dilution of immunogen. Substitution of the incomplete form of Freund's adjuvant did not increase the titer.

The guinea-pig antiserum did not react with any of the enzymes used in the preparation of the immunogen, i.e. trypsin, elastase or collagenase, at 10 mg/ml.

There are some apparent differences in specificity and crossreactivity between the guinea-pig and rabbit anti-elastin peptide serum, as shown in table 2. The increased cross-reactivity of the antibodies produced in guinea-pigs may be due to the higher antibody titer or to production by the rabbit of antibodies of lower titer but with higher specificity. Recently, the production of species-specific rabbit antibodies to canine lung elastin has been reported by Damiano et al.[13].

Although immuno-double diffusion against the human lung elastin peptides in the present study showed a reaction of identity between rabbit and guinea-pig antiserum, the possibility of a response against different antigenic sites cannot be completely ruled out. The guinea-pig antiserum-elastin peptide reaction produces a second precipitin line, which may arise from a different sequence. The association of antigenicity with more than one peptide sequence is also supported by the results obtained by the present authors in parallel experiments with elastin peptides from other species. Thus, peptides derived from adult sheep lung elastin elicited in rabbits antibodies which had a higher titer (1:8) than those obtained with the same amount of lung elastin peptides from humans (1:2). Unlike the anti-human rabbit sera but analogous to the anti-human guinea-pig sera, the anti-sheep rabbit sera cross-reacted with lung elastin peptides from other species, including man. Experiments designed to define the immunogenic peptide sequence(s) are in progress[14].

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Moult inhibition in the insect *Dysdercus cingulatus* (Insecta: Heteroptera) by the cerebral glands of the millipede *Jonespeltis splendidus* (Myriapoda, Diplopoda)

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Summary. Implantation of 2 pairs of cerebral glands of the diplopod *Jonespeltis* into the newly-moulted 4th or 5th instar nymphs of the insect *Dysdercus* delayed the moulting of these insects for a fairly long time. Implantation of cerebral glands into 1-day old 5th instars postponed the subsequent moult for a shorter period, whereas implantation of cerebral glands into 2-day old 5th instars had no effect. These observations suggested that a neurosecretory factor from the cerebral glands inhibited moulting in this insect, and in the case of 5th instar nymphs there was a critical period before which implanted glands were effective in moult inhibition.

Among myriapods, especially in chilopods, there is some experimental evidence that cerebral glands are the source of a moult-inhibiting principle[2,3]. Cerebral glands of the millipede *Jonespeltis splendidus* consist of swollen axonal terminations of neurosecretory cells of brain and visceral ganglia[4]. Earlier observations by the present author involving bilateral ablation of cerebral glands from well-tanned (intermoult) animals of *Jonespeltis* resulted in an acceleration of moulting which suggested that the cerebral glands are the source of a moult-inhibiting principle[5]. In the