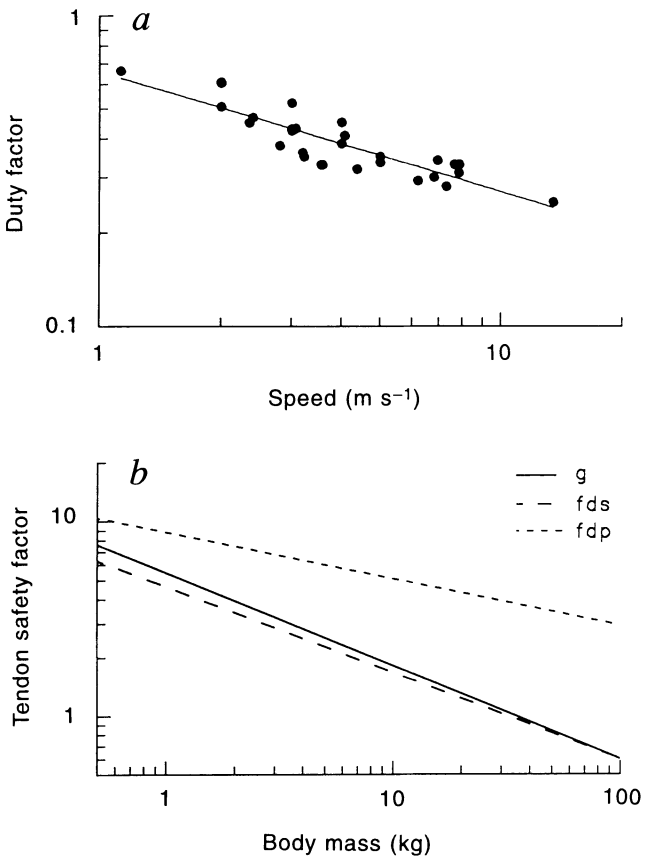

FIG. 3 Graphs on logarithmic coordinates of duty factor versus speed of hopping (*a*) and tendon safety factors versus body mass (*b*) in the Macropodoidea. Duty factors were determined from analysis of high-speed cine and video films of 8 species. Individual animals ranged from 1 kg to 50 kg body mass. Data were obtained from previous studies[3,4,14,17] and unpublished observations. Film footage included sequences of some species hopping at or close to their maximum speeds. Regression information is given in Table 1. Tendon safety factors (tendon strength/maximum stress to which the tendon is likely to be exposed) were calculated assuming a peak isometric muscle stress of 300 kPa. The ultimate tendon strength was taken to be 100 MPa[11]. Tendons with low safety factors make good 'springs' for hopping, but have a greater risk of failing under the applied loads. Least-squares regression line data are: $SF_{gastrocnemius} = 5.4M^{-0.49 \pm 0.05}$; $SF_{fds} = 4.6M^{-0.44 \pm 0.06}$; $SF_{fdp} = 5.4M^{-0.24 \pm 0.06}$.

(97 J s^{-1}) at 6.8 m s^{-1}. These are lowest estimates based on tendon forces being proportional to muscle-fibre areas[5]. Greater savings would be expected in larger, faster animals, and also if the pattern of muscle force sharing[15] was different.

A continuum exists from small macropods in which energy savings are trivial to large ones where tendon elasticity significantly reduces the cost of travel. However, 50–60 kg may be the optimum mass for this energy-saving system as larger animals risk breaking their tendons (Fig. 3). Extinct giant kangaroos of the Pleistocene (~150 kg)[16] probably used lower than predicted hopping speeds or may have scaled differently to modern species. In either case, the potential benefits of elastic energy storage would have been reduced, perhaps contributing to their eventual extinction. □

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Genetic polymorphism for alternative mating behaviour in lekking male ruff *Philomachus pugnax*

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ALTERNATIVE **male mating tactics are widespread among animal taxa[1–3], but there are few well documented examples of genetic polymorphisms for them[4–6]. The dimorphism in male courtship behaviour between independent and satellite ruffs, *Philomachus pugnax*[7,8] (a lekking sandpiper), has often been cited as a potential example but this has been questioned[9,10] because of the lack of data[11] and the widespread phenotypic plasticity in the development or expression of alternative tactics in other species[1–3,9,12–14]. By rearing ruffs in captivity, we now show that differential morph development is genetically controlled and consistent with a single-locus, two-allele autosomal genetic polymorphism. Several potentially relevant environmental factors do not appear to alter behavioural development.**

'Independent' male ruffs have a territorial breeding strategy of defending lek mating courts against other independents. Non-territorial 'satellites', representing about 16% of males[15] (D.B.L. and C.M.S., unpublished data), move among, are recruited to and share independents' courts[7,8,11]. Differences in mating strategy correlate with polymorphisms in the colours and patterns of elaborate male breeding plumage[7]. Plumage variation is similar within populations throughout the species' Palaearctic breeding range, indicating that behavioural variation is also ubiquitous. Males maintain a single plumage and behavioural phenotype throughout their adult lifetime. A single observation of short-term behavioural flexibility in the wild[8] is counterbalanced by a failure to report another case in many years of fieldwork by six research groups, including the original observer[7,11,16–19].

We obtained pedigree data for behavioural morph in two ways. In 1985, 1989 and 1990, we reared chicks from eggs collected from 43 ruff nests at three sites near Oulu, Finland. The paternity of the 1989 and 1990 chicks was determined by comparing a chick's DNA profile with that of its mother and of males captured at nearby leks (Fig. 1). From 1987 to 1993, we bred the ruffs we reared, and their offspring, in captivity, controlling paternity by housing females with a single male.

Table 1 presents pedigree data for behavioural phenotype by family. Of 27 sons of independent males, 25 were independents, whereas 14 of 30 satellite sons were satellites (corrected $\chi^2 =$

TABLE 1 Pedigree data on the behavioural phenotype of ruff sons, their fathers, and their mother's brothers, classified by their father's and their maternal grandfather's phenotype

Source*	Father	Mother	Mother's brother's morph(s)†‡	Morph(s) of son(s)†
Independent fathers × independent maternal grandfathers				
C	112	28	—	I, I
C	112	37	—	I
C	114	40	I	I, I
C	151	73	—	S
Independent fathers × unknown maternal grandfathers				
C§	G1M	G1F	—	I
W	B&W	K5	—	I
W	B&W	K10	—	I
W	SS	L11	—	I
W	LO	L13	—	I
W	LO	L15	—	I, I
C	112	03	I	I
C	112	05	I	I
C	112	11	I	I
C	112	18	I	I, I
C	112	U1	—	I
C	114	07	I, I	I
C	6247	11	I	I
C	6247	U2	—	I
C	6247	U3‖	—	I, I, I
Independent fathers × satellite maternal grandfathers				
C	112	67	—	S
C	113	24	I	I, I
C	114	62	I	I
Satellite father × independent maternal grandfathers				
C	111	59	I, I	S, I
C	111	53	—	S
Satellite fathers × unknown maternal grandfathers				
C§	G2M	G2F	—	S
W	BT	K19	—	I
W	BT	K22	—	I
W	TH	K3	—	I
W	BU	L5	—	I
W	BU	L16	—	I
W	BF	L16	—	I
C	111	11	I	I
C	111	17	I	S, S, I, I, I
C	111	61	—	S
C	158	6174	—	S, I
Satellite fathers × satellite maternal grandfathers				
C	111	22	S, S, I, I, I	S
C☚	111	71	S	S
C	111	6233	—	S, I, I
C	118	24	I	I
C	158	22	S, S, I, I, I	S
C	158	24	I	S, S, I
C	158	510	S, I	S

Chicks were reared in 'common garden' social groups that mixed individuals from different broods, with no adults present through the first year (1985 and 1989 cohorts) or the first 2 months of life (other years). Males of both morphs developed, even within the two cohorts raised in the absence of adults. We assessed the behavioural phenotypes of males by observing male–male interactions in mixed-morph groups during breeding seasons. The relationship between plumage and behavioural phenotype seen in nature[7] also developed in captivity. No birds changed morphs among years. We biased breeding assignments towards positive assortative mating to enhance our probability of detecting genetic differences, using expectations about a female's potential geneotype based on the phenotype(s) of her male relatives, where known.

* W, DNA paternity analysis of wild-bred chicks reared in captivity (Fig. 1); C, bred in captive flock from birds previously raised.

† I, Independent; S, satellite.

‡ Brothers of females with unknown grandfathers may be half or full sibs.

§ Published data[15].

‖ 1–3 females' offspring.

☚ Pedigree class assigned from a satellite brother, which implies a satellite grandparent under a dominant satellite allele model (inclusion with 'unknown grandfather' does not change the outcome of the analyses).

Note, Birds 22, 24, 118 and 510 are offspring of 111; 118 is a son of 17; 28, 37 and 53 are daughters of 114; 59 and 73 are daughters of LO; 40 is a daughter of 112; 6233 is a daughter of TH; 67 is a daughter of BU; 62 is a daughter of BF, and 510 is a daughter of 59.

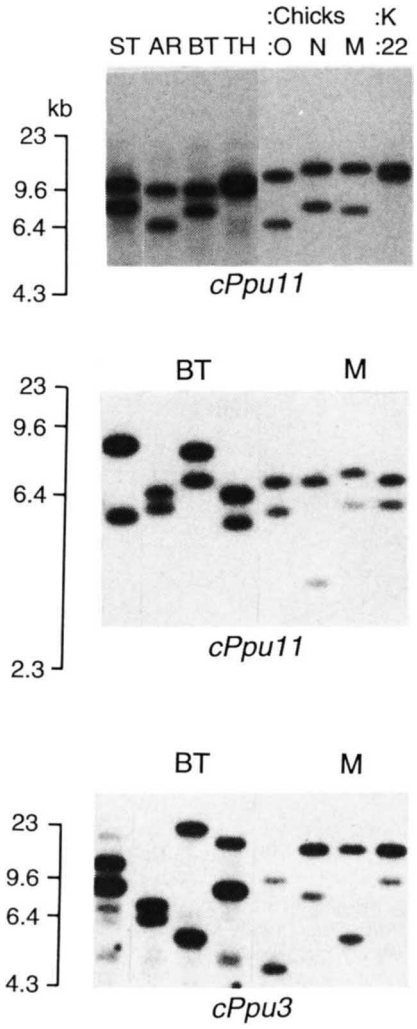

FIG. 1 Example of paternity assignment of a wild-caught chick using single-locus minisatellite probes. The paternal alleles of chick M, from female K22's nest, are only present in satellite father BT. Five single-locus minisatellite probes (c*Ppu*MS3, c*Ppu*MS4, c*Ppu*MS6, c*Ppu*MS8, c*Ppu*MS11) were isolated from a ruff size-selected genomic DNA library following a standard procedure[27,28]. The minisatellite loci were characterized by a large number of alleles (12.6 ± 1.82 s.d.) in our population. The probes were hybridized to a Southern blot of total *Mbo*I-restricted genomic DNA from the chicks, their biological mothers and potential fathers. Males were run on the same gel (chicks from females K3, K5, K10, K19, K22) or on a different gel (chicks from females L5, L11, L15, L16) from the chicks. In the latter case, an internal size marker mix was added to each digested DNA sample[29]. At least four probes were used for paternity assignments. Paternities were assigned only when an exact size matching of the paternal allele for all probes was achieved. Assuming an equal allele frequency at each locus, the paternal false inclusion probability was estimated to be $<5.4 \times 10^{-4}$ for each combination of four probes used (ref. 30, and D.B.L. *et al.*, manuscript in preparation).

29.87, 1 d.f., $P = 0.002$), indicating a strong effect of paternal inheritance on offspring phenotype. We tested observed offspring morph ratios against the predictions from four single-locus, two-allele genetic models with complete dominance, two each for autosomal and sex-linked (Z chromosome) genetic inheritance, assuming Hardy–Weinberg equilibrium in the grandparent generation (Table 2). Because females do not express the behavioural phenotype, we classified families by father's and maternal grandfather's phenotypes, including unknown grandparent cases, producing six pedigree classes (Tables 1, 2*a*). Offspring morph ratios were heterogeneous among classes (ANOVA of offspring ratios per male, weighted

TABLE 2 'Goodness of fit' tests of autosomal models for the inheritance of male mating strategies

(a) Morphs produced under satellite (Ss) and independent (Ii) complete dominance autosomal models

Pedigree*			Proportion of independent male offspring						
Father	MGF†	n‡	Expected (*Ss*)	Expected (*Ii*)	Observed	*P*(*Ss*)	Combined	*P*(*Ii*)	Combined
I	I	6	0.96	0.92	0.83	0.23		0.46	
I	U	19	0.92	0.89	1.00	0.40		0.16	
I	S	4	0.70	0.80	0.75	1.00	0.46	0.59	0.74
S	I+U§	19	0.44	0.61	0.62	0.11		1.00	
S	S	11	0.33	0.30	0.36	0.76		0.74	

(b) Heterozygote phenotype under both models

			Proportion of crosses producing both phenotypes			Goodness of fit	
Phenotype of father	Genotype	Expected frequencies	Expected	Observed	n‖	*P*	Combined *P*
			Satellite dominance model				
Independent	*ss*	0.84	0.08	0.00	5	1.00	0.49
Satellite	*Ss*	0.15	0.67	1.00	5	0.18	
Satellite	*SS*	0.01					
			Independent dominance model				
Independent	*II*	0.36	0.15	0.00	5	1.00	0.03
Independent	*Ii*	0.48					
Satellite	*ii*	0.16	0.35	1.00	5	0.01	

a, We compared the observed proportions of independent male offspring produced by residents and satellites crossed with three classes of maternal grandfather, including unknown morph, against expected values generated under each model. Expected allelic and genotypic frequencies in the grandparent generation were calculated from phenotypic frequencies of 0.84 independents and 0.16 satellites observed in the wild, assuming Hardy–Weinberg equilibrium, producing $p(S)=0.08$, $q(s)=0.92$ for the satellite dominance model, and $p(I)=0.60$, $q(i)=0.40$, for the independent dominance model, producing the expected genotype frequencies shown in *b*. Assortative mating would be one source of departure from Hardy–Weinberg equilibrium. Because the eggs of individual females are often fertilized by males of both morphs in the wild, this does not strongly occur (D.B.L. *et al.* unpublished data). The goodness of fit was tested using 2-tailed binomial probabilities, and an overall probability was calculated using Fisher's method for combining probabilities, d.f. = 4. Neither model is rejected for any single pedigree class (2-tailed binomial test), nor for the data set as a whole. Because different numbers of offspring per parent were used in this analysis, the genotype ratios within the dominant allelic class may not be representative of the population as a whole, and the probability of mistakenly rejecting a valid model increases. Our failure to reject either model despite this risk is thus conservative. *b*, We tested for the heterozygosity of independent versus satellite males by comparing the proportion of families with multiple male offspring of both morph types (Table 1) against expected proportions. The expected values were calculated under Hardy–Weinberg equilibrium and incorporated genotype-specific probabilities for producing mixed families given the number of males produced. Because we attempted to assort captive matings positively, which would bias against the production of mixed-morph families, the probabilities calculated for rejection of both models are conservative. The independent dominance model is rejected, because of a poor fit for satellites, using Fisher's method of combining probabilities, d.f. = 1.

*I, Independent; S, satellite; U, unknown.

† Maternal grandfather.

‡ *n*, Number of offspring per cross.

§ Because data from only three offspring were obtained from the satellite × independent pedigree (Table 1), and the expected morph ratios were most similar to those from pedigrees of unknown grandfathers, these two classes were combined and tested against sample-size weighted expected values.

‖ *n*, Number of families.

by number of offspring: $n=24$, $F=3.68$, 5 d.f., $P=0.018$), further demonstrating the effect of pedigree.

Neither sex-linked complete dominance model fits the data. Male birds are homogametic (ZZ), so neither model allows for the production of opposite phenotype sons from both matched-morph father × maternal grandfather pedigrees, as occurs in Table 1. Rejection of the sex-linked satellite dominance model relies on an assignment of paternal pedigree for female 73 based on DNA fingerprinting (Fig. 1)

TABLE 3 Gompertz growth parameters[26] and body mass of independent and satellite males as chicks

	Independents			Satellites				
Variable	mean	s.e.	*n*	mean	s.e.	*n*	*F*	*P*
K	0.234	0.004	59	0.245	0.009	17	1.57	0.22
t	10.3	0.2	59	9.5	0.4	17	3.40	0.07
a	142.5	1.8	59	134.2	4.0	17	3.62	0.06
Adult mass	167.3	1.9	57	155.9	4.7	14	5.14	0.003

Satellites are smaller, but growth parameters (*K* and *t*) do not differ as expected if poorly growing chicks develop into satellites. Mass of growing chicks was measured on alternate days through to day 26. *K*, Maximal slope of growth curve (1/day); *t*, inflection point of growth curve (days); *a*, asymptotic body mass at the end of chick growth (g); adult mass is the mass or mean mass measured during mid-winter(s) as adults (g). Variable values are least-square means from ANCOVA models controlling for significant differences in cohort growth rate; *F* and *P* values test for differences between phenotypes (1 d.f.).

Observed offspring morph ratios produced by each pedigree class fit expectations from both autosomal dominance models (Table 2*a*). We may discriminate between the two by specifically testing their predictions about the frequencies and phenotypes of heterozygotes. The satellite allele dominance model predicts that 15% of all males will be heterozygotes and satellites, whereas the independent dominance model predicts that 48% of males will be heterozygotes and independents (Table 2). Only crosses in which at least one parent is a heterozygote can produce mixed broods. Among families that produced multiple sons, all five satellite crosses produced both morphs, whereas all five independent crosses produced only independents (Table 1). The independent dominance model is rejected by this outcome (Table 2*b*), given the probabilities of mating with heterozygous females, assuming Hardy–Weinberg equilibrium, and the family sizes involved. More complex modes of inheritance cannot yet be adequately tested, but the satellite dominance model appears to account for the inheritance of behavioural phenotype.

Because our results pertain strictly to male development under captive conditions, natural environmental variation, eliminated in our study, could significantly affect phenotype development or expression[1,3,8,14]. Captive rearing did not drastically bias development, because 11% (5 of 45) of the males reared from eggs collected in the wild became satellites, similar to the proportion in the source population (16%). We eliminated common

environmental effects as a cause of father–son phenotypic similarity by rearing young in groups (Table 1).

A slightly smaller mean body size of wild satellites has been interpreted as suggesting that poorly growing chicks disproportionately develop into satellites[10]. Small body size differences between morphs developed among our captives, despite *ad libitum*. food availability and benign environmental conditions (Table 3). However, growth parameters do not indicate that satellites grew more poorly than residents (Table 3). Rather than reflecting conditional development, the small difference in mean body size may be a correlated adaptation to the demands of each reproductive strategy. Independent males fight to establish courts and remain on them throughout the day[18], both of which may favour larger size through increased social dominance and energy storage capacity. In contrast, satellites do not fight for courts, are freer to forage throughout the day, and spend more time flying, reducing the need for, and raising the physiological cost of, maintaining larger body size.

Theoretical and empirical investigations of behavioural development, social behaviour and life history of ruffs are henceforth justified in using a genetic equilibrium model for generating predictions against which to test field observations[15,20–22]. Our findings highlight the question of what selective conditions favour genetic polymorphism controlling morph development in this species[14], rather than quantitative inheritance[23] or nearly complete environmental control[24,25], as occurs in nearly all other species with alternative male mating tactics[1–3,9,12,13]. □

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Cell fate in the *Arabidopsis* root meristem determined by directional signalling

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POSTEMBRYONIC development in plants is achieved by apical meristems. Surgical studies and clonal analysis have revealed indirectly that cells in shoot meristems have no predictable destiny[1–3] and that position is likely to play a role in the acquisition of cell identity[4–7]. In contrast to animal systems[8–10], there has been no direct evidence for inductive signalling in plants until now. Here we present evidence for such signalling using laser ablation of cells in the root meristem of *Arabidopsis thaliana*. Although these cells show rigid clonal relationships[11], we now demonstrate that it is positional control that is most important in the determination of cell fate. Positional signals can be perpetuated from more mature to initial cells to guide the pattern of meristem cell differentiation. This offers an alternative to the general opinion that meristems are the source of patterning information[12].

The *Arabidopsis thaliana* root consists of single layers of epidermis, cortex, endodermis and pericycle, surrounding a vascular bundle[13]. The root meristem is derived from basal cells of the embryo proper that forms the initials for the different cell types present in the mature root, and from the hypophysis which generates the columella root cap and quiescent centre[11] (Fig. 1). The consistency in cell fate and position suggests that cells are committed to form particular tissue types. This is supported by two β-glucuronidase promoter fusions specific for vascular and root cap cells, which are also expressed in the meristematic initial cells (Fig. 2*a*, *b*).

To determine whether root meristem cells behave according to their clonal origin or to their position, we applied laser ablation. Dead cells are compressed towards the periphery of the root (Fig. 3*a*), allowing neighbouring cells to invade their position. By recording the fate of the invading cell, the contribution of lineage or position can be investigated.

Upon ablation of all quiescent centre cells, the dead cells become flattened and are displaced towards the root tip. Cells of the proximal vascular bundle occupy the former position of the dead cells. These cells no longer express the vascular marker (Fig. 2*c*) but instead express the root cap marker (Fig. 2*d*). Hence the clonal boundary set by the first zygotic division, separating future vascular and root cap cells, does not restrict developmental potential. Furthermore, information guiding cell fate along the apical–basal axis in the root tip is permanently present. These results confirm earlier regeneration studies[14–17] but also show that global redifferentiation is not necessary for cell-fate switching.

We investigated whether positional information can determine cell fate in the radial plane. Cortical initials divide asymmetrically to produce cortex/endodermis cells (Fig. 3*a*). Upon ablation of these initials, pericycle cells invade and divide periclinally. This maintains the pericycle cell file and generates new cells in the cortical cell file (Figs 3*a*, 4*b*). Pericycle cells are smaller than cortical cells, so more than one pericycle cell invades, thus enlarging the typical number of eight cortical cells. The former pericycle cells subsequently divide asymmetrically (Figs 3*b*, 4*b*), generating cortical and endodermal cell files. Suberin staining revealed a casparian strip in all prospective endodermal cells, illustrating their differentiation to endodermal fate (Fig. 3*c*). Therefore, pericycle cells switch fate when moved over radial clonal boundaries.