The grebe family is an ancient group, with no close relatives among living birds. As far as we know, all grebes have asymmetrically lobed toes and the mode of swimming described here is probably optimally suited to this morphology.

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Table 1 Frequency of rare microsatellite variants in the offspring of wheat plants

Type of variant	Frequency*		Ratio to control	P†	Wilcoxon test	
	Control	Exposed			z	P‡
Nulls (homozygotes)	0.0056	0.0066	1.17	0.8334	0.42	0.6754
Losses (heterozygotes)	0.0010	0.0066	6.45	**0.0054**	2.57	**0.0100**
Losses (homozygotes)	0.0015	0.0029	1.88	0.5461	0.74	0.4606
Gains (heterozygotes)	0.0031	0.0087	2.82	**0.0270**	1.84	0.0654
Gains (homozygotes)	0.0015	0.0021	1.34	0.9722	0.41	0.6831
Heterozygotes losses + gains	0.0041	0.0153	3.73	**0.0003**	3.19	**0.0014**
Homozygotes losses + gains	0.0031	0.0050	1.61	0.4686	0.53	0.5930

We screened 186 and 150 wheat plants grown from seeds collected from exposed and control plants, respectively (further details are available from the authors). Assuming selective neutrality, the equilibrium frequency of heterozygous variants, H_e, was approximated as $H_e = 4N_e u_e/(1+4N_e u_e)$, where N_e and u_e are effective population size and spontaneous mutation rate, respectively[9]. If $N_e = 1$ for self-fertilized plants, then $u_e \approx H_e/4$. Homozygosity in the exposed group, f_t, was approximated as $f_t = [(1/2N_e) + (1 - (1/2N_e))] \times (1 - u_e)^2$, where f_{t-1} and u_e are the homozygosity of the previous generation and mutation rate in the exposed population, respectively[9]. If $f_{t-1} = 1 - H_e$, then $u_e \sim (H_e - H_e/2)/2$.
*Frequency was estimated as the number of variants per microsatellite locus.
†Probability of difference from the control group (Fisher's exact test, two-tailed; statistically significant values are in bold).
‡Probability of Wilcoxon test (statistically significant values are in bold).

Germline DNA

Wheat mutation rate after Chernobyl

The accident at the Chernobyl nuclear power plant in 1986 has generated concern over the genetic consequences of chronic exposure to radiation. Here we describe a new approach to monitoring germline mutation in plants and find evidence for a remarkably strong induction of germline mutation in wheat upon chronic exposure to ionizing radiation produced by the Chernobyl accident.

We compared wheat plants descended from two genetically identical populations, derived from the same homogeneous parental line. One population was grown for one generation (10 months) in a heavily contaminated plot (900 Ci km^{-2}) near the Chernobyl nuclear power plant[1], the other was sown in a clean (< 1 Ci km^{-2}) control area 30 km away in soil with comparable agrochemical characteristics.

Using the polymerase chain reaction, we profiled offspring plants for 13 single-copy monomorphic microsatellite loci[2]. Evidence for alterations (variants) was obtained for all 13 loci, including gains and losses of repeats, as well as complete loss of microsatellite bands (nulls) (Table 1). Offspring derived from exposed plants showed no increase in the frequency of homozygous variants and a threefold increase in the frequency of heterozygous structural variants, attributed to all loci within this group of plants.

Differences between the two initially identical populations, presumably arising over one generation, may result from seed contamination, migration or mutation. Seed contamination is unlikely, however, because neither plot had previously contained wheat. Seed contamination would also have affected the frequency of all three types of variant, including nulls and other homozygous variants. Cross-pollination can also be excluded as wheat is an obligatory self-pollinator, preventing the migration of pollen between neighbouring populations. We conclude, therefore, that the increased diversity in the heterozygous variants in the offspring is probably due to a radiation-induced increase in microsatellite mutation in the exposed plants.

Assuming that the control population is in equilibrium (Table 1), we estimate that the spontaneous mutation rate is 1.03×10^{-3} per locus (95% confidence interval, 0.44×10^{-3}–2.03×10^{-3}), whereas mutation rate in the exposed group was 6.63×10^{-3} (95% confidence interval, 4.28×10^{-3}–9.70×10^{-3}). Thus we attribute the more than threefold increase in heterozygosity in the exposed group to a more than sixfold increase in the mutation rate over the single generation of exposure to ionizing radiation.

We estimate that the wheat plants have been exposed to relatively low doses of chronic irradiation of about 0.3 Gy, with external and internal components of 0.2 and 0.1 Gy, respectively[3]. Theoretically, this low-level exposure should not cause such a large increase in the mutation rate, suggesting that chronic exposure to ionizing radiation has effects that are as yet unknown. Other studies have shown that chronic internal exposure is far more efficient at inducing somatic recombination than acute external exposure[1,4].

The estimated increase in mutation rate in the offspring of exposed plants is too high to be due to direct targeting of microsatellite loci by ionizing radiation. The wheat genome contains 16×10^9 base pairs[5], and the mean size of the 13 microsatellite loci included in this study is about 100 base pairs. Attributing a sixfold increase in the mutation rate to direct radiation-induced DNA damage at the microsatellite loci would mean that the expected damage to the whole wheat genome was (increase of mutation rate) × (genome size)/(size of locus) ≈ 80,000 damaging events — much higher than any experimentally derived measurements of the initial yield of radiation-induced damage to a eukaryotic cell[6]. The increase in microsatellite mutation rate in plants may therefore be better explained by a non-targeted effect of ionizing radiation elsewhere in the cell, as described for mammalian minisatellite loci[7,8].

Our findings raise the important issue of the genetic hazard of chronic radiation exposure to the germ line, showing that the apparent rate of induced microsatellite germline mutation is much higher than existing estimates of absorbed doses of exposure would predict. Further study is needed to analyse the genetic effects of chronic radiation exposure.

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Metabolic scaling

Energy constraints on carnivore diet

The energy expenditure of mammals reflects their habits and environments[1], subject to limitations associated with body size. Carbone *et al.*[2] combined scaling relationships to argue that large species of the mammalian order Carnivora (weighing more than 21.5 kg) do not specialize on invertebrate prey. However, many tropical mammals that feed exclusively on ants and termites are much heavier than this, often weighing up to 60–70 kg; they survive by progressively reducing their metabolic rate to below that expected from their body size. I believe that this response indicates that it is not body size that limits the determination of diet, but rather the maximal rate of energy expenditure.

The size limit for a predator exclusively dependent on invertebrate prey is not absolute. For example, the sloth bear (*Ursus ursinus*), a carnivore that can weigh as much as 145 kg and feeds extensively (but not exclusively[3]) on termites, was considered by Carbone *et al.* to be an outlier — but outliers should not be ignored as they may tell us that our theories are incomplete. Their analysis[2] fails to recognize that all scaling relationships contain biologically relevant variation, and inherent in this residual scatter are adjustments that permit a large mass in carnivores and other terrestrial mammals that consume invertebrate prey.

Large mammals (over 20 kg) that specialize in eating tropical ants and termites include the aardvark (*Orycteropus afer*) and some pangolins (*Manis temmincki* and *M. gigantea*), tamanduas (*Myrmecophaga tridactyla*) and armadillos (*Priodontes maxima*). These[4,5] and the sloth bear[6] generally have lower standard rates of energy expenditure than other mammals. As ant and termite predators increase in size, their basal rate of metabolism decreases (Fig. 1), a trend that is particularly evident when species in a family are compared (to correct for any putative effect of phylogeny or ecological/behavioural uniformity).

A reduction in metabolic rate reduces the effective body size, which can be estimated from the total basal rate of the largest committed ant/termite eaters. If an all-mammal standard[7] for basal metabolic rate is used, a 15.9-kg *Manis crassicaudata* has the basal rate of a 3.4-kg standard mammal, a 30.6-kg *Myrmecophaga* has that of a 10.9-kg standard mammal, a 45.2-kg *Priodontes* has that of a 13.4-kg standard mammal, a 48-kg *Orycteropus* has that of a 36.2-kg standard mammal, and a 67.0-kg *U. ursinus* has that of a 56.5-kg standard mammal.

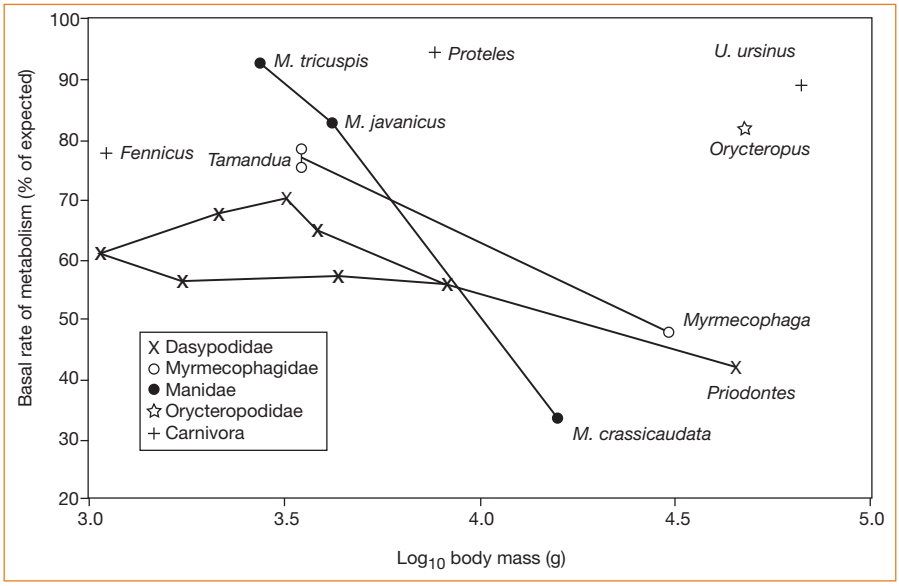

Figure 1 Basal rate of metabolism, expressed as a percentage of the basal rate expected from an all-mammal curve[7], in various mammals[4–6] that specialize on soil invertebrates, as a function of body mass. Species that belong to the same family are connected.

These calculations indicate that the maximum body mass in a standard mammal compatible with an ant/termite-eating habit is 11–13 kg, with the exception of the aardvark and sloth bear. This calculation may account for the comparatively high basal rate in *Proteles* (Fig. 1), which weighs less than 10 kg — at that mass, an adjustment of basal rate may not be required. What seems to be limited is the total rate of energy expenditure, not body mass: a limiting rate may be encountered in various masses at the expense of conforming to a standard curve and having effective endothermic temperature regulation.

Two of the species shown in Fig. 1 exceed the 11–13-kg limit to the 'adjusted' mass. The large mass and comparatively high basal rate of the sloth bear correlate with a diet that is about 50% fruit[3], although it is not clear whether addition of fruit to the diet permits a higher expenditure or size. The most distinctive large terrestrial specialist insectivore is the aardvark, which conforms neither to the original analysis[2], nor to the evasion described here. How it can have its comparatively high basal rate and a large body mass, and eat only ants and termites, is unknown. Under the assumption that a limiting energy expenditure exists, some other evasion may apply.

A limit to the exclusive use of invertebrates by terrestrial mammals, if one exists, may be associated with the cost of prey collection, which is why the largest species are tropical and feed on ants and termites: only these prey occur in sufficiently large colonies to make prey acquisition energetically feasible, and such large colonies occur only in the lowland tropics. In the absence of colonial ants and termites, terrestrial invertebrate-eaters might attain a maximal mass of 10 kg (ref. 5). The absence of an ant/termite specialization in large carnivores may occur because this niche was occupied by other mammals before the evolution or arrival of carnivores, the only opportunity available being at intermediate masses, which was exploited in Africa by the aardwolf and the bat-eared fox (*Otocyon megalotis*).

Although it might be argued that this analysis fails to take phylogenetic history into consideration, it has been pointed out[8] that 'corrections' for proposed phylogeny erroneously assume the priority of phylogeny as a factor influencing phenotypic characters, thus ignoring the complex interactions among determinative factors. The model of Carbone *et al.*[2] is ultimately called into question because it ignores the residual variation and therefore the biological flexibility inherent in all scaling functions.

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