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LIFE HISTORY AND BIOECONOMY OF THE HOUSE MOUSE

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I. INTRODUCTION

Every organism is the outcome of a complex series of interactions within and between ontogeny and phylogeny. The result is an 'oeconomia naturae' for every individual, population and ecosystem, a term used by Linnaeus in an essay published in 1749, called by Egerton (1973) 'the first sketch of a science of ecology'. Calow (1984) has developed the same concept as the 'economics of ontogeny', involving 'the metabolic allocation of limited resources between often-conflicting processes and structures'. This paper is a description of these interactions in the house mouse, *Mus domesticus* Schwarz & Schwarz (The common attribution of the name *Mus domesticus* to Linnaeus or to Rutty is incorrect, q.v. International Commission on Zoological Nomenclature, 1990). We have undertaken it to provide a general framework, and also to sharpen the precision of the house mouse as a model for the human condition. We believe that there are few, if any, other multicellular species analysable in this way.

Although there have been many reviews of house mouse biology from different points of view (Keeler, 1931; Snell, 1941; Grüneberg, 1943, 1952; Green, 1966; Crowcroft, 1966; Berry, 1970, 1981*b*; Lindzey & Thiessen, 1970; Simmons & Brick, 1970; Theiler, 1972; Klein, 1975, 1986; Cooke, 1977; Morse, 1978; Altmann & Katz, 1979; Green, 1981; Foster, Small & Fox, 1981, 1982, 1983; Potter, Nadeau & Cancro, 1986; Brain, Mainardi & Parmigiani, 1989; Lyon & Searle, 1989; Berry & Corti, 1990), this is the first major effort to knit together the main features of the species' niche and life history. We are conscious of the limitations of this attempt, but offer it as a skeleton for clothing, as a stimulus for research and synthesis.

Our aim is to describe the features of house mouse biology which contribute towards the animal's worldwide success, and the factors which influence that biology. It is easy to be glib about this. As R. A. Fisher (1930) pointed out

> There is something like a relic of creationist philosophy in arguing from the observation, let us say, that a cod spawns a million eggs, that *therefore* its offspring are subject to Natural Selection; and it has the disadvantage of excluding fecundity from the class of characteristics of which we may attempt to appreciate the aptitude. It would be instructive to know not only by which physiological mechanism a just apportionment is made between the nutriment devoted to the gonads and that devoted to the rest of the parental organism, but also what circumstances in the life history and environment would render profitable the diversion of a greater or lesser share of the available resources to reproduction.

Caswell (1989) has shown how this argument has led to an enormous interest in life-history strategies, and in particular the recognition that compromises (variously called 'trade-offs', 'bet-hedging' or 'risk-aversion') frequently result from interaction between different parts of the life cycle. He concludes, 'Life-history theory has from its beginnings faced some of the most difficult problems in evolutionary biology: the definition of fitness, interaction of traits and constraints on evolution.' It is frustrating that Fleming (1979), in an excellent review of life-history strategies in small mammals, does not even mention house mice. It means that this paper is necessarily a pioneering and hence somewhat tentative introduction; we discuss mouse life history in the traditional meaning of that term, and approach life-history strategy through consideration of the factors with mould or modify it.

II. HOUSE MOUSE SPECIES

The epithet musculus ('little mouse') was originally used by Pliny (A.D. 23–79) to distinguish house mice from rats. Linnaeus formalized the name *Mus musculus*, presumably basing his formal description of the species on specimens caught in his home town of Uppsala. Subsequent taxonomists indulged in a riot of splitting until Schwarz & Schwarz (1943) consolidated 133 named forms into 15 sub-species within a single species, *Mus musculus*. However, this proved to be over-enthusiastic simplification. In particular the white-bellied nominate species (*M. m. musculus*) meets the dark-bellied western European form (*M. m. domesticus*) in an apparently stable hybrid zone in Europe (Ursin, 1952; Selander, Hunt & Yang, 1969; Nance *et al.*, 1990); because of this, the two subspecies were raised to the status of semi-species and then full species (*M. musculus* and *M. domesticus* respectively) (Marshall & Sage, 1981; Corbet, 1988; International Commission on Zoological Nomenclature, 1990). This revision is supported by the high incidence of male sterility which occurs in crosses between *M. musculus* females and *M. domesticus* males (Forejt & Ivanyi, 1975; Avner *et al.*, 1988), and by the allozymic and mitochondrial DNA differences between the two forms (Thaler, Bonhomme & Britton-Davidian, 1981; Ferris, *et al.*, 1983*a*, *b*; Boursot *et al.*, 1984; Britton-Davidian, 1990; Sage *et al.*, 1990; She *et al.*, 1990). Hybrids from the meeting zone of *musculus* and *domesticus* in Southern Germany have much higher nematode and cestode infestations than either of the 'pure' forms, suggesting a breakdown of disease resistance on crossing (Sage *et al.*, 1986). Biogeographic and allozymic studies show that there are at least three other species in Europe [*M. spretus* in Iberia, *M. spretoides* = *M. abbotti* in the Balkans and *M. hortulanus* or *M. spicilegus*

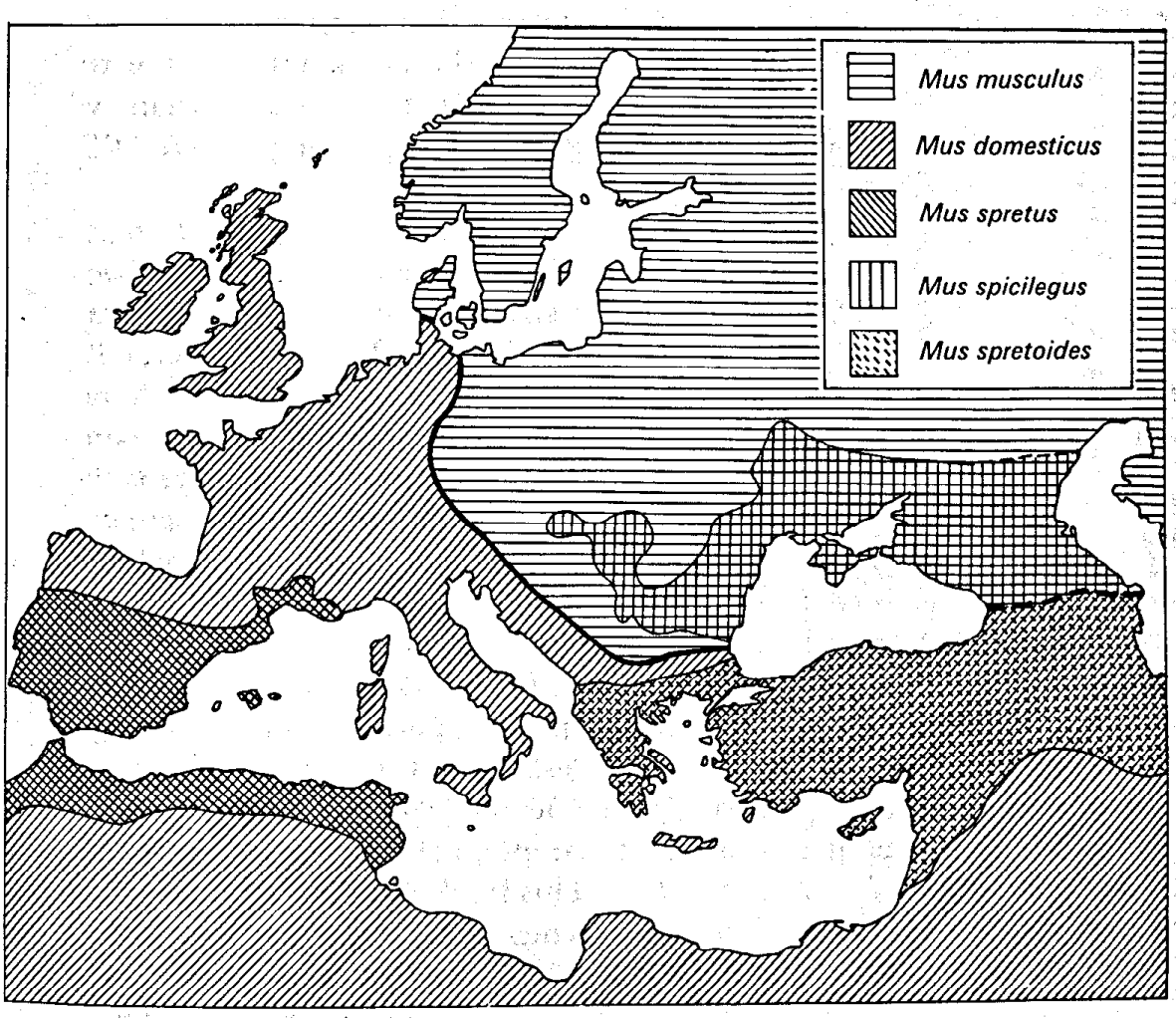

Fig. 1. Distribution of the five main *Mus* species in Europe. The species which has spread to most parts of the world as a commensal and domesticate is *M. domesticus* (after Marshall & Sage, 1981; Auffray *et al.*, 1990).

in Hungary, Yugoslavia and Western Russia: Bonhomme *et al.*, 1978; Marshall, 1986; Gerasimov *et al.*, 1990 (Fig. 1)]. In Asia, there are another two related species (*M. molossinus* and *M. castaneus*), and at least 14 more distant forms (Marshall, 1977).

Bonhomme & Guénet (1989) recognize four main groups of *Mus musculus* in Eurasia (*domesticus*, *musculus*, *castaneus* and *bactrianus*; they regard *domesticus* and *musculus* as subspecies only), although they emphasize that introgression can occur whenever different groups meet and the 'evolutionary fate (of the whole) is unpredictable'. Schwarz & Schwarz (1943) believed that the ancestral house mouse species spread with neolithic cultivation from the steppes of the Caucasus and Turkey. There are fossils of *M. domesticus* from the Achuelan period (*c.* 80000 years BP) in Israel (Tchernov, 1968), while Hesse (1979) has described specimens from a pre-agricultural site in Iran of neolithic date (7–8000 years B.C.). Auffray, Vanlerberghe & Britton-Davidian (1990) have catalogued the first appearance of mice in archaeological sites in Europe and western Asia (see also Thaler, 1986). Sage (1981) has suggested that their agricultural and commensal spread was due to a fortuitous preadaptation to life in rock crevices. The genetic distance between *M. domesticus* and *M. musculus* implies that their current contact zone arose through two already differentiated groups extending their range;

Bonhomme (1986) argues that they migrated to their present ranges by southern and northern routes respectively. However, there can be no doubt that the wild house mouse of the Americas and Australasia is largely *M. domesticus*, perhaps with some introgression from other species (Schwarz, 1945; Blank, Campbell & D'Eustachio, 1986).

Although Hooke, Priestly, Lavoisier and probably Mendel used mice in their research (Iltis, 1932; Morse, 1981; Berry, 1984), the modern use of the house mouse in science began in 1907 when C. C. Little started to study the inheritance of coat colour under the supervision of W. E. Castle at Harvard. Two years later, he set up the first inbred strain of mice (DBA) (Morse, 1978). There are now several hundred conventional, highly inbred strains, together with many segregating inbred strains, recombinant inbred lines, etc. (Lyon & Searle, 1989). The relevant point here is that a great variety of traits – morphological, physiological, pharmacological, biochemical – can be fixed in different strains and shown to be inherited. Randomly caught wild mice have a mean heterozygosity of 9 % per locus, which is greater than most small mammals (Berry, 1986*a*), and this variation is useable to study the influence of genetic factors. Furthermore, *c.* 1400 genes have been mapped on the 20 chromosomes of the mouse genome, which gives a vast (and largely unexploited) potential for examining the effect of particular chromosome segments or gene associations (Searle, 1981; Russell, 1985; McKusick & Roderick, 1987; Green, 1989; Berry, 1989*a*; Nadeau, 1989).

Much of the biological literature gives the impression that the mouse is scientifically significant only when in laboratory culture. This is false. Laboratory investigations can be calibrated, as it were, by studies of wild-living mice. Conversely, laboratory-derived data can be used to further the understanding of wild mice (Bronson, 1979; Berry, 1981*a*, *b*; Sage 1981; Potter, Nadeau & Cancro, 1986). This is a major change of emphasis from that of Grüneberg (1952), who in his definitive review wrote (albeit inaccurately, even for the time): 'The only major piece of research carried out on wild mice involves animals caught in the neighbourhood of Peking...' The rest of this paper is concerned with reciprocal syntheses of laboratory and field data on the species. The most complete review of data from laboratory mice is by Lyon & Searle (1989); the most recent of field data is by Berry & Corti (1990).

III. STAGES OF LIFE

Superficially, the life-cycle of a mammal such as a mouse is apparently uncomplicated and largely independent of environmental influences, but close analysis reveals a series of epigenetic and phenotype–environment interactions involving a cascade of physiological and behavioural compromises or trade-offs. To illuminate these, we have divided the life of an individual into the seven traditional (albeit arbitrary) stages, following William Shakespeare (*As You Like It*, Act 2, Scene 7) (Table 1).

(1) *Up to birth*

At first, the infant, mewling and puking.

Gestation lasts 19–20 days, with some variation between strains (Fekete, 1941); it is slightly increased in large litters (Biggers, 1963; McLaren & Michie, 1963). However, a female feeding young may have a pregnancy extended by 7–16 days by delayed implantation (Brambell, 1937; Fekete, 1940). Oestrus in laboratory mice can occur

Table 1. *The ages of wild-living house mice*

DURATION	CHARACTERISTICS	ENVIRONMENTAL INFLUENCES AND INTERACTIONS
1. *Up to birth* **Ovulation** every 4 days; gestation 19–20 days; pregnancy interval 4–5 weeks in laboratory	**Mother:** foraging and metabolic efficiency; litter size; sex ratio of litter **Offspring:** mortality and growth rate Epigenetic interactions	**Mother:** ambient temperature and food availability (energy needs up 70% during pregnancy); social factors, e.g. pheromonal and agonistic stimuli (Bruce, Whitten effects) **Offspring:** intra-litter competition for energy and nutrients; implantation site in relation to sex of neighbouring foetuses
2. *Nest life* **Weaning** at 14–15 days	**Mother:** maternal care milk production; nest quality **Offspring:** mortality-competitive ability Ultrasonic communication	**Mother:** ambient temperature and food availability; social stimuli **Offspring:** intra-litter competition for energy and nutrients; ambient temperature
	DISPERSAL	
3. *Sex life* **Puberty,** minimum 4 weeks in female, 7 weeks in males	Minimum age for fertility onset; sensitivity to social cues; tendency to disperse; tunnelling ability; mate choice	Ambient temperature and food availability Social factors, e.g. pheromonal and agonistic cues; soil characteristics for burrowing
4. *Social structure* Local deme persistence (2–12 adults) dependent on death (or disappearance) of dominant animals	Organized deme *vs.* disorganized neighbour–neighbour dominance system; territory size; aggressiveness; endocrine phenotype	Early social experiences Habitat characteristics: physical complexity, distribution of food and potential burrowing sites; population density; interspecific conflicts
5. *Population statics* End of breeding: 350 days in wild; 500+ days in laboratory	Heterogeneity of allele distributions Survival differences: breeding insulation; cold tolerance. Breed now if possible, otherwise later; here or elsewhere	Energy trade-offs: (72% used on maintenance; 10% on tissue repair) Food and temperature
6. *Senescence* Exhaustion of oocytes *c.* 12 months Decline of libido 12–30 months	Degenerative conditions Obesity?	Second winter Oncogenes?
7. *Death* 100–1000 days		Disease? Predation unimportant although crypsis necessary

every 4–5 days, under propitious environmental conditions which include the presence of male mice. Living in all-female groups disrupts this regular pattern (Whitten, 1959, 1966). Ovulation is independent of copulation (Allen, 1922; Bronson, Dagg & Snell, 1966). There is a post-partum oestrus 6–24 h after the birth of a litter. Ova number increases with both maternal age and parity up to the third litter (MacDowell, 1924; MacDowell & Lord, 1925). It is usually correlated with maternal size (Fowler &

Edwards, 1960). However, Batten & Berry (1967) found the correlation with size was absent in wild mice living on islands. Embryonic mortality may be affected by embryonic or maternal factors (reviewed by Whittingham & Wood, 1983). Litter size is 5–8 in wild mice (Laurie, 1946; Pelikan, 1974; Reichstein, 1978), although it is larger (6–10) in most laboratory strains (Crispens, 1979), and selected strains may have much larger litters (Falconer, 1960). However, mice selected for large size produce fewer litters than small-selected strains, and wean only half as many young (Roberts, 1961).

Equal numbers of males and females are born, although a few inbred strains have a sex ratio different from unity (in either direction) (Cook & Vlcek, 1961); starvation of females for a week (but not for two) before mating produces a deficiency of male young (Meikle & Drickamer, 1986), while female foetuses seem to be at particular risk from prenatal mortality produced by poor care of pregnant females (in laboratory conditions) (Howard *et al.*, 1955). The incidence of monozygotic twinning is about 1 % (Wallace & Williams, 1965).

Laboratory mice and wild mice kept under laboratory conditions breed all the year round; however, outdoor living mice in temperate regions are almost entirely seasonal breeders (e.g. Pearson, 1963; Breakey, 1963; Berry, 1968*a*), although mice in both the tropics and oceanic sub-Antarctic may breed continuously (Berry, Peters & Van Aarde, 1978; Berry *et al.*, 1981).

Using data of Myrcha, Ryskowski & Walkowa (1969), Grodzinski & Wunder (1975) calculated that pregnant and lactating females increase their energy intake by 78 % and their respiratory requirements by 65 %, when compared with non-reproducing females of the same weight. Bronson (1979, 1985, 1989; Bronson & Perrigo, 1987) has reviewed the factors controlling breeding. He concluded that six variables may at times be critical: total energy intake, specific nutrients in the diet, temperature variation, agonistic stimuli, specific tactile cues, and priming pheromones (Fig. 2). Seasonality arises from the interaction between energy intake and ambient temperatures; reproduction stops when food is scarce and temperature is low (Manning & Bronson, 1990). The house mouse is somewhat unusual among temperate small mammals in not being photoresponsive. Bronson (1979) has pointed out that this loose type of ambient cueing is rather typical of tropical species, but is ideally suited for the colonizing strategy of mice since it allows them to maximise their rate of population increase in a new environment (and in any season) when it is energetically and nutritionally possible. The emphasis of reproductive regulation is therefore biased towards flexible opportunism rather than static control.

(2) *Nest life*

And then the whining schoolboy.

Mice weigh *c.* 1 g at birth. Embryonic transfer experiments indicate that foetal genotype is important here (Snow, Tam & McLaren, 1981), but *in utero* litter size and maternal food intake are both potent determinants (Barnes *et al.*, 1973; Bronson & Marsteller, 1985). Post-natal growth is linear for 14–15 days, at which time weaning takes place, apparently dependent on milk availability (and hence litter size) (MacDowell, Gates & MacDowell, 1930; Barnett & Neill, 1971). Mothers moved from a warm (23 °C) environment to a cold (3 °C) produce milk with more fat (but less protein) than ones not moved; mothers from a stock maintained for many generations

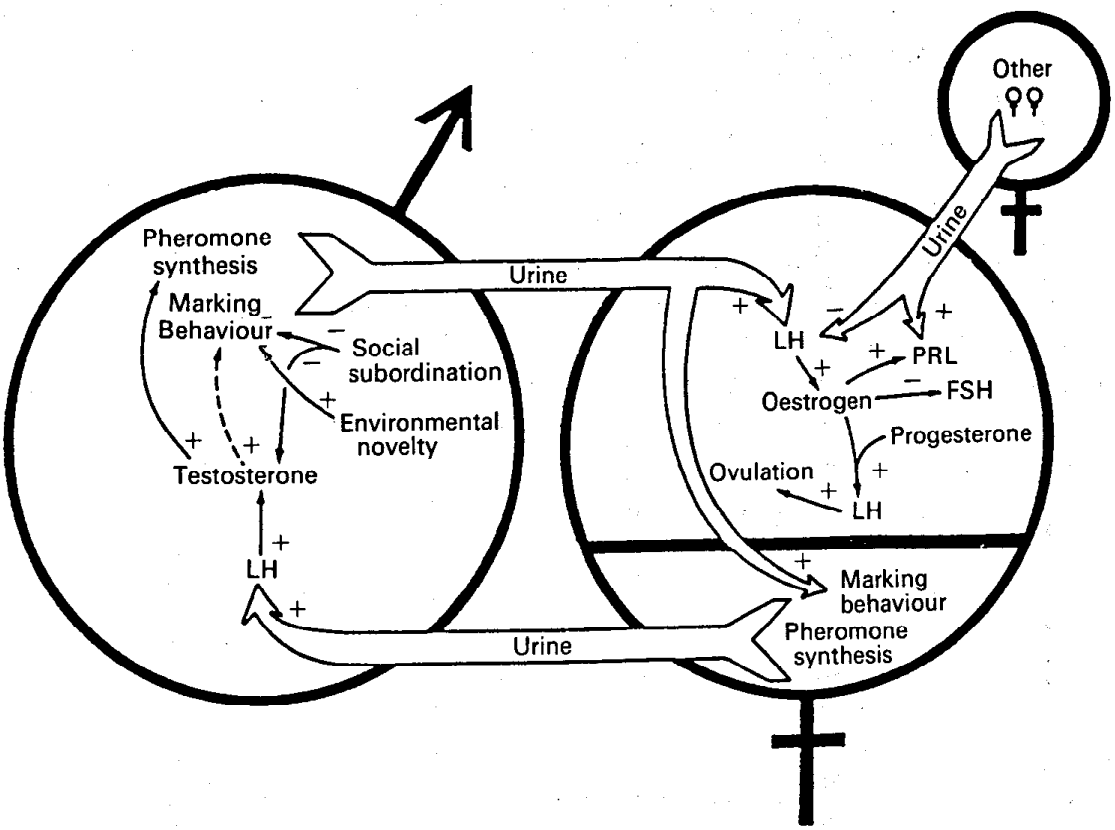

Fig. 2. The chemical priming system of house mice in which cues in the urine of one individual act in conjunction with tactile stimuli to influence the sexual activity of other individuals. Male mice induce the release of luteinizing hormone (LH) in both peripubertal and adult females. This results in the secretion of oestradiol and progesterone and then an ovulatory surge in LH secretion. This action of the male can be blocked in very young females by the presence of other females, through inhibiting LH secretion and enhancing prolactin (PRL) secretion. In adult females the presence of other females decelerates their oestrous cycle, although an adult male can override this effect. The secretion of follicle stimulating hormone (FSH) is uninfluenced by any social variable. Female marking behaviour and the synthesis of their urinary cues are independent of ovarian action. These cues cause a transient release of LH in males. In contrast to this situation in females, both marking behaviour and the synthesis of the relevant urinary cues are dependent upon LH and, hence, testosterone secretion (from Bronson, 1979).

at 3 °C have milk much higher in both fat and protein (Barnett & Dickson, 1984, 1989). The tail is *c.* 10% shorter in mice reared to weaning at −3 °C than at 21 or 28 °C (Biggers *et al.* 1958; Harrison, Morton & Weiner, 1959; Barnett, 1965).

The interaction between growth and developmental processes has been studied particularly by Grüneberg (1963; Grüneberg, Gray & Truslove, 1965). A particularly good example is third molar agenesis. This occurs in 17·9% of the CBA strain, 2·3% of the A strain, and occasionally in wild mice (Grüneberg, 1951; Berry, 1963). Crosses between animals with and without third molars may give progeny lacking such teeth, but in no Mendelian proportion and often clumped in particular litters, especially early or large ones. Strains (and crosses) which have missing third molars have smaller molars (where present) than strains which do not lack teeth. Maternal diet and lactation efficiency also affect the incidence of tooth agenesis. Grewal (1962) investigated the embryology of missing third molars. All the animals he studied had tooth germs. However, if these rudiments were below a critical size at 6 days after birth, they failed to invaginate to form a 'bell', and then regressed. The causal reason for tooth failure

is the lack of attainment of a threshold size by 5 days post partum. The size can be affected by loci influencing tooth development (such as shorthead, which is a cartilage defect; screw-tail, which is apparently mesenchymal in origin; or microphthalmia, which seems to be an endocrine defect) or overall body size (including hybrid vigour following outbreeding), by litter size (since young in small litters are larger at birth than those in large ones), and also by the rate of post-natal growth (Berry, 1968*b*).

Grüneberg (1963) concluded 'there is no doubt absence of third molars is a "satellite character" to small size of the tooth'. He proposed that traits inherited in this way should be called 'quasi-continuous', since they are determined by multiple genes despite their discontinuous phenotype. Such threshold traits may be morphological, physiological, behavioural, or pathological (Berry, 1969, 1989*a*). They are a model for characters (or 'strategies') where alternative states exist.

Both male and non-pregnant females may build nests, ranging in complexity from a simple shelf through a saucer-shape to an enclosed chamber. The form of nest built is clearly inherited (Lynch & Hegmann, 1972; Lynch & Sulzbach, 1984), although its size depends on ambient temperature (Lynch, Sulzbach & Connolly, 1988). The function of this activity is thermoregulatory as well as for care of the young (Barnett & Hocking, 1981).

Mice are efficient tunnellers in soft earth, and may dig burrows varying from a simple 2–3 cm diameter tunnel with one or more chambers, to a complex system with several exits and chambers (*c.* 10 cm in diameter), often lined with bedding material (Berry, 1968*a*) (Fig. 3). Away from buildings, their distribution may be limited by the lack of burrows. In Australian wheatfields, population increase is largely determined by rains softening the earth enough to allow the mice to burrow and move from refuge habitats into agricultural crops (Newsome, 1969).

Newborn mice are wholly dependent on their mother, although both males and females may show parental behaviour (retrieving unweaned young, grooming them, and lying in a lactating position) (Noirot, 1969). Maternal hormones are responsible for the onset of maternal behaviour at parturition, but olfactory and acoustic stimuli from the pups play an important part in eliciting and maintaining maternal behaviour (Smith, 1981). Indeed, pups may induce 'maternal' responses in both virgin females and in males who might otherwise kill them; males who have recently mated are less likely to kill pups (Labov, 1980; vom Saal, 1985).

In a wild-living population, Berry & Jakobson (1971) estimated that about half the young born (as calculated from foetal counts and pregnancy rates) failed to enter the adult population (i.e. immediately post-weaning). As we have seen, nest-building efficiency is under genetic control, and nest-type clearly affects nestling survival. Mice selected for high nest-building ability have greater basal metabolic rates, lowered food consumption and higher body temperature than others selected for low nesting ability/activity (Lacy, Lynch & Lynch, 1978).

Communal nests involving several mothers with litters are often found in dense colonies. Infanticide by mothers, other females, or (most often) by male mice varies with both strain and external conditions (Hrdy, 1979; McCarthy & vom Saal, 1985, 1986). The interactions of temperature, reproductive behaviour, and survival have been studied by several workers. In general, mice with access to insulating material have a comparable survival at experimental low temperatures to those raised at normal animal

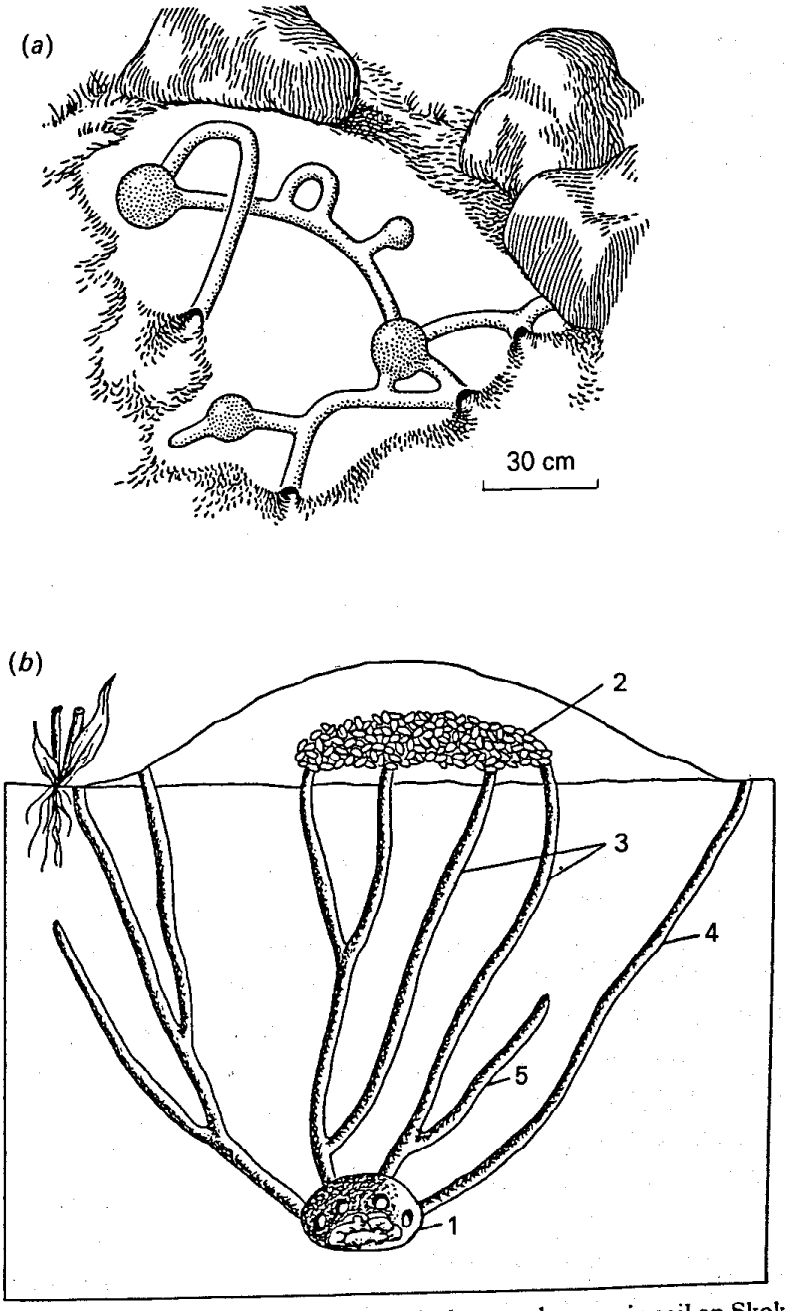

Fig. 3. House mouse burrows: (*a*) Excavation of a typical mouse burrow in soil on Skokholm. The runway system was in a 30° grass slope, with the actual runways up to *c.* 20 cm below the surface (from Berry, 1968). (*b*) Hillock of *Mus* (*spicilegus*) *hortulanus*. 1, nest chamber; 2, food chamber; 3, tunnels to food chamber; 4, tunnels to surface; 5, blind tunnels (from Muntyanu, 1990).

house temperatures, although their rate of breeding is slower with prolonged periods between litters (Barnett, 1962, 1973; Barnett *et al.*, 1975). Notwithstanding, the total number of young born is no different, at least as far as cold is concerned, to pairs acclimatized to either a warm or cold environment (Dickson, 1982; Barnett & Dickson, 1986). Newborn mice have little power of heat regulation; temperature control increases for the first 12 days of postnatal life (Barnett, 1956; Tarkonnen & Julka, 1966). There is an increased loss of animals, especially young males, if food is also restricted at low temperatures (Fuchs, 1982; Marsteller & Lynch, 1983). Bronson (1984; Perrigo & Bronson, 1985) have shown that the time necessary for foraging at low

temperatures is probably critical: when it is difficult to obtain food, peripubertal females allot their highest priorities to maintaining their energy balance, body growth is next, and reproductive development is least. Reproductive activity in males is less affected by food availability and temperature (Hamilton & Bronson, 1986).

(3) *Sex life*

And the lover, sighing like a furnace.

Puberty can occur as early as 4 weeks of age in the females of laboratory strains (later in laboratory-maintained wild stocks), but there seems to be considerable inter-strain variability. It is relatively easy to change the time of puberty by selection either for weight (or growth) (Falconer, 1984), or directly on the time of first oestrus (Drickamer, 1981*a*, *b*). Vaginal opening has been recorded as early as 24 days in the C57BL strain. Murray (1934) found the modal time for the first litter in DBA was 75–100 days, although 7% underwent their first oestrus at 4–5 weeks and produced their first litter before the age of 2 months. Puberty is up to 2 weeks later in males. Exposure of immature females to adult males or male urine hastens puberty (Vandenbergh, 1983), while exposure to a group of adult males or their urine retards it (Drickamer, 1977). Puberty is also delayed in females which have developed *in utero* between males (vom Saal, 1981) or who have been born into large litters (Drickamer, 1976), or, as already noted, by low temperature and food shortage.

Virtually nothing is known about specific dietary requirements (National Academy of Sciences, 1978; Ward, 1981) although added protein enhances breeding in both laboratory and wild (Bomford & Redhead, 1987). Domestication would be expected to produce selection for increased growth and accelerated puberty, but nothing is known about puberty in the wild (Wallace, 1981). However, Bronson (1985) has pointed out that the fact a running wheel in a cage leads to earlier puberty (Schneider, 1946; Perrigo & Bronson, 1985), suggests that the high level of foraging activity associated with acquiring food to sustain small body size has not been eliminated by any genetic changes accumulated during the comparatively short history of domestication.

The physiology of puberty in house mice is becoming increasingly well understood (Bronson, 1989). For the present purpose it is sufficient to assert merely that the timing of puberty in these animals reflects complex interactions between energy dependence and the social environment (Bronson & Rissman, 1986).

Mice have traditionally been assumed to mate promiscuously, so that the main determinant of parenthood would simply be presence in (and for a male, successful defence of) a territory. It is now apparent that a territory-holding male may not be the father of all litters born within his territory (Brown, 1953; Oakeshott, 1974; Busser, Zweep & Oortmersen, 1974). It is also recognized that mice can distinguish between genotypes of both the *H-2* and *t* haplotypes by urinary odour, and tend to choose mates by discriminating against *t*/+ heterozygotes and animals of the same *H-2* type as themselves (Boyse, 1983; Lenington & Egid, 1985; Lenington, Franks & Williams, 1988; Lenington, 1991). Moreover pregnancy blockage ('the Bruce effect') is increased when pregnant females are exposed to a different *H-2* than their mate (Yamazaki *et al.*, 1983). Direct observation of unconfined mice in a dense population suggests that females may actively choose their mate (Hurst, 1986). Notwithstanding, the current evidence is that this disassortative mating is fairly weak.

Intra-pair relationships may affect fitness. Pregnant females become increasingly aggressive as pregnancy progresses, which is presumably an adaptive response. St John & Corning (1973) found that maternal aggressiveness was high in inbred strains in which male aggressiveness was also high, although there was no increase in male aggressiveness when the trait was selected for in females (or vice versa) (Hyde & Ebert, 1976; Van Oortmerssen & Bakker, 1981).

(4) *Social structure*

> A soldier, full of strange oaths...jealous in honour, sudden and quick in quarrel.

Commensal mouse populations normally consist of a mosaic of male defended territories, each constituting a breeding sub-population or deme of 4–12 adults (Crowcroft, 1955; Crowcroft & Rowe 1963; Anderson 1964; DeFries & McClearn, 1972; Klein, 1975; Fitzgerald, Karl & Møller, 1981; Singleton, 1983). Laboratory experiments have shown that physical structure is an important part of the habitat; unless there is somewhere to hide, males will fight, often to death (Crowcroft, 1966). Territory size (or more strictly home range) varies from 2–6 m^2 in commensal populations (Southern, 1954; Selander, 1970) to 365 m^2 in open fields in the absence of voles (but only a third of this size if voles are present) (Quadagno, 1968).

However, extreme deme-rigidity occurs only in commensal (and by implication, most laboratory situations), where a territory-holder tends to be displaced by one of his own offspring. For example, in the hillock mouse of the Ukraine (*Mus hortulanus* = *M. spicilegus*), the adult of a pair gather a supply of seeds in the autumn. At the beginning of winter each 'hillock' is occupied by a pair of adults and their last litter of the season (Muntyanu, 1990). There is a high likelihood that the adults will die during the winter, resulting in the territory being taken over by members of the same family (Naumov, 1940; Anderson, 1970). In contrast to such a static situation, virtually every longitudinal study of mouse populations has shown a degree of churning. For example, Lidicker (1976) found a small amount of gene flow between established social groups in a large outdoor colony, with occasional more extensive mixing through the formation of new social groups (see also Myers, 1974*a*; Baker, 1981; King, 1982; Singleton & Hay, 1983, etc.). In a seven-year release–recapture experiment, Berry & Jakobson (1974) demonstrated that most adult animals remained faithful to a locality for extended periods, but more than a quarter of the mice bred at a site other than the one at which they were born. The maximum number of animals in a single group seemed to be six, but the composition of groups changed constantly because of a relatively high mortality at all ages.

A key experiment was carried out by Anderson, Dunn & Beasley (1964). They released *t* heterozygous male mice onto a small (7·5 ha) island, where no *t* allele was present. Their expectation was that the introduced *t* allele (haplotype) would increase in frequency because of its 95 % transmission rate in males (Lewontin & Dunn, 1960). In fact the *t* allele spread very slowly and eventually became extinct (Myers, 1974*b*). Anderson *et al.* (1964) concluded that 'social and ecological factors' limited its spread. Their assumption of no fitness deficit in the heterozygotes was incorrect (Pennycuik *et al.*, 1978; Lenington, 1983, 1991), but more importantly, another island introduction experiment showed introduced allozymes and Robertsonian chromosomes spread

rapidly, reaching apparent equilibrium within about 3 years (Berry *et al.*, 1991). The clear conclusion is that social structure only slows population mixing; the rigidity of exclusive deme organization is only temporary.

Despite the qualification about siring by young male non-territory holders, there can be no doubt that the majority of litters are fathered by a resident dominant (Dewsbury, 1982). The factors that lead to territorial acquisition are partly fortuitous: an existing resident always has an advantage over a challenger, particularly if he is larger (which may merely mean older) (Oakeshott, 1974). On the other hand, some males are more aggressive and successful in fights due to either genetic factors or previous social experience (Bronson, 1973; Mackintosh, 1981; Brain, 1989). The interactions involved have not yet been fully dissected, but involve multiple – apparently additive – genetic factors as well as environmental ones (*c.* 16% of identified mouse genes are concerned with behaviour: Berry, 1989*a*). Clearly endocrine control and variation are important (Shire, 1989; Charlton, 1984; Beamer, Wilson & Leiter, 1983), but the expression, activation and transmission of the endocrine phenotypes depend on both intrinsic and

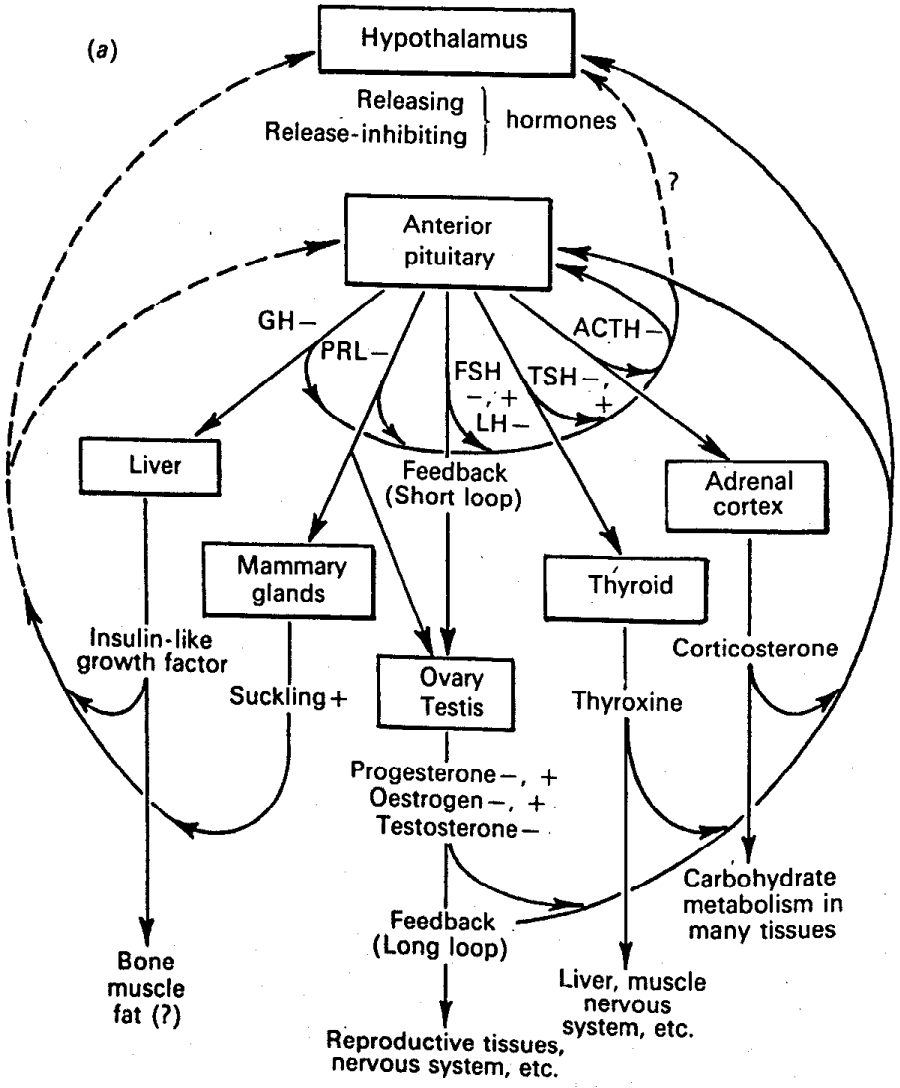

Fig. 4. Endocrine control in house mice. (*a*) Organization of the multi-levelled feedback systems regulating anterior pituitary gland hormones. The positive (+) and negative (–) signs show the nature of the feedback control. For simplicity, only hormones involved in feedback are shown; sex steroid feedbacks are included for LH and not FSH; feedback by pituitary hormones on other hormones are omitted. (Based on Beamer, Wilson & Leiter, 1983.)

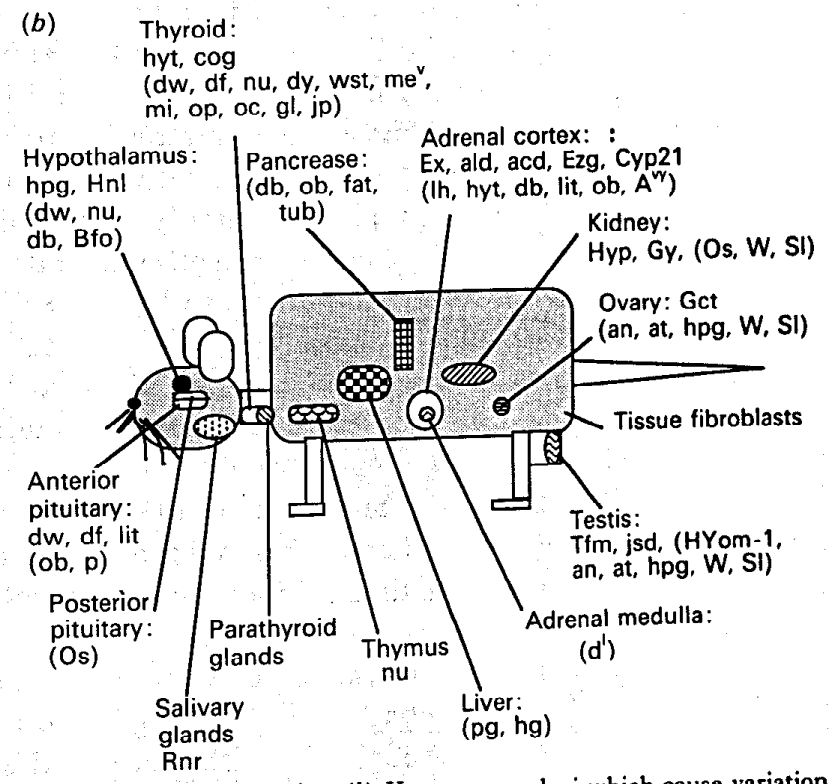

Fig. 4. Endocrine control in house mice. (*b*) Known gene-loci which cause variation in endocrine secretions (reproduced by permission of Wesley Beamer).

extrinsic factors (Barnett, Dickson & Wrath, 1980; Cains & Gariépy, 1989) (Fig. 4). An extreme example of such an interaction is the occurrence of the *rodless-retina* mutation in C3H and a number of other widely distributed inbred strains which puts animals from these strains at a disadvantage when matched with normally sighted mice (Fuller & Wimer, 1966; Simmel & Bagwell, 1983).

In inter-specific conflicts, house mice are poor competitors, apparently due to interference with successful reproduction rather than increased mortality. For example, house mice caged with *Peromyscus maniculatus* commonly win fights (King, 1957), but in the field, numbers decline in the presence of voles or other mouse species (Caldwell, 1964; Gentry, 1966; DeLong, 1966; Quadagno, 1968). Dueser and colleagues (Dueser & Brown, 1980; Porter & Dueser, 1982; Dueser & Porter, 1986) have shown that house mice always have the poorest habitats when co-existing on islands with one or more of six other small mammal species.

Lidicker (1966) and Berry & Tricker (1969) have described the extinction of island house mouse populations when challenged by *Microtus californicus* and *Apodemus sylvaticus* respectively. In both cases, reproduction was depressed in the house mice, and extinction followed as a result of inadequate recruitment. These studies complement investigations of physically unconfined house mice populations. In the latter, mice consistently move from disturbed habitats to colonize empty ones (Justice, 1962; Stickel, 1979; Singleton, 1985; Lidicker & Patton, 1987). House mouse strategy is that of a weed: rapid local establishment and expansion, alternating with comparatively long-term dispersal and colonization (Berry, 1977; Anderson, 1989).

(5) *Population statics and stability*

And then the justice, with full round belly... with eyes severe... he plays his part.

Wild-caught mice described by taxonomists or experimentalists (whether physiologists or biochemists/molecular biologists) tend to be collected when animals are easy to trap (i.e. when populations are large, which means towards the end of the breeding season). They may give an artificially static view of the pressures which have been and are operating on the populations. Significant variables include:

(*a*) *Fortuitous genomes.* Mouse colonies are repeatedly extinguished and re-established from immigrants (Berry, Cuthbert & Peters, 1982). Because of the amount of inherited variation in *Mus domesticus*, new founders are likely to differ genetically both from the existing (or previously resident) animals and from the population whence they are drawn (Berry, 1964, 1986*b*). Mouse populations continue to show significant amounts of continuous genetic change, even after isolation for decades (Berry & Jakobson, 1975*a*).

(*b*) *Cold.* Food availability and ambient temperature interact closely, and it is artificial to separate them (Fig. 5), yet this is commonly done in the laboratory. Theoretical and ecological evidence indicate that the most acute physiological problem suffered by a small mammal like a mouse is cold – or more strictly, episodes of cold (Hart, 1953, 1957; Brown, 1963; McNab, 1963*a*, *b*; Berry, 1968*a*; Berry, Jakobson & Triggs, 1973; Grodzinski & Wunder, 1975; Jakobson, 1978, 1981; Hayward & Phillipson, 1979; Kaplan, Brewer & Blair, 1983). This is because of their large surface area relative to mass and their lack of fat stores. At normal room temperature starved peripubertal mice lose up to one-third of their fat in 14 h; at 11 °C they exhaust their fat reserves in less than 1·5 days (Bronson, 1987; Manning & Bronson, 1990). Body temperature is 34–40 °C at most environmental temperatures. The temperature below which mice respond to cold by increased heat production is 30–32 °C (Mount, 1971), which is similar to that chosen when mice are placed in thermal gradients. From nest and huddle temperatures, Barnett, Munro, Smart & Stoddart (1975) suggest that mice may spend most of their resting phase close to thermal neutrality. Fur contributes 30–40% of total insulation, with a 20–80% improvement in cold adapted mice (Barnett, 1959). Remaining insulation reflects vasoconstriction and differential cooling of peripheral tissues. Only about 5% of the autumn (end of breeding) population may survive a particularly cold winter, whereas the numbers living through a mild winter may lead to plague conditions in the following autumn, although the rate of increase in the breeding season is the same (Berry & Jakobson, 1971). Metabolic rates differ between strains (Pennycuik, 1967, 1972).

(*c*) *Food.* Judging by stomach content analysis, mice apparently prefer insect to plant food. The proportion of insects in the diet increases greatly in early spring (Whitaker, 1966; Berry, 1968*a*; Berry & Tricker, 1969; Badan, 1978; Gleeson & van Rensburg, 1982), whereas most of the food in winter is plant-derived (Stueck & Barrett, 1978). Water is probably never limiting in normal conditions (Haines & Schmidt-Nielsen, 1967; Fertig & Edmonds, 1969; Haines, Ciskowski & Harms, 1973). Cold acclimated mice can survive experimental periods of cold better than non-acclimated ones, but this improvement disappears after fasting for 24 h (Hart, 1957). Two-month-old laboratory mice (kept at 24 °C with unlimited food) use 72% of their daily energy intake on maintenance and a further 10% on body tissue deposition (Stephenson & Malik, 1984).

(*d*) *Recruitment.* As already noted, temperate wild-living mice are seasonal breeders, and inter-specific disturbance may reduce recruitment, even in the normal breeding

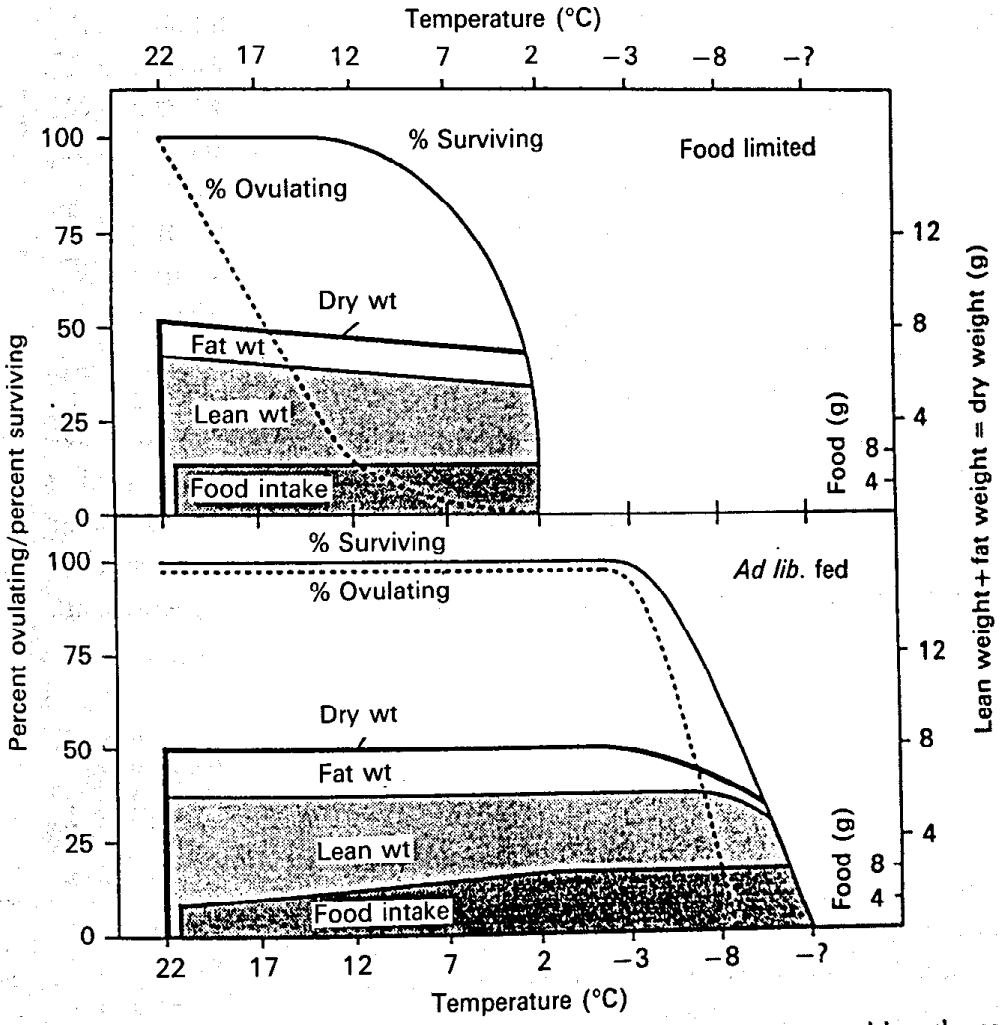

Fig. 5. The effect of food availability and ambient temperature on body composition, the capacity to ovulate and survival. In the bottom graph the females were allowed to increase their food intake at lower temperatures. Under the conditions of this study, a limit on the amount of food that could be processed occurred at 2 °C. At lower temperatures they began to lose fat and the capacity to ovulate was lost just above the temperature at which survival was compromised (mice were not allowed to die in this experiment; impending mortality was assessed on the basis of behavioural observations and rapid loss of body weight). Females in the top graph were not allowed to increase their food intake at lower temperatures. Under these conditions ovulation ceased at a much higher temperature and the animals maintained their fat content and their lean body weight until survival was compromised at between 2 and 12 °C (from Manning & Bronson, 1990).

season. This means that the age structure of mouse populations changes throughout the year. Under conditions of low disturbance, favourable climatic conditions and ample food, the breeding season may be extended. In situations of low mortality and dispersal, Southern & Laurie (1946) and Berry & Jakobson (1971) found that mouse populations double every two months. This leads to typical peak density in agricultural land of 50–60 animals per hectare. In plagues (recorded in the USA, but most commonly in Australia: Pearson, 1963; Newsome & Crowcroft, 1971; Newsome & Corbett, 1975; Singleton & Redhead, 1990) densities may rise to more than 1000 per hectare, while in barns or artificial enclosures, numbers may reach 50–70000 per hectare (Selander, 1970; Lidicker, 1976).

(e) *Mortality*. Most mortality in mice is temperature dependent; other causes of death are discussed below (under death).

Because there are so many environmental pressure acting on so many genotypes, it is meaningless to regard any phase of a mouse's life as intrinsically stable (Berry, Jakobson & Triggs, 1971; Berry, 1989*b*). It is more accurate to regard different pressures as producing different amounts of stress. Interpreted in this way, stress is extrinsic to the organism, as distinct from the common mammalian usage which concentrates on the intrinsic (largely hormonal) responses of an animal and hence tends to isolate it conceptually from its environment (Selye, 1956; Calow & Berry, 1989). Response to stress may either be phenotypic, as when haemoglobin amounts increase and basic metabolic rates decrease at the advent of cold (Jakobson & Moore, 1971; MacLean & Lee, 1973) or when agonistic interactions increase blood corticosterone levels and depress reproduction (Bronson, 1979), or genetic, as shown by seasonal or ontogenetic changes in allele frequencies as a result of differential survival or reproduction of different genomes (Berry, 1978). Fitness (i.e. successful reproduction) is inversely correlated to stress. By determining the consequences of stress, we are likely to arrive at more sensitive measures of fitness than by direct measurements (Berry, Jakobson & Triggs, 1973; Berry, 1985).

(6) *Senescence*

> The sixth age shifts into the lean and slipper'd pantaloon... His youthful hose well sav'd a world too wide for his shrunk shank; and... pipes and whistles in his sound.

The mean life-span of laboratory mice is around 550–600 days, differing somewhat between strains (those not selected for overt pathology). Male longevity is slightly less than that of females in most strains (Russell, 1966). Hybrids between strains commonly live more than 650 days. Females which breed tend to have 15–20% shorter lives than virgins. In contrast, Berry & Jakobson (1971) reported that the average life of mice on a Welsh island was about 100 days, with a similar expectation of life at all times until the beginning of the second winter. No animal was found that survived two winters. DeLong (1967) found similar mortalities in mice living on flat land around San Francisco Bay. However, Varshavskii (1949) found that 4% of mice of open steppe in the USSR and over 22% of urban animals lived over 21 months. Wild-caught mice survive better in captivity than in their natural circumstances: 1·5% of a large sample of wild-caught animals were still alive after 30 months (900 days) in captivity (Gardner *et al.*, 1974).

Laboratory males often remain fertile throughout their adult life, although their libido may decline with age (Bronson & Desjardins, 1986). Two factors affect female fertility: depletion of the oocyte population (reaching zero in CBA mice by 300–400 days: Jones & Krohn, 1961), and deterioration of the uterus itself (Biggers, Finn & McLaren, 1962; Talbert & Krohn, 1966; Finn, 1970). Nothing is known about the effective reproductive span of wild-living mice.

In the light of the markedly different life-spans of wild as compared to laboratory mice, most studies of senescence in the latter have no relevance to the evolution and life of wild-living mice (Bellamy *et al.*, 1973; Bellamy, 1981). However, there is a positive correlation in inbred strains of 0·9 between female life-span and litter size, which implies that longevity may be affected by natural selection for fertility – or that intermediate litter sizes might be favoured (Roderick & Storer, 1961). A similarly strong

correlation and perhaps more important finding is that cold tolerance decreases with age, and this is also correlated with life-span (Talan & Ingram, 1986). It is not known whether the decline with age in preferred temperature and the change in preference with season are expressions of the same association (Ogilvie & Stinson, 1966).

Notwithstanding, ability to cope with a harsh environment certainly decreases with age, as shown by the failure of animals which survive one winter (and therefore have the physiological capability to respond to winter – presumably mainly cold – stress) but succumb early in the second winter. It is not known which systems fail (Berry, Jakobson & Triggs, 1963; Berry, Jakobson & Peters, 1987).

(7) *Death*

Last scene of all that ends this strange eventful history is... mere oblivion, sans teeth, sans eyes, sans taste, sans everything.

Studies of both mammal and bird predators indicate that comparatively few house mice are killed by them, except in plagues where owls and foxes may kill large numbers. For example, Harris (1965) found only one pellet containing a house mouse in an intensive study of gull diet, although mice were living in the nesting colonies on one of the two islands where his gulls lived. Glue (1967) recorded 1·4% by weight of house mice in 32353 vertebrate prey items from British barn owl (*Tyto alba*) pellets. However, mice form 25% of the diet of barn owls in Washington, DC and Illinois, 10% in Pennsylvania, but virtually none in Michigan and Wisconsin (data summarized by Varshavskii, 1949). Evans (1949) recorded a barn owl hunting over 25 ha of a high-density mouse population. This bird ate at least 283 mice in a year (28% of all its food items), but it is not known how many individuals were potentially available to it. Pearson (1963) noted how cats preferentially preyed on voles (*Microtus californicus*) until these were reduced to very low numbers; the cats then switched to mice.

In New Zealand house mice are the only small mammal. Feral cats and stoats are major predators (King, 1983; Fitzgerald & Karl, 1979). Although predation does not affect the occurrence of mouse plagues (which erupt in *Nothofagus* forests after a heavy seed fall), it may influence the size and timing of the pest population and subsequent decline (King, 1982, 1989).

The only reports of epidemic disease in wild mice are from populations at high densities: pneumonia seems to have been an important cause of death in a mouse plague in California (Piper, 1928), while large numbers of mice dead from disease were reported during two Russian outbreaks (Fenyuk, 1934, 1941); Pearson (1963) described 'large haemorrhagic patches of unknown aetiology' in the lungs when one of the populations he studied was decreasing in number during the winter; DeLong (1967) found mice carrying 'an enteric streptococcus in the spleen and liver' and apparently dying from septicaemia at a time when the population density decreased eight-fold in one month; Sherriff (cited by Newsome & Crowcroft, 1971) has suggested that sick mice in one dense (cereal) rick population might have suffered from 'a combination of an eperythryzoan infection coupled with murine hepatitis'. At more usual densities, Anderson, Dunn & Beasley (1964) found young mice in an island population 'fatally parasitized' by larvae of botflies (*Cuterebra* sp.); Bellamy *et al.* (1973) recorded infertility and uterine oedema in a number of adult females harbouring berry bugs (*Neotrombicula autumnalis*). Only negligible titres of antibodies to bacterial and viral

diseases have been recorded in British wild-caught mice (Berry, unpublished). As far as laboratory mice are concerned, Munro (1972) found that the commonest causes of death in mated females were colonic blockage (nematodes) and liver necrosis.

Experimental and commensal studies of different crypsis and survival have shown that coat colour may be important for protection against predators in house mice (Brown, 1965; Smith & Watson, 1972; Kaufman & Wagner, 1973), but the adaptive significance of coat colour has never been demonstrated in truly wild populations. The case of the light-coloured mice of the sandy North Bull Island near Dublin claimed as adaptive by Jameson (1898) and cited by Huxley (1942) is probably irrelevant; although light coloured (and hence potentially cryptic) mice occur on the island, they are also common throughout the neighbouring city (Fairley, 1971; Berry, 1977).

Oncogenic viruses are found in virtually all laboratory and wild-caught house mice, but do not produce any pathological change until after the age when most wild mice will have disappeared from the population (Longstreth & Morse, 1981; Gallahan, Escot & Callahan, 1986; O'Brien & Evermann, 1988; Gardner, Kozak & O'Brien, 1991). It is not known if they have any effect on fitness.

IV. FITNESS TRADE-OFFS

> Animals are not always struggling for existence. They spend most of their time doing nothing in particular. But when they do begin, they spend the greater part of their lives eating.
>
> (Charles Elton, 1927)

House mice are weeds, able to colonize (and hence tolerate) environments as diverse as coral atolls in the Pacific to near-Antarctic conditions in South Georgia, from bird cliffs in Faroe to 2500 m above sea level in Hawaii or 4000 m in the Andes, from central heating ducts to refrigerated stores (Berry, 1981*a*, 1987; Sage, 1981; Efford, Karl & Møller, 1988). Although the limited information available suggests that many colonizations fail and that populations frequently become extinct (Berry & Johnston, 1980; Berry, Cuthbert & Peters, 1982), the geographical and habitat range of the species indicates that colonizing groups (which means in effect, a small number of individuals) are able to adjust to conditions significantly different to their origins (Berry & Jakobson, 1975*b*). There is direct evidence of such adjustment from the apparent ease with which laboratory escapers achieve successful feral life, and from experimental studies (MacLean & Lee, 1973; Jakobson, 1981). However, more convincing support for house mouse adaptability comes from data on the response range of individuals to different conditions (particularly the work of Barnett and his colleagues, q.v. Barnett & Dickson, 1989; but see also Berry & Jakobson, 1975*b*, and studies of mice moving between refuge/survival and colonization habitats: Newsome, 1969; Anderson, 1970, 1978, 1989; Singleton, 1989). It has been the purpose of this paper to present these data in terms of a model of life-history strategy and selection pressures. This model can only be qualitative at the moment, but the fact of its existence should make a quantitative one achievable.

Stearns (1976, 1989) and others (Calow & Sibly, 1983; Calow 1984; Sibly & Calow, 1985; Begon, 1985; Boyce, 1988; Caswell, 1989) have reviewed the causes and dilemmas of life-history adaptation. Maynard Smith (1978) has set 'life history theory' in the framework of optimization theory (see also Southwood, May, Hassell & Conway,

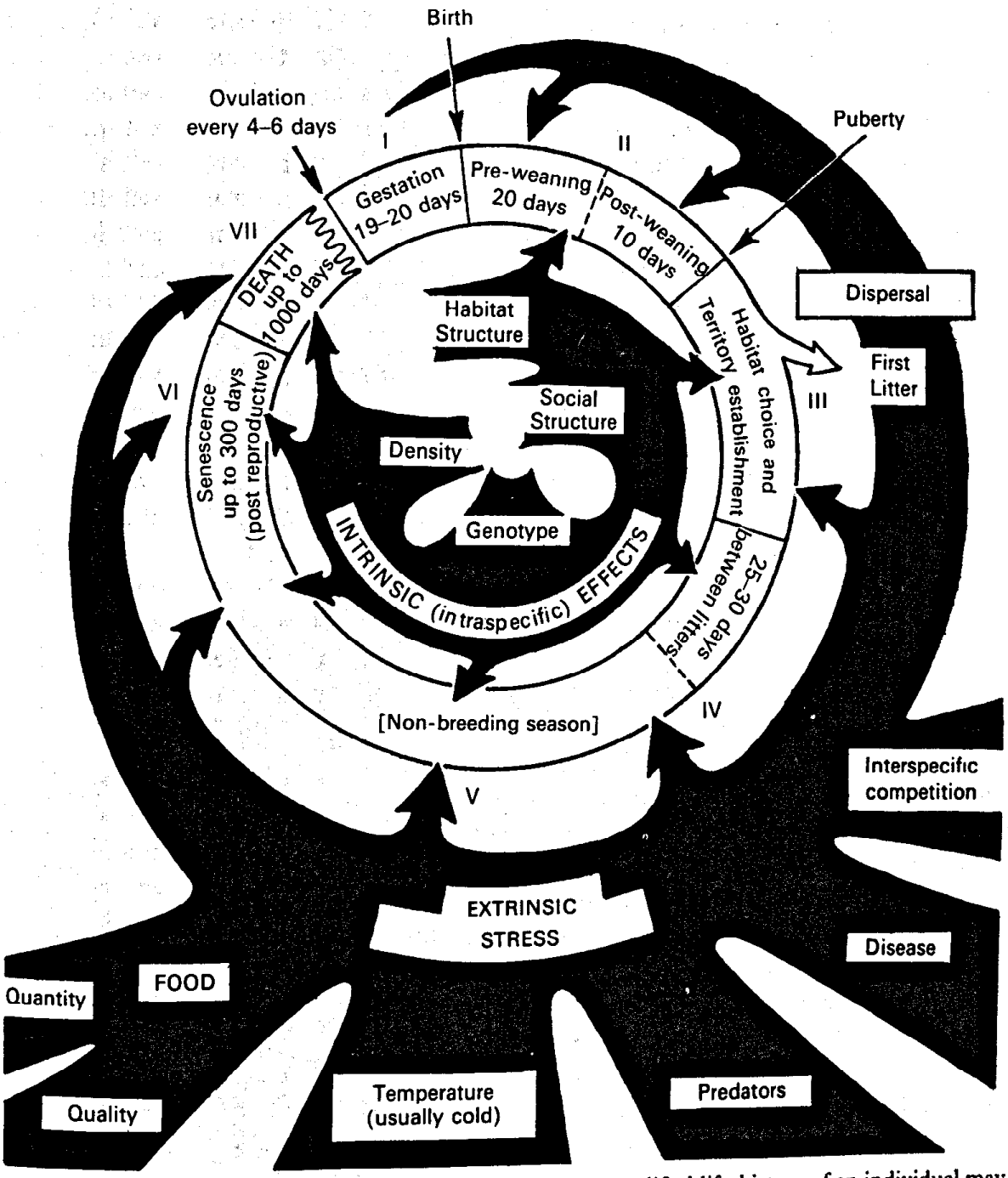

Fig. 6. Formalized life-cycle of a non-commensal mouse. The modified life-history of an individual may be influenced by extrinsic factors (outer arrows) or intrinsic (intraspecific) factors (inner arrows). Habitat structure affects population parameters and has been included as an intrinsic influence, but it may also be an extrinsic factor.

1974; Southwood, 1977, 1988; Rose, 1983; Parker & Maynard Smith, 1990). Like all adaptation, life history evolution will take place if:

(*a*) The variation in a particular trait is inherited.

(*b*) Environmental variance produces a differential stress on different inherited phenotypes.

(*c*) The environmental stress(es) are not cancelled out by phenotypic responses (homeostasis).

We have described the intrinsic responses to extrinsic variables which house mice

experience. Our aim was to identify key interactions worth further study. However, there are so many interactions between genes, phenotypes, biotic and abiotic influences, and time that no simple matrix is possible. Virtually every intrinsic trait has genetic variation in wild populations or different inbred strains; the most important phenotypic variants seems to be body size (increasing fighting success and litter size – albeit reducing the number of young weaned, at least in the laboratory), endocrine expression (affecting both male and female aggressiveness, lactation, general maternal care, pheromone production and response), intra-specific communication (sound, sight and smell), nest building, food gathering and use, density and inter-specific responses. The most important extrinsic factors are habitat quality; food quantity, distribution and quality; and cold. Genetic variation and temporal changes (both seasonal and age) interact with the more obvious intrinsic and extrinsic factors. It is not possible to produce a simple gene–environment model of these interactions; notwithstanding, there can only be a finite number of intrinsic (gene and ontogenetic) and extrinsic (biotic and abiotic) interactions with time (Fig. 6). A tractable model of house mouse life-history variation seems in principle feasible.

It is possible to quantify key elements in such a model? Current ignorance about the likely costs of the various components of fitness in relation to climatic change and competition are such that a simple energetic model is not yet achievable. However, an alternative approach incorporating historical and climatic indeterminacy (or opportunism) with epigenetic possibilities may be nearer. For example, the genetic and environmental contributors to litter size (and survival to weaning), nest building efficiency, body weight, cold tolerance, competition (including disease and parasite susceptibility), and density dependence are all known in principle; unfortunately, the environmental influences are known better than the genetic. Notwithstanding, the possibility of analysing the genetical contributions to a model is probably closer than is apparent from the vagueness of much ecological literature; it is a similar approach to Waddington's 'epigenetic landscape' (Waddington, 1957, p. 36). There are four simplifying considerations.

(*a*) Direct analysis of particular traits is possible. It has been successful for vertebral axis development (Grüneberg, 1957), litter size (Falconer, 1960), third molar loss (Berry, 1968*b*) and mandible shape (Bailey, 1985, 1986*a*). A general model is available (Bailey, 1986*b*), which is supported by the emerging evidence of homeobox control (Rossant & Joyner, 1989).

(*b*) Biometric analysis of multigenically controlled traits rarely gives information about individual gene action. However, there are biometrical techniques available for isolating and characterizing the effects of single genes within additive complexes (e.g. Holt, 1945; Wallace, 1972; Wallace & MacSwiney, 1976), and knowledge about molecular actions and interactions are now approaching the stage when molecular data can begin to contribute to developmental and hence more general biological problems (e.g. Levinton, 1986).

(*c*) Fitness can be analysed by selective crossing and inbreeding of mouse strains to reveal important contributants to genetic architecture. It is clear that there are significant blocks of genes which interact heterotically and are only broken up by continued inbreeding (Wallace, 1965; Connor & Bellucci, 1979). Comparison of the human and mouse chromosome maps shows large amounts of linkage conservation

(Sawyer & Hozier, 1986; McKusick & Roderick, 1987; Nadeau, 1989), and indeed, 'genetic architecture' is likely to be similar for most mammals.

(*d*) Comparative studies may indicate which structures or processes are determined or limited by allometry, and which indicate particular adaptations (Adolph, 1949; Caswell, 1989; Harvey & Page, 1989). As far as house mice are concerned, little useful information has so far emerged from this approach (e.g. Mousseau & Roff, 1987); an additional limitation is that comparison of pathological processes in mouse and human shows many are time-dependent, and hence the mouse is not a useful disease model for all human conditions (Erickson, 1989), although the conservation of genetic sequences for molecules important in pathology indicates that mice may have an increasing role in the treatment of molecularly-defined diseases (Erickson, 1990). This means that the comparative approach is likely to have an increasing value.

The next stage in analysing the bioeconomy of the house mouse will be to integrate existing knowledge of mouse genetics and development with physiology and behaviour, and seek to identify specific gene–environment links with relevant environmental variables. Traits suitable for such an approach include body size, tail length, litter size, age of puberty, life-span, agonistic and aggressive behaviour, disease resistance, and competitive ability. This will be a formidable task. Although we have described in this paper the main elements in traditional life history traits, there are aspects of house mouse biology we have not even mentioned. An obvious omission is the extraordinary commonness of Robertsonian translocations of the chromosomes in *Mus domesticus*. Standard fitness theory suggests that such changes should be rapidly eliminated, but in some places they seem to be spreading (Berry, 1986*b*; Berry & Corti, 1990). But the depth of knowledge of house mice suggests that the effort to carry out studies of the interactions of gene and gene-environment interactions along the lines of the third molar determination model will be well worthwhile.

V. SUMMARY

1. More is known about the western European house mouse, *Mus* (*musculus*) *domesticus* than any other non-human mammal. If laboratory and field information is combined, an extremely valuable understanding of the species' bioeconomy could be obtained.

2. The seven stages of mouse life-history are surveyed (up to birth, nest life, sex life, social structure, population statics and stability, senescence, and death), and the interactions between the changing phenotype and the environment are described.

3. These interactions can be used to build up a model of the opportunities and compromises which result in the fitness of individual mice. It is not yet possible to quantify such a model, but this should in principle be achievable.

VI. ACKNOWLEDGMENTS

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