Cognition, 40(1991) 159–201

The evolution of the critical period for language acquisition*

James R. Hurford

Department of Linguistics, University of Edinburgh, Adam Ferguson Building, George Square, Edinburgh, EH8 9LL, UK

Received March 14, 1990, final revision accepted May 15, 1991

Abstract

Hurford, J.R., 1991. The evolution of the critical period for language acquisition. Cognition, 40: 159–201.

Evidence suggests that there is a critical, or at least a sensitive, period for language acquisition, which ends around puberty. The existence of this period is explained by an evolutionary model which assumes that (a) linguistic ability is in principle (if not in practice) measurable, and (b) the amount of language controlled by an individual conferred selective advantage on it. In this model, the language faculty is seen as adaptive, favoured by natural selection, while the critical period for language acquisition itself is not an adaptation, but arises from the interplay of genetic factors influencing life-history characters in relation to language acquisition. The evolutionary model is implemented on a computer and simulations of populations evolving under various plausible, if idealized, conditions result in clear critical period effects, which end around puberty.

1. The phenomenon to be explained

A body of evidence suggests that there is in humans a critical period, or at least a sensitive period, for the acquisition of (first) language. The critical period hypothesis was most prominently advanced by Lenneberg (1967), a work which

*I thank the following for helpful discussion or comments on this paper: Jean Aitchison, Ellen Bard, James Cooke Brown, Phil Carr, Peter Caryl, Grev Corbett, Tim Ingold, Steve Isard, Aubrey Manning, David Smillie, John Maynard Smith, Jim Monaghan, Len Nunney, Linda Partridge, Katharine Perera, Steven Pinker, Bill Watson, and several anonymous referees. Work on this paper was also greatly helped by a Visiting Research Fellowship generously awarded by the University of Melbourne, Australia, for a stay at its new Department of Linguistics.

remains a classic source on this topic, since when further evidence, surveyed below, has emerged. Lenneberg's evidence was drawn from (1) recovery from traumatic aphasia, lateralization of speech function and hemispherectomy, and (2) Down's syndrome children.

Before puberty, a child struck by aphasia has a reasonable chance of recovering and developing normal language, the recovery possibly taking some years. But adults so afflicted seldom recover language in full, and as a rule never recover basic verbal capacities for communication beyond the level achieved three to five months after the impairment. People whose language ability is destroyed after puberty seem to have diminished resources for rebuilding it.

Lenneberg summarizes the evidence from Down's syndrome children thus:

> In a study by Lenneberg, Nichols, and Rosenberger (1964), 54 mongoloids (all raised at home) were seen two to three times a year over a three-year period. The age range was from 6 months to 22 years. The appearance of motor mile-stones and the onset of speech differed considerably from individual to individual, but all made some progress – although very slow in many cases – before they reached their early teens ... But interestingly enough, progress in language development was only recorded in children younger than 14. Cases in their later teens were the same in terms of their language development at the beginning as at the end of the study. The observation seems to indicate that even in the absence of gross structural brain lesions, progress in language learning comes to a standstill after maturity. (Lenneberg, 1967, pp. 154–155)

(Lenneberg, 1972, pp. 388–390 also summarizes his evidence for a critical period for language acquisition.)

Lenneberg was sceptical of the conclusions that can be drawn from studies of wolf-children and radically neglected children (discussed, collected and summarized, respectively, in Koehler (1952), Singh and Zingg (1942) and Brown (1957)). Lenneberg was perhaps too skeptical; I would say that, though the evidence is sketchy and unsystematic, at least some support for the critical period hypothesis comes from such cases of people whose exposure to any language at all is artificially delayed until about puberty or later; these people seem to have missed the chance to acquire language fully. Since Lenneberg wrote, further such evidence has come to light, in the case of Genie, who was kept away from social contact for most of the first 13 years of her life (described in full in Curtiss, 1977). Curtiss carefully studied Genie's progress in language acquisition after her discovery, and administered tests to determine Genie's lateralization of language function. She summarizes her findings thus:

> From our observations and testing, Genie appears to be a right-hemisphere thinker. Most importantly, she uses her right hemisphere for language. Genie's language is abnormal in specific ways. Her language resembles that of other cases of right-hemisphere language as well as the language of those generally acquiring language outside

> the "critical period". Her case, therefore, supports Lenneberg's "critical period" hypothesis and furthermore suggests specific constraints on the nature of language acquisition outside of this maturational period.
>
> The fact that Genie has right-hemisphere language may be a direct result of the fact that she did not acquire language during the "critical period". It suggests that after the critical period, the left hemisphere may no longer be able to function in language acquisition, leaving the right hemisphere to assume control. (Curtiss, 1977, p. 234)

(Compare the case of Genie, isolated until the age of 13, with the case reported in Lazar et al. (1983, p. 54) of "a male patient, successfully treated for SCID [severe combined immunodeficiency disease], who lived in reverse isolation from 9 months to 4 years 4 months of age and who has demonstrated nearly complete recovery of language usage." Although the two cases obviously differ in many factors besides the age at which isolation ended, the comparison is nevertheless of some interest.)

There is also evidence of a critical period effect in signed human languages, such as American Sign Language (ASL):

> There also seems to be a critical period: children who learn ASL after, say, 7 years of age, will have a sort of foreign accent phenomenon, as Eric Lenneberg called it; they will not speak like native signers. (Ploog, 1984, p. 88)

The conclusion that a critical period exists for the acquisition of ASL is further fleshed out in an unpublished study by Newport and Supalla, reported in Johnson and Newport (1989):

> This study thus provides direct evidence that there is a decline over age in the ability to acquire a first language. It also tells us, however, that Lenneberg's portrayal is at least partially incorrect in two regards. First, the results show a continuous linear decline in ability, instead of a sudden drop-off at puberty as his hypothesis implies ... Second, it should be noted that, while the postpubescent learners did not reach as high a level of proficiency as the native or early learners, language had not become totally unlearnable for them. This rules out any extreme interpretation of the critical period hypothesis. (Johnson & Newport, 1989, p. 63)

I am not entertaining an extreme form of the critical period hypothesis. "At least some degree of first language acquisition seems to be possible beyond the critical period" (Fromkin, Krashen, Curtiss, Rigler, & Rigler, 1974 (p. 299 of 1978 reprinting)). Enough language acquisition is possible after puberty even to permit limited verbal communication. But, to judge from Genie's case, some quite central aspects of grammar are difficult, if not impossible, to acquire after puberty, including interrogative structures, third person, relative, indefinite and demonstrative pronouns, and much of the structure of the auxiliary verb. The critical period for language acquisition is not a single unified phenomenon, just as the object acquired – a language – has separate components. Thus the fact

that Ploog, quoted above, somewhat arbitrarily identifies the 7-year-old stage, that is, before puberty, as the stage after which ASL learners show a "foreign accent phenomenon" is not disturbingly at odds with the gist of the idea of a critical period as envisaged here. No doubt the various facets and layers of the structure of a language have different, though substantially overlapping, critical periods. Acquisition of the low-level phonetic rules probably diminishes at a stage when acquisition of grammatical structure is still relatively easy. And acquisition of vocabulary is still relatively easy after the facility to acquire grammatical rules has dwindled or disappeared. The gross phenomenon of the critical period is an aggregate of a number of roughly coinciding particular phenomena. This fact relates to the question of polygenic inheritance, taken up later (section 2.3).

The term "sensitive period" is sometimes used for a less marked kind of critical period. The difference between a critical period and a sensitive period is fuzzy, like that between a mountain and a hill. The explanation to be offered in this paper in fact points to the likelihood of a period with relatively steep, but not absolutely abrupt, boundaries. A recent data survey on the critical period (Aitchison, 1989) concludes: "There is no evidence of a sudden onset, or final endpoint of the supposed critical period. Instead, we are dealing with a phenomenon well known in animals, the fact that young brains are more flexible than older ones" (Aitchison, 1989, p. 89). But how sudden is "sudden"? Clearly there is no instant at which the curve of language-learning ability drops vertically from some high value to zero. What shape, then, *is* the curve of language-learning ability against age? The evidence available seems to indicate a curve which rises early, maintains a high level before puberty, and then falls, with the steepest downward slope coinciding roughly with puberty. Of course, no one is in a position actually to draw this curve. "It is not clear how to scale the ease of learning a language at different ages without having that assessment placed in the context of the child's general learning capacity" (Bever, 1981, p. 179). Like Bever, I am led to accept:

> as a pretheoretic truth that there *is* a critical period for language learning. Scholars who claim that it is "unproven" that languages are harder to learn in middle age than childhood may be technically correct, in the sense that there is little "scientific" data demonstrating that relative difficulty. However, the claim is also disingenuous, since there is ample individual evidence that the older one is the harder it is to master a second language well enough so that a native speaker cannot detect that one is a foreigner. I think that there is very little argument about such facts: it clearly is the case that people who learn a new language after age 20 rarely do so with the proficiency of people who start learning before age 10. This difference persists even after many years of exposure to the language. (Bever, 1981, p. 179)

Impressionistic statements such as Bever's above introduce the further question of whether the critical period effect can be found in *second* language acquisition and, if so, whether this is relevant to the question of a critical period for first language acquisition.

Johnson and Newport (1989) survey relevant research in second language acquisition, and point out that the apparent contradictory nature of the research conclusions can be resolved by distinguishing between performance early in the learning process and the eventual level of ability achieved. Although adults may initially outstrip children at second language learning, people who begin learning in childhood reach higher eventual levels of ability than those who begin learning as adults. This general conclusion is also echoed by Aitchison (1989, p. 88) (see Johnson & Newport, 1989, p. 65, for references to relevant studies). Johnson and Newport develop the idea of a critical period by distinguishing between two distinct claims that might be made:

> *Version one: the exercise hypothesis.* Early in life, humans have a superior capacity for acquiring languages. If the capacity is not exercised during this time, it will disappear or decline with maturation. If the capacity if exercised, however, further language-learning abilities will remain intact throughout life.
>
> *Version two: the maturational state hypothesis.* Early in life, humans have a superior capacity for acquiring languages. This capacity disappears or declines with maturation. (Johnson & Newport, 1989, p. 64)

Johnson and Newport report on their own experiments, in which they studied the eventual levels of attainment of Chinese and Korean learners of English as a second language. Their results show:

> (1) Before age 15, and most particularly before age 10, there are very few individual differences in ultimate ability to learn language within any particular group; success in learning is almost entirely predicted by the age at which it begins.
>
> (2) For adults, later age of acquisition determines that one will not become native or near-native in a language; however, there are large individual variations in ultimate ability in the language, within the lowered range of performance. (Johnson & Newport, 1989, p. 81)

These results, they claim:

> support the maturational state hypothesis, and not the exercise hypothesis. Human beings appear to have a special capacity for acquiring language in childhood, regardless of whether the language is their first or second. (Johnson & Newport, 1989, p. 95)

Unfortunately, one cannot reason quite so easily from second language results to a critical period for first language acquisition. Johnson and Newport's results are compatible with a different hypothesis, which they do not consider, and which would not entail a critical period for first language acquisition. This could be called the "interference hypothesis", expressed as follows:

> *The interference hypothesis:* second language learning is (to some extent) inhibited by prior attainment in a first language.

This hypothesis would predict results such as Johnson and Newport's: if it were true, ultimate level of attainment in a second language would be inversely correlated with age of onset of learning the second language. Johnson and Newport's work gives an important pointer to the possibility of a critical period in first language acquisition, but it cannot yet be accepted as conclusive evidence until the interference hypothesis can be eliminated. Johnson and Newport do give statistics which suggest that:

> entirely nonmaturational explanations for the age effects would be difficult to support. Certainly the attitudinal variables (motivation, American identification, and self-consciousness) were unable to explain away the age effects, in accord with Oyama's (1978) study. This held true in the present data even when all three variables together were pitted against age. (Johnson & Newport, 1989, p. 91)

(Oyama (1976, 1978) also gives evidence for a sensitive period for the acquisition of the phonological system of a second language. And Marcotte and Morere (1990) state that their results "suggest the presence of a sensitive period in the first 3 years of life that parallels the myelinization of Broca's area (Milner, 1976) during which environmental deprivation associated with profound hearing loss alters the normal left hemispheric lateralization of speech" (Marcotte & Morere, 1990, p. 150).

To my knowledge, the evidence for a critical period for first language acquisition remains just that surveyed above. The evidence for a critical period for language acquisition in humans is probably no stronger than it is because of the ethical limitations on experimentation. The nature of the hypothesis means that the most compelling evidence comes from cases of deprivation up to and beyond the end of the period. Animal researchers can deafen sparrow chicks and sew up kittens' eyelids to get such evidence, but the human evidence can in general only come from circumstances which our society strives as strenuously as it can to avoid, such as those of Genie and Down's syndrome children.

A recent book (Singleton, 1989) devotes more space to discussion of the critical period for language acquisition than almost any work since Lenneberg's. Singleton argues that the evidence for such a critical period is not strong and that there is evidence against it. I agree to some extent with Singleton that the positive evidence is not strong, and for the same reasons as he mentions, in particular the impossibility of conducting crucial experiments on humans. Singleton also argues that there is evidence against the critical period hypothesis, that is, evidence of capacity for first language acquisition after puberty. But all the evidence he cites pertains either to a period not long after puberty, that is, late adolescence, or to aspects of language which are generally considered peripheral to basic competence in its structural core – aspects such as vocabulary, pragmatic skills, proficiency in writing the standard language, conceptual and logical reasoning, memory, reading, and even scholarly productivity.

Wachs and Gruen (1982, pp. 194–197) survey a range of evidence for critical periods in early human life in non-linguistic domains. Rovee-Collier and Lipsitt (1982, p. 76) also suggest critical periods for the acquisition of certain protective gestures early in life by human babies; they indicate a possible evolutionary explanation for this critical period phenomenon – the same general kind of explanation as will be proposed here for the case of language acquisition.

The general phenomenon of critical periods for learning is clearly established in a number of non-human species. Evidence includes: East Greenland Eskimo dogs learning the topography of their territories (Tinbergen, 1972, pp. 249–252); dogs' responses to humans (Freedman, King, & Elliot, 1961); song learning in chaffinches (Nottebohm, 1969) and in white-crowned sparrows (Marler, 1972, pp. 336–376); the visual functions of rhesus monkeys (Harwerth, Smith, Duncan, Crawford, & von Noorden, 1986); acquisition of binocular vision in monkeys (Blakemore, Garey, & Vital-Durand, 1978) and in cats (Wiesel and Hubel, 1963a, 1963b, 1965a, 1965b, Hubel and Wiesel, 1963, 1965); imprinting in ducks (Hess, 1973); socialization in dogs (Scott, 1978).

Nottebohm's (1984, pp. 69–72) survey of evidence for a critical period in the song learning of birds turns briefly to the question of explanation: "Why is there a critical period at all?" And, pertinently for my topic, Nottebohm presumes that it has something to do with "the way in which the learned song is used" which "presumably relate[s] to reproductive success". I now turn to this question of explanation, and invoke broadly similar considerations to those suggested by Nottebohm.

2. Theoretical background

I will demonstrate that, under certain plausible conditions, organisms with just the kind of sensitive period for language acquisition that we find in humans would tend to be selected by the evolutionary process. The method of demonstration used is computer modelling and simulation. A computer program was written in which the aspects of the relevant variables deemed crucial were represented and related to each other in plausible (though obviously extremely idealized) ways. Thus a population was set up, and the individuals in it were made to live, reproduce and die in regular ways. As the population turned over, simulated genetic characteristics of the individuals relating to the periods in their lives in which they are open to language acquisition were propagated through the population, and the individuals actually acquired language according to these genetic characteristics. The completeness with which they had acquired language at any given stage in their lives was related in various ways to their potential for reproduction or survival.

The computational models used make three crucial assumptions. The first is

that control of the aspects of language acquirable only (or with far greater ease) during the sensitive period conferred selective advantage on individuals. The second assumption is that the body of language controlled by an individual can be conceived as having a certain *quantity*; that is, languages have *size*. The third assumption is that life history traits such as puberty, menopause and various aspects of maturation and ageing are under genetic control, and that such genetic control of life history traits can extend to determination of periods in which language acquisition is more or less facilitated. These three assumptions are built into a computational model of evolution, which is then used to show how, under plausible, if idealized, conditions, the sensitive period phenomenon could be expected to emerge. These crucial assumptions and other relevant theoretical issues are discussed in subsections below; but first it will be useful to outline the broad strategy of the argument of this paper, which brings these assumptions together in the context of the critical period phenomenon.

Consider the following informal, pre-theoretical syllogism: possession of language is beneficial to an individual; therefore the longer the period of one's life that one possesses the whole of one's native language, the greater the overall benefit one will enjoy; therefore individuals with a capacity to acquire language early in life will tend to arise by natural selection. Experience shows that reactions to this simple argument are extremely diverse. Some scholars find it self-evidently, even trivially, true, and standing in no pressing need of elaboration or justification. Others find one or more of the "legs" of the argument too vague, not specific enough, or "speculative", so that, granted that there is a critical period for language acquisition, the proposed premises do not add up to a convincing explanation for it. Still others, more alert, whether or not they find the simple argument convincing or unduly speculative, notice that the argument gives no reason to expect the capacity to acquire a beneficial possession to actually *diminish* at any stage in life, as in fact happens with the critical period. The aim of this paper is to put each leg of the informal argument on much firmer ground, by discussion of what it means and how it can be justified, and then to define specific detailed versions of the premises with a degree of rigour and specificity that will make computational modelling of the envisaged evolutionary mechanism possible. In this way, it is hoped, some reservations about the vagueness and speculativeness of the original idea can be dispelled. And the puzzle about why the capacity to acquire language should diminish will be resolved in a clear way.

2.1. Possession of language conferred selective advantage

If certain aspects of linguistic competence and performance cannot be acquired (or can only be acquired with great difficulty) after puberty, then I shall assume that, at the relevant evolutionary stage in the past, an individual who controlled this knowledge and these skills had an advantage in life over one who did not,

and that this advantage was reflected specifically in the likelihood of the individual reproducing or surviving. The reservation about "the relevant evolutionary stage in the past", and the past tense in the heading of this subsection, are important. If we think in terms of modern urban humans, there is no obvious correlation of linguistic ability with reproductive success. Vining (1986) has thoroughly documented "evidence showing an inverse relationship between reproductive fitness and 'endowment' (i.e., wealth, success and measured aptitudes) in contemporary urbanized societies" (167). This is no surprise. A justification must be given for an assumption about a matter of fact (that possession of language conferred selective advantage), which Vining's modern evidence seems to go against.

Fox expresses concisely a point made by a number of peer commentators on Vining's article:

> The human organism is now trying to adapt to an environment wildly beyond the range of its environment of evolutionary adaptation (EEA). If it no longer seems to be acting in its own best interests this is scarcely surprising. (Fox, 1986, p. 192)

A class of genotypes ("the human organism") cannot actually act with intention or volition ("try"), so Fox's wording is strictly inaccurate, being one of the anthropomorphic metaphors common in this field, but his intended point is valid. The capacity to acquire language evolved in prehistory, over a period of time when bipedalism was a relatively recent development, and for which the specific modern hallmarks of success to which Vining refers (e.g., wealth) are scarcely conceivable. It is surely uncontroversial that the (capacity to acquire the) ability to communicate has brought advantage to humans. Chomsky speaks of the language faculty as "highly useful and very valuable for the perpetuation of the species and so on, a capacity that has obvious selectional value" (1982, pp. 18–19). Jerison writes:

> The socialized life of a predacious primate is so obviously benefited by linguistic skills, and language is so manifestly the peculiar human development, that changes in the brain to permit that advantageous supplement to perception and communication would have had obvious selective advantages throughout the period of hominid evolution. (Jerison, 1973, p. 405)

And Jacques Monod writes:

> A soon as a system of symbolic communication came into being, the individuals, or rather the groups best able to use it, acquired an advantage over others incomparably greater than any that a similar superiority of intelligence would have conferred on a species without language. (Monod, 1972, p. 126)

Pinker and Bloom (1990) devote several pages (their section 5.3) to arguments for the "reproductive advantages of better grammars". Possession of the language

faculty has enabled *Homo sapiens* to create the means (physical and/or social) for survival in almost all natural environments on the earth's surface. And this relation between language and survival presumably operated significantly at the level of the individual members of the species (which is not to deny the possibility of a significant group selection effect as well). The application of such a principle of usefulness in a putative explanatory model for an innate property of humans (here the sensitive period for language acquisition) demonstrates its power as an explanatory principle.

It is not actually feasible to quantify and represent in a graph the language abilities of individuals in a population, with the level of language ability on the *y*-axis, and numbers of individuals with these levels on the *x*-axis. But if such a graph could be drawn, for any stage in evolution, the curve would presumably be roughly bell-shaped. The modal level of ability would be near or at the mean, the bulk of the population would have levels of ability within two or three standard deviations of the mean, and there would be tails, occupied by small numbers of individuals, at both ends of the ability range. Even in modern times there are rare individuals with virtually no realizable language ability, and the highest modern level is presumably the highest there has ever been, so that the total modern range is wider than the total range at previous evolutionary stages. The gross evolution of the language faculty can be seen as the progressive change in the gross shape of the bell-shaped curve, certainly involving rightward shifts of the central hump and of the right-hand tail, while the left-hand tail has remained anchored at virtually zero language ability (though it may well have become attenuated). Much more speculative, and quite beyond discussion for the present, is the question of more detailed changes in the shape of the curve, in its possible narrowness or skewedness, and in the percentage of a population which at any time fell within, say, the central three-quarters, what might be considered the "normal" part, of the total range. In the early stages of the evolution of the language faculty it seems reasonable to assume that natural selection operated across the total extant range. Thus, mutant individuals capable of acquiring new, higher levels of ability would have been relatively strongly favoured, pushing the right-hand tail rightward; more average performers would also have enjoyed (dis)advantage in relation to their abilities; and the unfortunate individuals in the left-hand tail would have been correspondingly disadvantaged. Natural selection would have applied in this way until such time as more modern factors began to intervene, which may have been as late as the beginnings of urbanization.

The effects noted by Vining, that in modern urbanized societies the more socially successful individuals tend to have fewer offspring, is in fact hardly relevant to my long-term evolutionary argument. The obvious specifically modern factors which alter the relation between language ability and reproduction or survival include (a) a more caring attitude to the disabled, so that individuals who might earlier have been abandoned or marginalized are now helped to cope

and even to have families, and (b) the creation of extremely rich and complex material cultures, providing talented individuals with wholly new kinds of space in which to assert and demonstrate their superiority. The crude, blunt question "If you're so smart, why ain't you got more kids?" is not one that modern urbanized cultures accept as relevant to their standards of success. This is a big philosophical and political topic, but the following proposition bears scrutiny: modern man has taken his struggle with nature so far as to challenge natural selection itself. This is reflected, in modern life, at both tails of the bell-shaped curve; the innately disadvantaged are now helped, and the naturally advantaged now choose not to cash out their advantage in the Darwinian currency of survival and reproduction. This is to echo the essence of Fox's point, quoted above. Relevant also is Medawar's remark that "in the last seventy-five years, the whole pattern of incidence of selective forces on civilized human beings has altered ... We have already entered a new era in the biological history of the human race" (Medawar, 1952, p. 46).

I do not speculate very far about what the detailed mechanisms of a selective advantage conferred by language might be. They could involve the propositional function of language: an individual may be hampered (and thus its chances of survival diminished) by an inability to express certain classes of truth-conditional meaning, or at least to express them without difficulty. Or mechanisms relating language ability to reproductive potential might involve the more phatic aspects of language, so that an individual lacking certain aspects of the group's code was to some extent excluded from certain social relationships. There are two quite distinct kinds of theoretical consideration motivating a comparison of reproduction and survival as the vehicles of natural selection for the language faculty. The comparison is interesting for the theory of the functional origins of language. And the reproduction versus survival issue is also interesting, involving rather different considerations, from the point of view of evolutionary theory. I will first briefly discuss the linguistic considerations, and then turn even more briefly to the general evolutionary considerations.

Basing an argument, as I do here, on the premise that control of language confers selective advantage, one is under some obligation to say in what ways this might happen. In my experience, poeple who admit to being convinced of the general selective advantage conferred by language often still find it difficult to imagine specific real-life instances of the use of language which might be directly relevant to evolutionary considerations. Obviously, if one is able to understand the utterance "That rock is about to fall", one stands a better chance of avoiding being crushed and surviving to have offspring. And coitus between humans is typically preceded by verbal negotiations, often quite complex. The problem with these obvious connections between language and survival or reproduction is that such incidents are far from typical of our everyday uses of language. Modern humans can pass whole days, using language most of the time,

without a single utterance being either of this directly life-saving sort or an immediate prelude to sex. We use language to maintain our complex material culture (get the vacuum cleaner fixed, argue with the tax man, find out train times), and our web of social relations (tell friends when to come to dinner, phone mother, chat with a neighbour or colleague), and the vital concerns of reproduction and survival are seldom overtly at stake. But the fact that we do so much else with language should not lead us to forget that we still make crucial use of it to help each other survive and to negotiate collaboration in potentially reproductive acts. Imagine the paralysing effect if we were specifically *forbidden to utter anything potentially conducive, however indirectly*, to survival or reproduction.

Given, then, that modern use of language *is* used in the service of survival and reproduction, it is still in no way possible to isolate different components of the structure or use of language serving, however indirectly, these respective functions. (Perhaps the closest that any taxonomy of the functions of language gets to this is Halliday's (1973) distinction between (inter alia) "ideational" and "interpersonal" "macro-functions" of language. It might be superficially tempting to try to identify Halliday's interpersonal macro-function as primarily serving reproduction, and his ideational macro-function as primarily serving survival, but such an analogy in fact gets nowhere, even allowing the proposed connections to be very indirect.) The question "was it via survival or via reproduction?" arises in speculation about the origins of human language, and its possible relation to the communication systems of other species. Some species (e.g., honeybees, vervet monkeys) seem to communicate factual messages about their environments (e.g., there is nectar at such-and-such a distance in such-and-such a direction; there is a leopard (or eagle, or python) nearby). Such communication systems are advantageous presumably because of the direct effect they have on survival. Other animal communications, in particular mating signals, have been selected through their direct effect on reproduction. The question arises: which, if any, of these types of communication system reflects most closely the remote origins of human language? Speaking of the language faculty in Chomskian terms as a bodily organ, does its evolutionary prototype seem likely to have been primarily a reproductive organ, or primarily an organ aiding survival?

In fact, modern language gives absolutely no clue whether reproduction or survival was the main channel through which selection pressure for language operated, or indeed whether either was primary. This versatility in language is interesting from the point of view of evolutionary theory, showing how the evolution of language took humans into a qualitatively new evolutionary "ball-game". In standard evolutionary theory, survival and reproduction exert opposing pressures:

> Other things being equal, the greater the reproductive effort of an individual (that is, the larger the proportion of resources allocated to reproduction), the lower its chance of survival. (Charlesworth, 1990, p. 313)

> The balance which must be struck is between the competing benefits of (i) living longer by being better able to cope with random damage, and thereby being able to reproduce over a greater timespan, or (ii) reproducing at a greater rate. (Kirkwood and Holliday, 1986, pp. 6–7)

An animal's physical resources are finite, and survival and reproduction "compete" for them. But a language, once acquired, is not a resource that is depleted by use. Words are cheap: no matter how many words I use for one purpose, I still have as many left to use for other purposes as I had before. (The physical effort of talking is negligible.) So, where language as a resource is concerned, one would not expect pressures of reproduction and survival to compete with one another. That is, there is no reason to expect adaptation for reproductive function to tend to reduce adaptation for survival, or vice versa. The evolution of the capacity to acquire such a non-depletable resource, though it may conceivably have originated in one function or the other, would tend to maximize adaptation for both, at the expense of neither. This accounts for the versatility we see in modern language and perhaps explains why it is neither transparently solely an instrument of reproduction nor solely one of survival.

If one is theorizing, as I am, about the evolution of an aspect of the human language faculty, one should, for thoroughness, take into account any of the plausible channels through which selection pressure for language operated. To cover the theoretical possibilities adequately, the simulations to be described experimented with two different connections between language and selective advantage. In one condition, knowledge of an amount of language at a given stage in an individual's life was correlated directly with the probability of that individual reproducing at that stage. In the other condition, knowledge of an amount of language correlated directly (and inversely) with the probability of an individual dying before the end of its "natural" lifespan. These are two simple ways in which one can envisage language being correlated with selective advantage. In both conditions, a marked sensitive period effect emerged.

In trying to explain why the sensitive period for language acquisition coincides roughly with pre-puberty, one must avoid the strict adaptationist pitfall of assuming that this coincidence *must* be an adaptation to some particular need. One must bear in mind the pervasiveness of pleiotropy, the capacity of a gene to have a simultaneous influence on several, possibly many, characters. It is hard to find technical authors advocating particular adaptationist explanations without due caution and reservation about adaptation. Detailed, careful, and wise discussions of adaptation as a general issue are to be found in Williams (1966), Dawkins (1982, especially Ch. 3) and Sober (1984, especially Ch. 6); Pinker and Bloom

(1990) put the adaptationist debate, as it affects the evolution of language, into a balanced and sensible perspective. But the impression given by some popular authors is that the whole concept of adaptation is erroneous, and this impression has been disseminated in serious journals (e.g., by Piattelli-Palmarini, 1989). Here is not the place to enter into the methodological/philosophical debate over "adaptationism". Suffice it to say that the evolution of some traits *is* adaptive, moulded by natural selection, and the evolution of other traits is non-adaptive (but not particularly maladaptive). I claim that a beneficial effect which the capacity to acquire language conferred on its possessors was instrumental in the evolution of this capacity. This does not entail the extreme adaptationist view that every aspect of the language acquisition capacity, including specific aspects of its timing, must also be adaptive.

Clearly, though we are in a position to say "the critical period evolved", we are not in a position to say that the critical period effect confers any beneficial effect on the organisms which exhibit it. One can conceive of unusual circumstances (e.g., the "stroke" condition mentioned in the conclusion to this paper) in which it would be advantageous *not* to have lost one's language acquisition capacity; but I can think of no plausible circumstances in which it would be advantageous to lose, or to have lost, the capacity. The apparent puzzle can be resolved by avoiding premature judgement about what exactly is the phenomenon to be explained. To take an everyday case, the question is often put, "Why do animals need to sleep?" But this may be the wrong question to ask. Conceivably, the waking state is the "marked case", for which a better functional account in terms of selection pressures can be given. Similarly, in the case of the critical period, the question, "Why does the language acquisition capacity get switched off?" may also be a misleadingly phrased question, and a better question might be, "Why does a language acquisition capacity arise at all, and, given that it does, why is it concentrated in early, rather than late, life stages?" The model to be presented answers this latter question quite naturally.

In summary, the capacity to acquire language *is* an adaptation. It did not "just happen", but was helped to happen by selective pressure resulting from the enormous usefulness of language. But, given that the language acquisition capacity is an adaptation, the drop-off in language acquisition capacity associated with the critical period phenomenon need not be seen as an adaptation. I argue that the critical period effect "just happened", and was allowed to happen because of the lack of selective pressure to acquire (more) language (or to acquire it again) once it has been acquired. The central argument of this paper can only survive in the moderate mixed atmosphere between absurd extreme adaptationism ("everything is an adaptation") and its absurd opposite ("nothing is an adaptation"). More details will emerge in section 2.3, on polygenic inheritance, and in the account of the computer simulations.

2.2. Languages have size

Of course, it is quite beyond present-day linguistics to assign actual numbers to, say, my command of English. But the central psychological realist assumption of modern (generative) linguistics is that language users enjoy potentially infinite use of finite sets of representations stored in their finite brains. If these representations, or mental grammars, are finite, then in principle they can be assigned actual numbers indicating the amount of information they contain, even though in practice the determination of what the numbers should be is out of the question. The models investigated here depend on this quantifiability in principle of the linguistic knowledge that individuals possess.

Vocabulary size varies from language to language. And individual native speakers of the same language can control lexicons of markedly different sizes. But vocabulary is of somewhat less interest to us than grammar, as the capacity to acquire new vocabulary continues after puberty, albeit at a diminished level. So it must be argued that sets of partially or completely productive grammatical (phonological, morphological, syntactic, semantic) rules "have size". And a plausible concept of "size" which will be relevant and useful in this context must be identified.

All discussion of language acquisition in the generative framework, and much outside that framework, makes the tacit assumption that children proceed from knowing "some" of a language to knowing "all" of it. It is possible to identify (as Chomsky, 1969, did, for instance) parts of the language (i.e., rules) which are known by the child and parts which remain to be discovered. The final set of rules known is finite. The common reference to sequences of developmental stages in language acquisition, normal and abnormal, presupposes a general trend of increase in stored grammatical information. And, conversely, studies of aphasia typically speak of (partial) loss or impairment in terms which assume some kind of subtraction or deletion from a pre-existing finite store of information.

Internalized grammars, then, may have size. But the notion of language-size needed here has to take into account performance as well as competence. An internalized grammar exerts little or no causal effect on the world if its possessor is hampered by total non-functioning of input and output modalities. Without effective input and output modalities, an internalized grammar of any size is of hardly any more use than no grammar at all. (There might still be the possibility of language being used as some kind of purely internal representation as an aid in problem-solving.) The notion of language size that is needed in the present argument has to be something like "grammar size as mediated by the available devices for use", where devices for use are systems such as a speech production system, or a speech perception system. An alternative formulation might be "total linguistic resources at the disposal of the individual". Thus we are concerned with

the acquisition of a totality, comprising both knowledge (linguistic competence) and skills (involved in linguistic performance). Although vague, the notion of such a totality seems clearly to correspond to something real. Young children have less of it, aphasics may lose some of it (from either the knowledge or the skills), and then perhaps regain some of what they have lost. And our remote ancestors had less of it than we have; the more remote the ancestors, the greater the discrepancy between their total disposable linguistic resources and those of the normal modern adult human. In the research reported here, I assume that the total linguistic resources available to individuals in a community can vary upward or downward as evolution progresses. But I moderate this variable-language-size assumption by placing a notional upper limit on language size. The postulation of such an upper limit requires some justification.

It seems that normal children have some *spare capacity* for language acquisition. That is, they can acquire their language, and perhaps even one or two other languages, in good time before their language acquisition capacity has run out. A child may, for instance, acquire as much as she will ever acquire of English by the time she is 8 years old, then suffer brain damage with some resulting language impairment, and still have capacity to recover the loss completely. But if she had suffered no brain damage, her language, being essentially complete, would have remained at the same level after her eighth year. The spare capacity used to recover loss after impairment is not used, in the absence of impairment, to "push further ahead". In other words, the size of languages fits comfortably inside the typical child's capacity for language acquisition. Many normal children stop acquiring the core components of their first language before the end of the sensitive period, because, in some sense, they have acquired all that there is to acquire. This is apparently not merely a result of the adult exemplar being limited, with children not acquiring competence beyond the exemplars presented to them. Goldin-Meadow (1979) documents deaf children's creation of language-like systems in the absence of conventional language models. The rise of creoles from pidgins (Bickerton, 1981; Givon 1979, p. 224) also shows that children can, at lower levels, create language well beyond their adult exemplars. (This role of child language acquisition in creolization is accepted by creolists who do not share Bickerton's and Givon's views on the dramatic speed of the process.) But at higher levels, that is with a variety more elaborate than a pidgin as a model, language acquisition seems to meet some kind of natural limit.

It is difficult to see clearly what principles govern this maximum language size, beyond which language acquirers do not proceed, even though they have spare capacity. One possible explanation could be the existence of an abstract limit, very broadly in the same way as the theory of Turing machines and recursive function theory mark an upper bound, given Church's thesis (Church, 1935), to the informal intuitive notion of "effective procedure". This suggestion, when adequately fleshed out, may need to call on results of learn-ability theory (e.g.,

Wexler & Culicover, 1980), or perhaps on some theory of communication and possible message, appropriately framed to suit the case of human language (e.g., digital, rather than analogue, representation).

2.3. Polygenic inheritance of characters related to language

Of the aspects of the simulations described in this paper, probably the one requiring most detailed comment is polygenic inheritance, which is in a sense the converse of pleiotropy, mentioned earlier. Since the explanation proposed here for the critical period rests centrally on a mechanism of genetic inheritance, it is essential that this aspect of the simulations should be modelled reasonably closely to what we know of the reality of the relation between genes and language.

Spuhler (1979) concisely summarizes the relevant facts:

> The living organism is not merely a mosaic of "unit characters", each representing an anatomically distinct part of the body, and each determined by a special gene. Pleiotropism or manifold effects are characteristic of most major genes. It is convenient to name major genes for an easily observed unit character, preferably an early gene product, but the named character may represent only a small sector of the total range of characters affected by the gene.
>
> Studies on transmission genetics in family lines and twin pairs show that a large number of major genes do in fact modify speech and language function. Thus far, none of these have been named "genes for language" as such. Every gene that includes a manifold effect involving the normal structure and function of the central language system in the brain could be called a "gene for language" ... [but] it is better to identify the locus by the enzyme or primary phenotype.
>
> The fourth edition (1975) of McKusick's catalogue of known human major genes includes 1218 autosomal dominants, 947 autosomal recessives, and 171 X-linked genes, a total of 2336. Hundreds of these genes are known to be relevant to normal development of speech and language. As expected, more major genes are known to affect the four peripheral language modalities than the central language system. (Spuhler, 1979, pp. 29–30)

Some specific, more recent developments are mentioned by Pinker and Bloom (1990):

> Bever, Carrithers, Cowart, and Townsend [in press] have extensive experimental data showing that right-handers with a family history of left-handedness show less reliance on syntactic analysis and more reliance on lexical association than do people without such a genetic background.
>
> Moreover, beyond the "normal" range there are documented genetically transmitted syndromes of grammatical deficits. Lenneberg (1967) notes that specific language disability is a dominant partially sex-linked trait with almost complete penetrance (see also Ludlow & Cooper, 1983, for a literature review). More strikingly Gopnik (1989) has found a familial selective deficit in the use of morphological features (gender, number, tense, etc.) that acts as if it is controlled by a dominant gene. (Pinker and Bloom, 1990, pp. 26–27)

(On this last item, see now Gopnik, 1990a, 1990b; Fletcher, 1990; Vargha-Khadem and Passingham, 1990.)

In the simulations reported here, the time profile of an individual's innate language acquisition programme is treated as a set of variable characters, each character pertaining to some particular stage, or slice, of the individual's life-history. Each such character is determined by many genes, and the same genes often contribute to the variation of different characters; so there is simulated polygenic inheritance and pleiotropy. An individual time profile for language acquisition may be visualized as a graph of language-learning capacity (on the y-axis) against age (on the x-axis). The highest points on the curve correspond to the periods in life when the individual's language-acquisition capacity is at its greatest. Where the total area under the curve is not greater than the notional maximum language size, this area represents the total amount of language acquirable during the individual's lifetime. If the total area under the curve comes to more than the maximum language size, the area represents the total amount of language that the individual could in principle acquire, lose and regain throughout life, if suffering repeated impairments. Individuals inherit the genes determining such profiles from their parents, some coming from the mother, some from the father.

I take as a starting point a straight-line horizontal graph, coinciding with the x-axis, representing a uniform lack of capacity for language acquisition throughout life. The simulated populations all start with individuals characterized by such perfectly level, and empty, language acquisition profiles. What is to be explained is how human populations end up with innate profiles radically skewed from this flat initial shape towards a marked peak in the years before puberty.

I assume that mutations can produce small effects in individuals' language acquisition profiles, either increasing or decreasing the amount of language that can be acquired at a particular stage in life, so that humps and hollows may appear in the evolving profiles. But, since the determination of the overall profile is polygenic, a hump in a parent's profile is not necessarily passed on whole to the offspring, but tends to be diluted by the contribution of the other parent. Only if both parents contribute a hump in the same place in the profile is the offspring likely to inherit "the whole hump". Polygenic inheritance has a dampening effect, tending to keep characters close to some measure of central location, in the absence of selection pressures.

The relevant measure of central location hypothesized here is zero language acquisition capacity, for all stages of life. This represents a perfect balance between inhibiting (or "negative") and facilitating ("positive") factors, throughout life. If, for some stage in life, an individual has more facilitating than inhibiting genes, this individual will have the capacity, during that lifestage, to acquire a certain amount of language. If this individual's mate is similarly positively endowed, for the same lifestage, their offspring is likely to inherit a similar capacity. But if one parent has more facilitating genes and the other has more inhibiting

genes for language acquisition at some lifestage, their offspring is likely to inherit a decreased, possibly zero, language acquisition capacity for that lifestage. There are no phenotypic effects of differences between various strengths of "negative" endowment; the language acquisition capacity in all such cases is zero. But the descendants of "strongly negative" individuals are further from evolving to positive values than descendants of less strongly negative individuals. The present research investigates the effect of plausible selection pressures on the evolution of such positive and negative balances of language acquisition capacity, for various stages in life.

The existence of genes which are "inhibiting" in relation to a specific character needs to be seen in the wider context of all other characters possessed by the organism. For example, adaptations which facilitate one form of locomotion (walking, swimming, flying) often inhibit the others; the best runners among the animals are poor (or non-) flyers and swimmers, and so on. Bearing in mind that the polygenic character we are concerned with here is the capacity to acquire the "total linguistic resources at the disposal of the individual", including both competence and performance resources, it is quite conceivable that adaptations could arise which are beneficial in some non-linguistic domain (such as eating or breathing), but actually disadvantageous in the linguistic domain. The prevalence of pleiotropy makes this all the more probable.

To make this discussion more concrete, I give an example of a conceivable, if implausible, language acquisition profile in a histogram in Figure 1, with a hypothesized ten lifestages, and a hypothesized maximum language size of ten 'units'.

An individual with the profile in Figure 1 would be capable of acquiring up to 70% of the notional whole language during its lifetime, but the period of most active acquisition would be in mid-life. This 70% would be composed of 10% during the second stage of life, 30% during the fourth lifestage, and so on. The total shaded area in such a histogram represents the total amount of language acquirable by an individual in its lifetime. The profile in Figure 1 is notably spiky, an implausible feature, as one would expect genes relevant to several lifestages to affect adjacent stages, making language acquisition capacity in contiguous lifestages somewhat similar, and thus preventing such marked spikiness in profiles. The task of this paper is to explain why a different class of profiles, in which acquisition of language is concentrated largely or even wholly in the first two (pre-puberty) stages of life, has actually evolved. Such profiles are illustrated in Figure 2.

The precise method of modelling the influence of genes on language acquisition at various lifestages (fuller details of which will be given later – see Figure 3) must not give rise to a circular argument. The hypothesized starting point for the evolutionary process should ideally belong to a large possibility space of genetic dispositions to age-related language acquisition capacity, and should be

Figure 1. *Hypothetical language acquisition profile.*

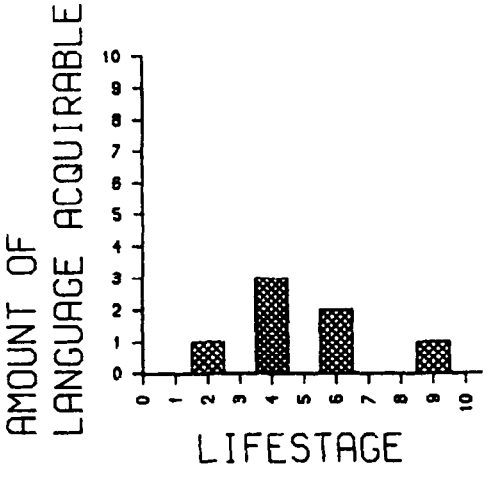
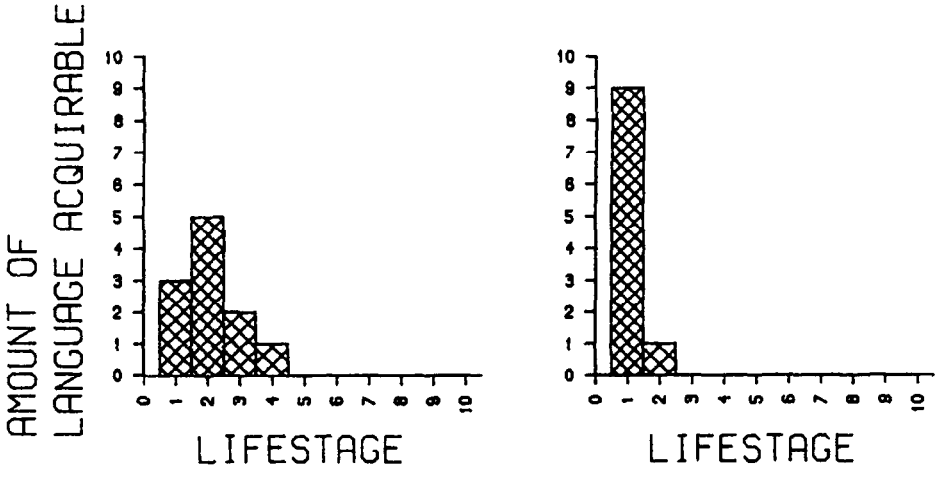


Figure 2. *Language acquisition profiles which would show a critical period effect.*



chosen to be as neutral as possible. The hypothetical initial gene pool must contain no hint of a critical period effect, but clearly, in order for an explanation to be possible, the model must make available mechanisms by which the gene pool can (but not must) evolve toward the state to be explained. It will be shown that the addition of an element of selection pressure is what pushes the gene pool in the direction of the state to be explained. But without such pressure, the method for modelling the genetics of the timing of language acquisition is in no way biased in favour of a genotype exhibiting a critical period effect.

It may be helpful to compare the critical period for language acquisition with Huntington's chorea, a hereditary disease whose onset is age-related. As Medawar points out:

> Its disabling and clinically important effects first become manifest not in youth or old age but at an intermediate period, its time of onset – later in men than in women – being most commonly in the age-group 35–39. Its age of onset does however vary, and I want you to assume (what is almost certainly true, though it would be hard to collect the evidence for it) that its age of onset, like the disease itself, is also genetically determined. (Medawar, 1952, p. 66)

Medawar proposes an argument, now widely accepted, that natural selection must act so as to postpone the onset of Huntington's chorea. My argument concerning the critical period has exactly the same logic as his, but deals with an advantageous genetic effect, rather than a disadvantageous one, and so the evolutionary trend is in the reverse direction. The strength of Medawar's argument lies just in the fact that it assumes very clearly that genes determining onset of Huntington's chorea during childhood could exist, and then proceeds to explain why they do not spread through the population; according to his now classical argument, these genes have been weeded out by natural selection. The underlying assumption made here is crisply stated as "Every period of the human lifespan is a product of selection" (Scarr-Salapatek, 1976, p. 171); this is not an isolated view; it echoes similar aphorisms by Mayr (1970, p. 84) and Freedman (1967, p. 489), and is a basic assumption of surveys of life-history evolution studies, such as Partridge and Harvey (1988). I would, however, add as a note of caution that, to avoid an extreme adaptationist interpretation, one might better say that every period of the human lifespan is a product *or by-product* of selection.

In the section below, I first describe the details of my simulations pertaining to the sensitive period hypothesis, and then present the results.

3. The simulations

3.1. The simulated individuals

A population of 30 individuals was set up. Thirty is roughly the number of individuals in many primitive human hunter–gatherer groups, and in many troops of primates. The structure of each individual is a sextuple as in (1):

(1) [Name, Stage, Language, ParentID, Dominant-LAP, Recessive-LAP]

The functions of the elements of these sextuples are described in turn below.

"Name". This is simply for programming convenience in keeping track of individuals during the simulation, and has no theoretical significance.

"*Stage*". There are ten successive stages in an individual's life from simulated birth to simulated death in old age, expressed as the integers 1–10. With each cycle: individuals already at stage 10 "die"; that is, they are removed from the simulation; a corresponding number of "newborn" individuals are created and enter stage 1; and individuals previously at stages 1–9 move on to stages 2–10. In one simulation condition (the "survival" condition), up to three further, "premature" deaths occur to individuals at stages before the tenth stage, and these individuals are also replaced by newborns. Thus the entire population is replaced once every ten cycles, and in one condition can be replaced more frequently than this. Ten cycles is termed one "generation", though perhaps "lifetime" would be a better term, as it is quite possible for five successive generations (from ego to great-great-grandmother) to be all still alive at the same point in a simulation. The first two stages of life are designated as pre-puberty: individuals at either of these two stages cannot be nominated as parents in the cyclic regeneration process. These numbers roughly mirror the human and some primate cases. Humans (without benefit of modern medicine) may live to about 60, and puberty is at about 12 years. Chimpanzees may live to about 40, and chimpanzee puberty is at about 8 years.

"*Language*". This is the total disposable linguistic resource (knowledge of rules, ability to use them, etc.) available to an individual, expressed as a positive number (including 0). Zero represents complete lack of language. The higher the number, the more language is attributed to the individual. A theoretical maximum is set to language size. The maximum number represents complete command of "the language", whose "size" is thus notionally set at this maximum number. It might help conceptualization of what is being simulated to identify the maximum value with the "evolutionary target" of a body of linguistic knowledge and abilities comparable in size with a modern human language. The choice of a particular number as maximum is not significant, but determined by programming convenience; the number used in these simulations was 10 "units".

"*ParentID*". This records the name of one of an individual's parents. This information is used in one of the simulated conditions (the "mother" condition), where an individual acquires its language through its lifetime only from a single designated parent (as long as that parent is alive). In another simulated condition (The "wholepop" condition), an individual acquires language from one parent for the first stage of life, but from the whole adult population thereafter.

"*Dominant-LAP*". This represents the set of genes determining the time profile of an individual's innate language acquisition programme. In these simulations it takes the form of a list, consisting entirely of +1's and −1's. An entry of +1 at a particular place in the list represents an allele with a facilitating effect on

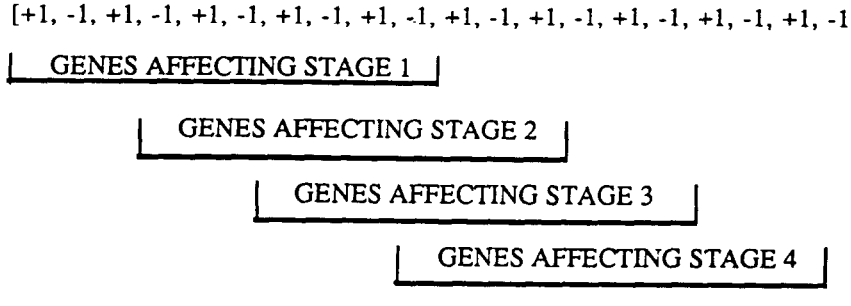
language acquisition: a −1 represents an allele with an inhibiting effect. Language acquisition at each of the ten lifestages is affected by a fixed number of genes; the number was set at ten in these simulations, modelling substantial polygeny. And there is "overlap" between the genes affecting one lifestage and those affecting adjacent lifestages; the degree of this overlap was set at seven, modelling a substantial degree of pleiotropy. The structure and its use is schematically illustrated in Figure 3. Such a scheme relating genes to a developmental profile is consistent with the kind of picture drawn by Harwerth et al. (1986, p. 235) of their findings on rhesus monkeys: "there are multiple, partially overlapping sensitive periods of development and the sensitive period for each specific visual function is probably different".

From its genes affecting language acquisition at some lifestage, an individual's capacity for language acquisition during that stage is determined by adding their numerical values. Thus a preponderance of +1's will yield a positive capacity, in proportion to the number of +1's, and a preponderance of −1's will yield zero capacity. If all ten genes affecting some lifestage should happen to be +1, the individual would have a capacity to acquire 10 "units of language" during that stage; 6 +1's and 4 −1's gives a capacity to acquire 2 units; 4 +1's and 6 −1's gives zero capacity, and so on. This treatment is in tune with Charlesworth's (1980) conclusion:

> it is, of course, likely that life-history evolution involves variability at a great number of loci. Provided that gene effects are approximately additive across loci and that linkage is loose, the results for multi-locus situations can be obtained by simple addition of the contributions to phenotypic change from individual loci (Falconer, 1960; Crow & Kimura, 1970). (Charlesworth, 1980, p. 205)

Postulating a simple distinction between language-inhibiting and language-facilitating alleles is a simplifying idealization. The real situation could well be more complex, with a negative gene inhibiting not language but one kind of rule or mode or processing while facilitating another kind. For this first attempt at an

Figure 3. *Schematic model of language acquisition capacity at various lifestages treated as overlapping polygenic traits.*



evolutionary explanation of the critical period, it would have been too complicated and too speculative to build a model with such properties. Incidentally, one should of course resist any temptation to interpret the list of genes in Figure 3 as physically contiguous genes on a chromosome: the genes affecting language acquisition could be located anywhere in the genome; the hypothesized relevant genes are artificially assembled together into this computational data structure merely for convenience in simulating their overlapping functional effects.

Given that genes determine development, and that development slows down and may even stop during an individual's life, it is actually somewhat implausible to posit such an even effect of genes on language acquisition capacity at all lifestages. Probably, definite events in life history are more under genetic control at early lifestages than at later lifestages (although menopause shows how long the arm of biological management can be). Kirkwood and Holliday refer to a

> principle originally pointed out by Haldane (1941), and later clearly enunciated by Medawar (1952), which asserts that the force of natural selection reduces progressively with age. The reason for this is that selection on genes acting early in life will affect a greater proportion of individuals than genes acting late, when the proportion of survivors will be smaller and the remaining fraction of their lifetime expectation of reproduction will be less (see also Williams, 1957; Hamilton, 1966; Edney & Gill, 1968; Kirkwood & Holliday, 1979; Charlesworth, 1980). (Kirkwood & Holliday, 1986, pp. 4–5)

The uniform scheme in Figure 3 was chosen, however, because it represents an unbiased null hypothesis with respect to the major claim made in this paper. That is, we see language acquisition concentrated in a particular period of life, and want an answer to the question, "Why this period, and not some other period(s) in life?" We should pose this question within a framework which does not automatically bias the answer towards a picture of eventful early years, followed by monotonous later life. So the scheme in Figure 3 was adopted, representing all lifestages as within equal reach of the managing influence of the genes. Probably a more realistic assignment of genetic influence to different lifestages would have been more biased in this respect; but I wanted to avoid the risk of apparent "rigging" in these simulations, and settled for the framework in Figure 3, which forces my main argument to prove itself "against the odds".

At regular intervals (every 30 births) during the simulations, the Dominant-LAP of a newborn individual underwent a simulated mutation. The form of this mutation was the switching of the value of a randomly chosen allele; an original +1 mutated to −1, and vice versa. Thus, over time, the original genotype common to all individuals became diversified, subject to selection pressures and normal random genetic drift, and Dominant-LAPs other than the initial very regular one shown in Figure 3 occurred.

At this point, a reasonable query would be: "The initial populations are as-

sumed to have zero knowledge of language, but of course they have language-facilitating genes. How did these protohumans acquire such genes in the first place (and thus differ from apes or the common ancestor)?" Presumably, the quite remote ancestors of humans had no language-facilitating genes (in principle recognizable as such) at all. The evolution of the human genome must have involved changes which added new loci, as well as introducing new alleles at existing loci. The first language-facilitating genes to arise may have had no phenotypic effect – they may have been pre-adaptations, by-products of other evolutionary changes, happening, by chance rather than selection, to lay a basis for the later adaptive emergence of the language faculty. The evolution of the vocal tract clearly went through various such preliminary stages that paved the way for modern human speech. In fact we cannot be sure that the apes do not also possess at least something of such a foundation, even though they have not undergone the later mutations that affected humans. All we know is that there must have been some pre-adaptive platform from which the evolution of the language acquisition device took off. The schema in Figure 3 is an attempt to represent this platform, in as neutral a way as possible with respect to the explanandum under consideration, namely the innate timing in life-history terms of the period during which the acquisition device is disposed to be most active.

"*Recessive-LAP*". This has the same form as the Dominant-LAP. It has no effect on the individual's language acquisition (i.e., its developmental–behavioural phenotype), but aspects of it may be passed on to its offspring as aspects of the offspring's Dominant-LAP and Recessive-LAP, depending on random choices. Sexual reproduction is simulated somewhat closely. On nomination of an individual as a parent, a simulated gamete is produced by making a random choice between an element of the Dominant-LAP and the corresponding element in the Recessive-LAP, for each position in the list of genes. Thus parents produce gametes reflecting the composition of their whole genotypes, not just their phenotypes. Conception is simulated by forming a new Dominant-LAP/Recessive-LAP pair from the gametes of two nominated parents. Depending on the random toss of a computational coin, the new individual's dominant allele for one locus comes from the sperm and its recessive allele from the ovum, or vice versa. (Of course, the alleles inherited from both parents will often be the same.)

The labels "Dominant-LAP" and "Recessive-LAP" are possibly slightly misleading. Given two different alleles contributed to a fertilized ovum at the same locus, the choice of which allele becomes the "Dominant-LAP" of the newborn individual and which the "Recessive-LAP" is, in these simulations, a matter of random (50/50) choice on each occasion of simulated conception. It is not the case that a particular allele always dominates over another, as in reality the brown-eyes allele dominates over the blue-eyes allele. This treatment was necessary to avoid any suggestion of "rigging" of the simulations in favour of either

language-enhancing or language-inhibiting genes, bearing in mind that we know nothing in any detail about language-related genes. Perhaps "Phenotype-LAP" and "Cryptotype-LAP" might have been more appropriate labels, but I avoided these because of the relative unfamiliarity of the term "cryptotype".

3.2. The simulated evolutionary cycle

At the start of the simulation, the 30 individuals are distributed equally over the ten lifestages, with three individuals at each lifestage. Each cycle consists of two basic operations, the nomination of a set of parents, and the subsequent turnover of the population, involving between three and six deaths and the corresponding new births.

3.2.1. Nomination of parents

Parents are nominated from the whole adult population (i.e., from all individuals in any of the stages 3–10). The choice of individuals as parents is random, but may be weighted in favour of individuals with higher language scores, depending on the experimental condition. In the "Survival" condition, each adult individual has an equal probability, at each cycle in the simulation, of being nominated as a parent. In the "Reproduction" condition, the probability of being nominated as a parent, on any cycle, is proportional to the *cube* of an individual's language score. As between three and six individuals "die" on each cycle, between six and 12 nominations of parents are made each cycle. The simulation does not go to the trouble of distinguishing male and female individuals, but does in fact prevent an individual from being a parent more than once in any cycle. The rule that an individual cannot be a parent more than once in one cycle would be realistic for females, if not for males. It has the consequence of, probably somewhat unrealistically, dampening down any selective advantage mediated by rate of reproduction, since an individual male with a head-and-shoulders advantage over others might in reality sometimes expect to beget several offspring in a short space of time.

3.2.2. Turnover of population

With each cycle, the individuals at stage 10 are removed from the simulation and replaced by new individuals at stage 1. In one experimental condition, the "Survival" condition, up to three further individuals are killed off by "accidents". The probability of dying from such an accident is calculated to be proportional to the individual's age and inversely proportional to its language score – more details will be given below. Individuals dying from accidents are also replaced by new individuals at stage 1. So the population size remains constant over time – a simplifying assumption. The Dominant-LAP and Recessive-LAP of each new individual are determined by random processes from the Dominant-LAPs and

Recessive-LAPs of a pair of nominated parents. All remaining individuals at stages 1–9 are aged a stage further to stages 2–10. All individuals are credited with an update to their language score, the resulting new score being determined by the individual's (new) lifestage and an exemplar, which, depending on the condition simulated, can either be one of the individual's parents or the whole adult population. The rules for acquiring language are given below.

Individuals can never acquire more language than their Dominant-LAP permits for their particular stage in life, no matter how rich the exemplar to which they are exposed. Thus, for an individual at lifestage n, if its capacity $C(n)$ for acquiring language during this stage is less than the number representing the language score of the exemplar to which it is exposed, then the language it acquires during that stage is limited to $C(n)$. The individual's accumulated language score at this stage is then $C(n)$ plus whatever it had previously acquired, up to the notional maximum language size. If, on the other hand, $C(n)$ is greater than the language assigned to the exemplar, then the language acquired during that stage is set at the mean of the two numbers, that is, halfway between what it is capable of at this stage and the exemplar it is given. This quantifies (arbitrarily of course) the degree to which appropriately equipped individuals can go beyond the data to which they are exposed: it simulates a degree and kind of inventiveness in language acquisition, still restrained in some way by the richness or poverty of the input data. At the beginning of all simulations, all individuals were assigned a "Language" value of exactly 0; that is, the initial populations were assumed to have zero language.

The proposed model in no way relies on there being any large discrepancy between the "size" of an individual's capacity for language acquisition and the "size" of the language to which he is exposed. In fact this model effectively precludes the possibility of a language of modern proportions being modelled to a child with an unevolved (though normal for that evolutionary period) capacity of much smaller size. This is because the population's gene pool remains fairly homogeneous, despite the crucial variation fuelling the evolutionary process, and it is unlikely in the extreme that an individual with so low a language acquisition capacity could be born into a population with a far higher capacity, as demonstrated by its actual acquisition performance. Similarly, a large difference in the other direction, with an "extreme genius" child being born into a population of very low achievers, is extremely unlikely to arise, again because of the relative homogeneity of the population. It *is* at the heart of this model that mutant newborns can have a language acquisition capacity in excess of the "size" of the extant language of the community they are born into. But given the large number of relevant genes postulated, a single mutation has a relatively very small effect. It is the sum of such small effects, accumulated over the generations that, as I will argue, amounts to the evolution of the critical period. In these simulations, at any given stage, the size of the community's language is closely related to the

genetic capacities of its members, and over time the language size and the capacities stay roughly in step, though, by the nature of the model, with the genetic capacities always tending to be just a shade ahead.

3.3. Variables in the simulations

A 2 × 2 table (Table 1) shows the range of possibilities simulated. The Reproduction and Survival conditions differ as shown in Table 2, where S represents life-stage ($1 \leqslant S \leqslant 10$) and L represents language ($0 \leqslant L \leqslant 10$). Informally, at any given point in the simulation, in the reproduction condition, better linguistic performers are more likely to be parents; and in the survival condition, better linguistic performers are less likely to die before their allotted span, although all become more vulnerable with increasing age.

The device of correlating a power (here the cube) of language score (or its inverse) with reproduction or survival is a way of exaggerating the differences between individuals, while maintaining the ranking of their respective "scores". The technique is analogous to that of a physical scientist turning the force of gravity simulated in a centrifuge up to several *g*. With a weaker relationship between language and reproduction or survival, normal random genetic drift would have exerted greater influence, and simulations would have taken longer to move towards their attractor states. Nevertheless, the same general trends would presumably have been discoverable, but by means of longer simulations.

A further simplifying idealization postulated no correlation (inverse or otherwise) between adult age and likelihood of becoming a parent at the next cycle in the simulation. This assumption may be approximately appropriate for males,

Table 1. *The four experimentally simulated conditions*

	Acquisition exemplar	
Language correlated with:	Mother	Wholepop
Reproduction		
Survival		

Table 2. *The relation between the "reproduction" and "survival conditions"*

	Reproduction condition	Survival condition
Probability of adult being parent	Proportional to L^3	Same (= chance) for all
Probability of dying before stage 10	Same (= 0) for all	Proportional to $(S/L)^3$

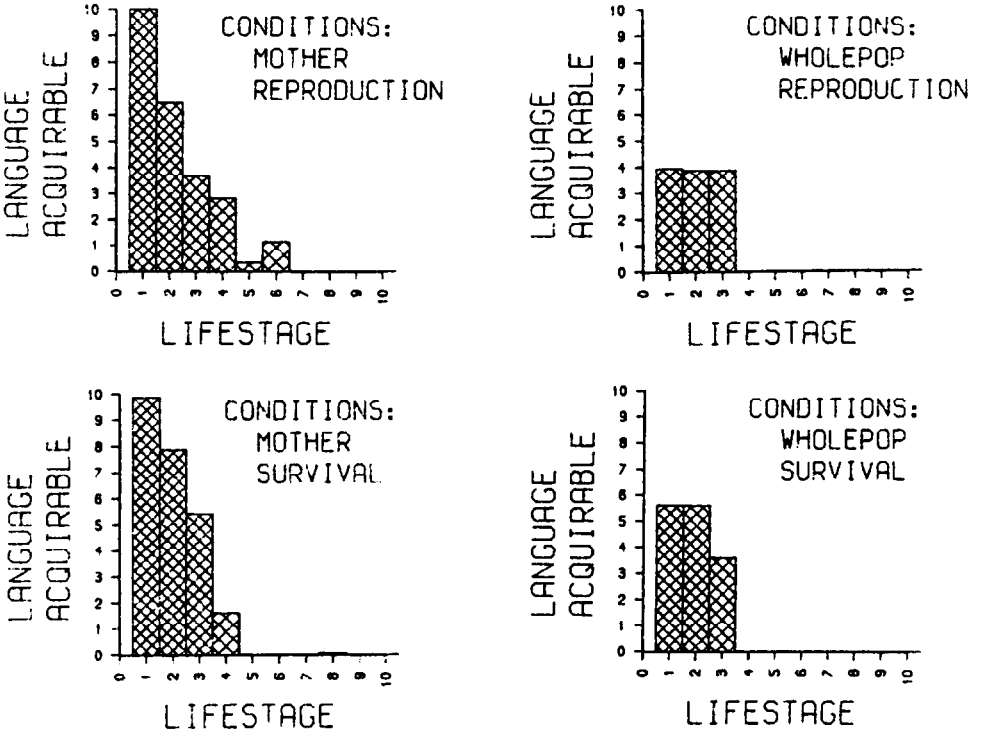
but it is certainly not for females: it might have been more realistic to simulate a female menopause after, say, six lifestages. The simplifying idealization involved in allowing females to reproduce until their last life-stage does not, however, bias the simulations in any undesirable way by predisposing the model to produce the very result which is to be explained. On the contrary, thoughtful comparison of the two extreme possibilities (earlyish menopause versus no menopause) will show that the former possibility, rather than the latter, would tend to exert greater selection pressure in favour of early language acquisition. Individuals who are out of the game earlier in life, as postmenopausal women are, have correspondingly less time in their lives to reap the benefits of possessing language; thus any delay, after reproduction becomes possible, in acquiring this possession is correspondingly somewhat more disadvantageous. A woman who, say, does not acquire the last instalment of her language until the lifestage immediately after puberty, and who undergoes menopause after the sixth lifestage, spends one-fifth of her reproductive life at below the optimum level of linguistic achievement. However, a woman with the same language acquisition history but who does not undergo menopause spends a smaller proportion, one-eighth, of her reproductive life at below optimum level. These specific fractions assume in both cases survival until the tenth lifestage; earlier death would further enhance the difference between the two cases, and hence increase the selection pressure to acquire language early in women who undergo menopause, relative to those (hypothetical) women who do not.

(In fact, in present-day language acquisition, there is probably a very slight superiority of girls over boys, as reported and discussed by Dale (1976, pp. 309–312) and Wells (1979, pp. 383–385). It is beyond the scope of this paper to discuss whether the fact that women are reproductively viable for less of their lives than men might contribute to an evolutionary explanation of this slight superiority; but it may be noted that what male–female difference there appears to be is in the direction which would be predicted from the general model adopted here.)

The "mother" and "wholepop" acquisition exemplar conditions differ in the exemplars used by individuals in acquiring language. In the "mother" condition, the exemplar for an individual's acquisition of language is one of its parents (chosen randomly at birth, and here conventionally referred to as the "mother"). As this mother individual acquires more language, the off-spring is capable, subject to the limitations imposed by its Dominant-LAP, of acquiring more. But once the mother dies, the offspring's language can increase no further.

In the "wholepop" condition, a newborn acquires language for the first stage of life from the exemplar of one parent, but thereafter uses the mean language score of the adult population as an exemplar. In this condition, since there are always adults present in the population, an individual's acquisition exemplar does not disappear during its lifetime, as happens under the "mother" condition.

Figure 4. *Results of the simulations in the four combinations of conditions after 1000 generations.*

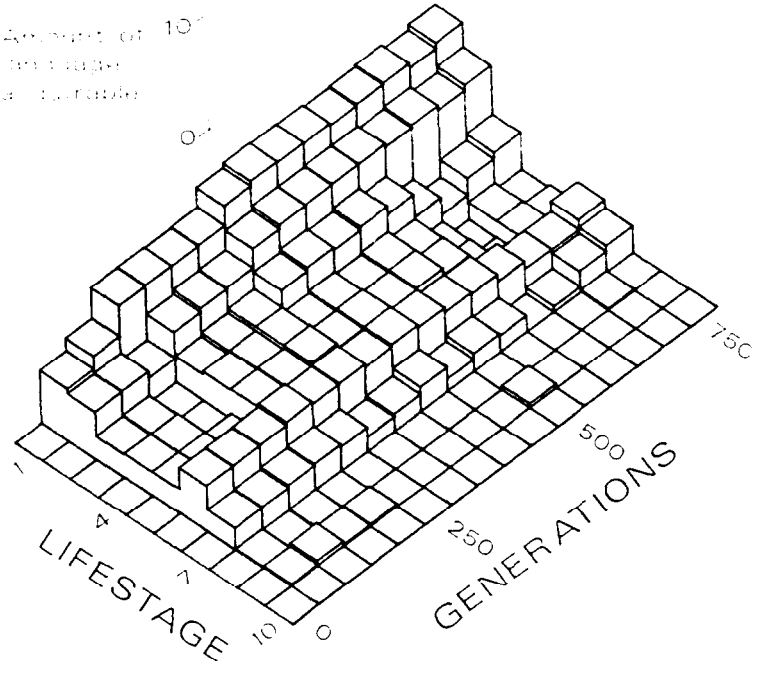


3.4. Results

Each of the four simulations mentioned in Table 1 above was run for many simulated generations. I present in Figure 4 histograms representing the mean language acquisition profiles of the whole population after 1000 generations, for each of the four combinations of conditions.

To aid visualization of the dynamic evolutionary process simulated, two further diagrams (Figures 5 and 6) are presented, in which histograms are stacked behind each other, giving a kind of skyscraper landscape. This landscape represents a succession of stages in the simulated evolution of the population's mean language acquisition profile from a flat zero profile at the beginning (0 generations) toward a mean language acquisition profile showing a marked critical period effect after 750 generations. The successive stages in these diagrams (slices of the "skyscraperscape") are snapshot histograms of the situation every 50 generations. From such diagrams, one can gain an idea of the appearance and gradual invasion

Figure 5. *Evolution of the critical period (in the "mother/reproduction" condition).*



of the population, over the generations, of genes favouring early language acquisition.

In all simulations, once a critical period effect had evolved, it remained stably present. That is, the tallest columns in the population's mean language acquisition profile were always at a very early lifestage, usually the two pre-puberty lifestages. But from time to time other lower humps at later lifestages appeared and disappeared over the generations. Some examples can be seen in the dynamic ("landscape") diagrams Figures 5 and 6.

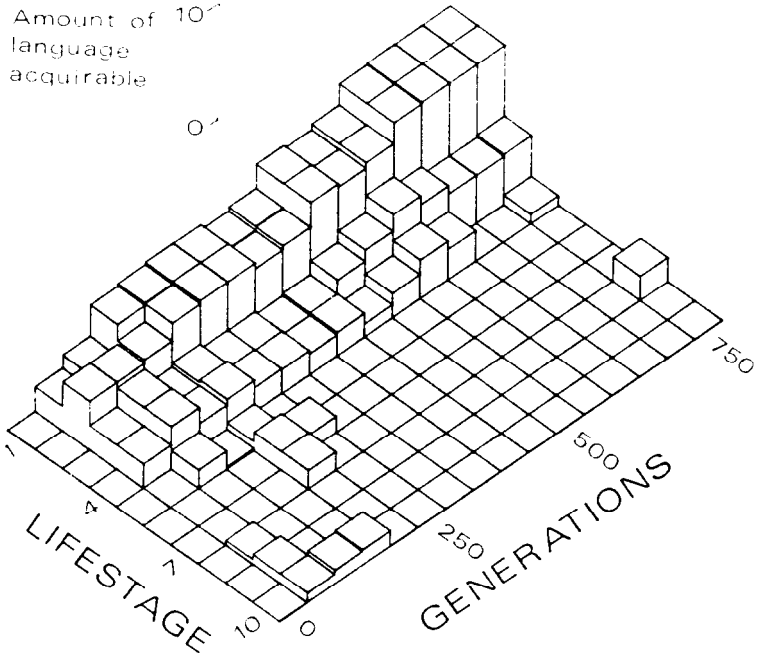
4. Conclusion

4.1. Interpretation of results

As the histograms in Figure 4 show, all four combinations of conditions produced clear critical period effects by 1000 generations.

The "mother" exemplar condition produced a more marked critical period effect, after 1000 generations, than the "wholepop" condition (compare the left-hand diagrams in Figure 4 with those on the right). Further running of the simu-

Figure 6. *Evolution of the critical period (in the "mother/survival" condition).*



lations showed continued, though slower, evolution toward a more marked critical period effect in the "wholepop" condition. This difference between the two acquisition exemplar conditions is to be expected. If one's exemplar for acquisition can disappear very early in one's life, those individuals predisposed to acquire most of their language very early in life will tend to have an advantage, whereas if an exemplar is guaranteed to be present for one's whole life, this evolutionary pressure is less. But even with the "wholepop" acquisition exemplar, there is pressure towards strains which learn their language early, as potential for reproduction or survival at any stage in life is related to one's knowledge *at that stage*. An individual with complete command of its language throughout its adult [redacted] only achieve this complete command late in life.

The "survival" condition produced a very slightly more marked critical period effect than the "reproduction" condition (compare the top diagrams in Figure 4 with the bottom ones). Perhaps if the rate of premature "accidental" deaths had been higher than three per cycle, the "survival" condition would have outstripped the "reproduction" condition by a greater margin.

The lower humps that appeared from time to time, representing small surges in language acquisition capacity at later lifestages, can be attributed to random

genetic drift, exaggerated by the inbreeding effect in a small population. A well-known effect in population genetics is that when neither of two alleles is favoured by selection pressure, sooner or later one of them will be eliminated by genetic drift. The time taken depends on the size of the population; smaller populations settle more quickly to one allele or the other, while larger populations retain a mixture of alleles for longer. "*In the absence of mutation, migration, and selection, the ultimate outcome of genetic drift is always fixation of one allele and loss of the other (or others)*" (Cavalli-Sforza & Bodmer, 1971, p. 388). In the conditions simulated here, once an individual has acquired language to the maximum language size, there can be no advantage in possessing a capacity to acquire more, at later stages in life. Language acquisition capacity reaches its limit of evolutionary advantage when the organism is equipped to acquire all that can be acquired. So there is no selectional pressure for acquisition-facilitating "positive" alleles at later lifestages. As positive and negative alleles are constantly produced by regular (random) mutation processes, the normally prevailing situation shows a fairly even mix of positive and negative alleles. But occasionally, a small population may quickly settle to a (temporarily) stable surplus of positive (or negative) alleles before mutation pressure has time to shake up the situation again and re-establish the usual balanced mixture of facilitating and inhibiting alleles.

In these simulations, the rate of mutation is unrealistically high, at one mutation per 30 births. A simple justification for this is the need to see results in a reasonable time. But a less pragmatic justification can also be given, in that this fast mutation rate actually serves the same function in these simulations as would a factor of intergroup migration, or exogamy. Both rapid mutation and intergroup migration are devices which keep the gene pools of small groups well stirred. "*Mutation and, in general, linear systematic pressures have the same effect as migration in counterbalancing drift*" Cavalli-Sforza & Bodmer, 1971, p. 403).

The later-lifestage surges in language acquisition capacity which occasionally showed up in the simulations would tend to be evened out by intergroup migration. To confirm this, a situation in which such a surge occurred was taken, and the population from this situation was "thrown into the melting pot" with three other populations (from the other combinations of experimental conditions) taken after the same number of generations (1200). Thus a larger group of 120 (= 4 × 30) individuals was formed (as if, after 1200 generations, the four separate experimental groups had come together for a jamboree). The mean language acquisition profile for the (sub)group showing the surge is given in Figure 7, and the mean language acquisition profile for the larger amalgamated group is in Figure 8. Evidently, the somewhat anomalous late life surge in language acquisition capacity for one subgroup has been evened out, leaving the whole population showing a clearer critical period effect.

The simulations show that there seems to be some advantage in stopping acquiring (linguistic) knowledge at a quite early stage in life. Yet for some there

Figure 7. *Situation after 1200 generations, showing late surge.*

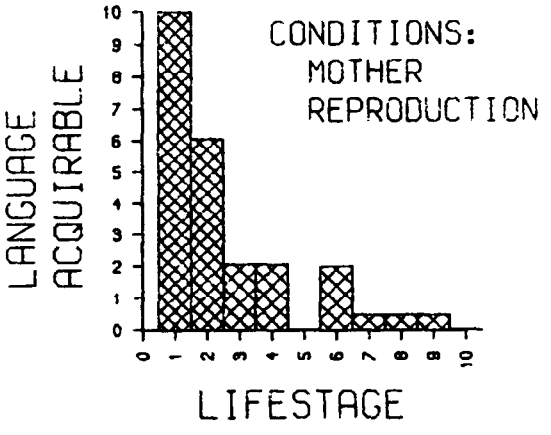
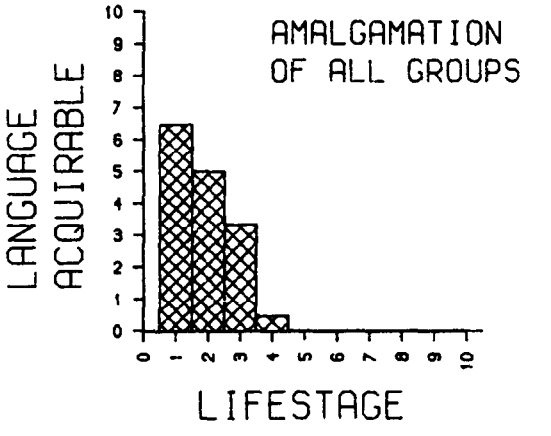


Figure 8. *Surge eliminated after mixing with other groups at 1200 generations.*



remains something paradoxical about this. If knowledge (e.g., of language) brings advantage, then it seems strange that there should be some advantage in actually *stopping* acquiring knowledge. The puzzle arises, I believe, because we are too accustomed to considering only the positive side of language acquisition, the facilitating factors. Reproduction, mutation and embryology can also "conspire" to produce language-acquisition-*inhibiting* factors, and these are actually likely to be more common in the space of possible alleles than facilitating factors, for two reasons. Firstly, as is well known, most mutations are deleterious, so any mutation affecting the language acquisition capacity is more likely than not to be in the direction of inhibition; and secondly, the language acquisition capacity, being a very rare phenomenon in nature, seems correspondingly less likely than more

common phenomena (e.g., flight, vision, etc.) to arise from the chance permutations of genetic material on which selection pressure may operate. The end of the critical period at around puberty is thus not so much a marked "switching off" point of language acquisition capacity, but rather a point where the selection pressure in favour of facilitating factors ceases to operate, because of success at earlier lifestages. Thinking of the end of the critical period as "switching off", like the deliberate switching off of a light, is less appropriate than thinking of it as a point where the "energy" in the system, the selection pressure in favour of positive alleles, is dissipated, and the "light" goes out for lack of pressure to keep it "on".

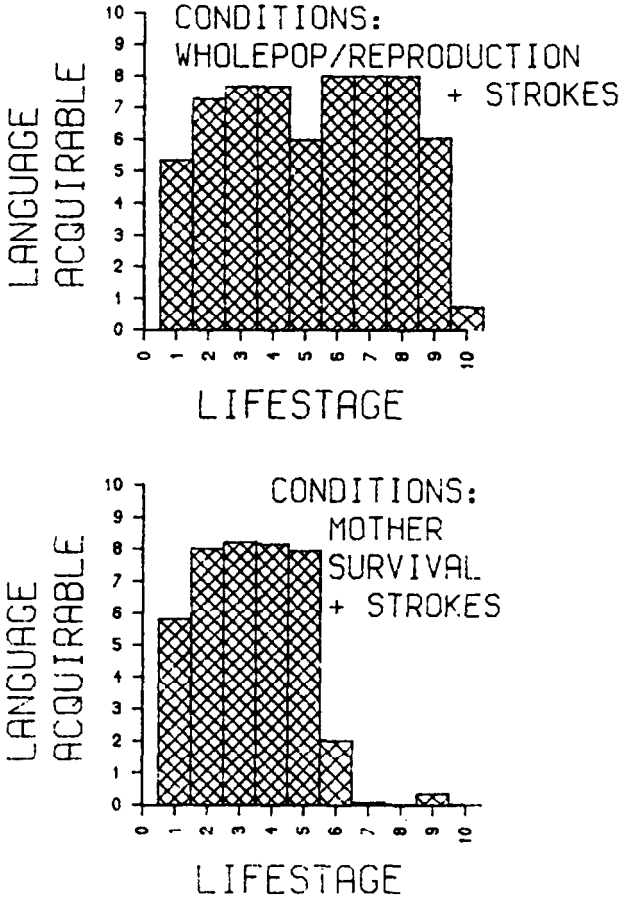
This account of the critical period for language acquisition as a non-adaptive by-product of natural selection is closely parallel to current accounts of the evolution of other life-history traits, in particular of senescence. Williams (1957) proposed a theory of the evolution of senescence by natural selection broadly similar to that of Medawar (1952). Williams' theory, that senescence is an evolutionary by-product of selection for other beneficial traits, has been developed further by Kirkwood and Holliday (1986), and experimentally supported by Ross and Charlesworth (1980).

Presumably, if humans underwent language-impairing brain damage frequently throughout their lives, but without actually dying, there would be selection pressure for capacity to regenerate language at later lifestages, rather as a lizard has the capacity to grow a new, albeit imperfect, tail, after the original one is lost. This possibility was in fact verified by two further simulations, in the "wholepop/reproduction" and "mother/survival" combinations of conditions, but with the added complication that at each cycle each individual had a 50% chance of losing 90% of its language. Thus, individuals typically underwent several severe language-impairing "strokes" during their lifetimes. The mean language acquisition profiles for these groups, after 1000 generations, are given in Figure 9, and should be compared with the upper right and lower left histograms, respectively, in Figure 4.

In the "reproduction" condition, all individuals live until the tenth lifestage, and, with the high risk of language loss throughout life, language acquisition capacity at all lifestages up to, but not including, the tenth is selected for. Individuals at stage 10 are about to die, so there is no selection pressure for language acquisition capacity at this last lifestage. In the "survival" condition, where individuals often die before the tenth stage, clearly, selection pressure for language acquisition capacity is still generally greater in earlier life, but, given a high risk of language loss later in life, this pressure now makes some impression in middle life as well.

Another hypothetical possibility, mentioned by a referee, is that of a situation in which "for example, children were not exposed to speakers until the second or third life period, and/or their sensorium remained undeveloped until that time

Figure 9. *Language acquisition capacity facilitated in middle and late life if severe language loss may occur.*



(so that exemplars were ineffective), and/or the child had many other things to acquire in the first lifestage so that it could not give undivided attention to the speaker. Then, presumably, the critical period would be set back." Although I have not simulated this particular possibility, it is clear that the referee's presumption would be borne out, and language acquisition would be facilitated at a somewhat later period.

This referee also poses a rather searching and radical question, which, if I understand it correctly, goes beyond the scope of the assumptions made in this paper, but which deserves attention. I assume throughout that competence in an actual language is acquired, though the faculty to acquire it is innate. The referee asks whether an evolutionary model can be proposed, along the general lines of my model here, showing why indeed language *is* acquired, rather than innate.

This is a good question, which would take another paper (at least) to investigate, but I suggest that Hinton and Nowlan's (1987) approach (which also uses computer simulation as a tool) makes a good starting point for this investigation. Pinker and Bloom comment briefly on Hinton and Nowlan's work in the context of the innateness or otherwise of language. Here I will do no more than quote their suggestive comment:

> Though there is always a selection pressure to make learnable connections innate, this pressure diminishes sharply as most of the connections come to be innately set, because it becomes increasingly unlikely that learning will fail for the rest. This is consistent with the speculation that the multiplicity of human languages is in part a consequence of learning mechanisms existing prior to (or at least independent of) the mechanisms specifically dedicated to language. Such learning devices may have been the sections of the ladder that evolution had no need to kick away. (Pinker & Bloom, 1990, section 5.2.3)

4.2. Other explanations of the critical period

Bever (1981) claims to give an explanation for the critical period for language acquisition. His article is valuable for an important argument concerning the relation between perception and production mechanisms, on the one hand, and a "bidirectional" (or inherently non-directional) internalized grammar, on the other. Bever argues that the language learner builds an internalized grammar as a device for maintaining necessary coordination between production and perception. Without such coordination, there would be far greater divergence than there (still) is between the set of producible utterances and the set of interpretable utterances. When the language has been learned, the adult possesses knowledge of closely coordinated sets of producible and interpretable potential utterances (types, not tokens, obviously), thanks to the mediation of the grammar being internalized during language acquisition. At this stage, the adult language having been learnt, and the known sets of producible and interpretable utterances being well coordinated, the internal grammar-building device has served its purpose and becomes "decoupled" from the twin performance systems. At this point, Bever's argument goes, it is no longer available to perform its original task, should it be called upon to mediate in the acquisition of some new (say foreign) sets of producible and interpretable utterances.

> A reasonable hypothesis is that when the speech production and perceptual system are well aligned with respect to a linguistic property [as they have become for an adult], then internal communication between them is no longer needed for that property. The communication channel [the major part of which is the internalized grammar] falls into disrepair because of disuse. (Bever, 1981, p. 193)

Bever's emphasis on the independence of production and perception, and on the role of the internalized bidirectional grammar in aligning them, is a valuable

insight. This fundamental function of the LAD, specifically constructing declarative knowledge about the two-way relation between *signifiants* and their *signifiés*, from observation of independent acts of transmission and reception by others, is usually taken for granted, and seldom discussed. The question hardly ever asked by language acquisition research is "*Why does* the child find it natural to construct a system of two-way signs, in response to observed data consisting of separate acts of perception and production?" Bever recognizes this as an issue, and proposes that this is indeed a function of the LAD, or device for building a "psychogrammar", as he puts it. (A very similar view of this basic function of the LAD is found in Hurford (1989), which takes the above "Why?" question a step further, and suggests an evolutionary reason why organisms disposed to internalize two-way declarative knowledge of the sort basic to language structure would be capable of constructing more effective communication systems, in a certain well-defined sense.) But I believe that Bever has not *explained* the critical period for language acquisition, in the sense of showing *why* there is this "decoupling" of a declaratively known system from behavioural systems at a certain stage when language acquisition is deemed to be complete. In fact, the very idea of the possibility of acquisition being complete, of there being a recognizable stable adult form of the language at the end of the dynamic process of language acquisition, is at the heart of the matter.

Bever's paper presupposes throughout that with the ontogeny of language, as with physical growth, a stable "adult", complete, stage is reached. I accept that this is the case, as a contingent fact, and ask the question why language growth and physical growth should be roughly synchronized in this way. I assume that a language is a finite bounded system which can be known in its entirety by a speaker. But I do not start with any assumptions about the stage in life when a speaker can attain this complete knowledge. To illustrate how the acquisition of a property can in principle be out of synchronization with physical growth, consider the property of wisdom, as conventionally, no doubt humorously, conceived. Why should language not be like wisdom, which, according to some at least, can only be fully attained with great age? The acquisition of wisdom does not seem to be synchronized with physical development, in this view.

Bever's paper builds on a metaphor, which in some ways might be quite fruitful, likening language growth to cell growth:

> The essential moral is *the facts of a critical period are accounted for by the normal processes of growth* – that is, the critical period is not a special property of growth in its own right, but rather is the loss of plasticity of function which occurs as cells become more specialized and independent. The model can be articulated as the following:
>
> (1) Development initially proceeds within partially independent systems (e.g., cells). Each system differentiates internally and is influenced by the development and properties of adjacent systems.

> (2) Due to internal processes of growth, the adjacent systems "decouple", becoming independent of each other's influence with respect to further development. This decoupling is "critical" in the sense that under ordinary circumstances it is irreversible. (Bever, 1981, p. 184)

There is nothing at all in the form of Bever's model which dictates that the "decoupling" to which he attributes the loss of language-learning ability should happen around puberty. He does not mention puberty in his article, nor make any reference to other events in an individual's biological life history. Why should the decoupling not be synchronized, not with puberty, but rather with menopause, or even with death, in which case, of course, there would be no (non-trivial) sense in which there actually was a critical period? If the language-learning ability did not fade until (near) death, the range of people who were physically adults would presumably not display the homogeneity associated with the phrase "adult language". But Bever's putative explanation rests on the assumption of a pre-existing difference between adult language and developing (i.e., child) language. In fact, according to the explanation put forward in the present paper, the difference between stable adult language and developing child language only arises because there is a critical period. The critical period itself is explained in evolutionary terms of natural selection.

To my knowledge, no other author besides Bever has advanced a detailed explanation for the critical period. Bever's explanation, as noted, is not an evolutionary explanation. In the literature on evolution I have only been able to find two relevant suggestions, both, as it happens, by theorists of great eminence: George Williams and Peter Medawar. Both suggestions foreshadow in different ways the explanation offered in the present paper. Williams' rather light-hearted hint at an explanation for the critical period for language acquisition is:

> Many of the accidental deaths of small children would probably have been avoided if the victims had understood and remembered verbal instructions and had been capable of effectively substituting verbal symbols for real experience. This might well have been true also under primitive conditions. *The resulting selection for acquiring verbal facility as early as possible* might have produced, as an allometric effect on cerebral development, populations in which an occasional Leonardo might arise. (Williams, 1966, p. 15–16, emphasis added)

Medawar was concerned with the evolution of senescence, and developed very general ideas on how natural selection works on age-related genetic factors. The following passages amount to a prediction that any favourable genetically determined age-related life-history event will tend to "migrate", over an evolutionary timespan, to ever earlier stages of life, processing just as far "back" as puberty:

> If hereditary factors achieve their overt expression at some intermediate age of life; if the age of overt expression is variable; and if these variations are themselves inheritable; then natural selection will so act as to enforce the postponement of the

> age of expression of those factors that are unfavourable, and, correspondingly, to expedite the effects of those that are favourable – a recession and a precession, respectively, of the variable age-effects of genes. (Medawar, 1952, p. 67)
>
> There is always a pre-reproductive period ... and during this period the average reproductive value of an individual must therefore rise to a maximum, irrespective of whether or not it falls later. If my reasoning is correct – there is no time to go into details – the precession of the time of action of genes comes to a standstill at the epoch when the reproductive value is at a maximum ... (Medawar, 1952, p. 69)

The detailed selection mechanism clearly needed more exploration, despite the eminence of Williams and Medawar as intellectual antecedents. This paper has made a start in that direction. Different variants of the model presented here, and indeed alternatives to it, will need to be considered, parallel with work on the biochemical correlates of the timing of language acquisition potential.

References

Aitchison, J. (1989). *The articulate mammal* (3rd Ed.). London: Unwin Hyman.

Bever, T.G. (1981). Normal acquisition processes explain the critical period for language learning. In K.C. Diller (Ed.), *Individual differences and universals in language learning aptitude* (pp. 176–198). Rowley, MA: Newbury House.

Bever, T.G., Carrithers, C., Cowart, W., & Townsend, D.J. (in press). Tales of two sites: The quasimodularity of language. In A. Galaburda (Ed.), *Neurology and language*. Cambridge, MA: MIT Press.

Bickerton, D. (1981). *Roots of language*. Ann Arbor: Karoma.

Blakemore, C., Garey, L.J., & Vital-Durand, F. (1978). The physiological effects of monocular deprivation and their reversal in the monkey's visual cortex. *Journal of Physiology, 283*, 223–262.

Brown, R. (1957). *Words and things*. Glencoe, IL: Free Press.

Cavalli-Sforza, L.L., and Bodmer, W.F. (1971). *The genetics of human populations*. San Francisco: Freeman.

Charlesworth, B. (1980). *Evolution in age-structured populations*. Cambridge: Cambridge University Press.

Charlesworth, B. (1990). Life and times of the guppy. *Nature, 346*, 313–314.

Chomsky, C. (1969). *The acquisition of syntax in children from 5 to 10*. Cambridge, MA: MIT Press.

Chomsky, N.A. (1982). *The generative enterprise: A discussion with Riny Huybregts and Henk van Riemsdijk*. Dordrecht: Foris.

Church, A. (1935). An unsolvable problem of elementary number theory: Preliminary report. *Bulletin of the American Mathematical Society, 41*, 332–333.

Crow, J.F., & Kimura, M. (1970). *An introduction to population genetics theory*. New York: Harper & Row.

Curtiss, S. (1977). *Genie: A psycholinguistic study of a modern-day "wild child"*. New York: Academic Press.

Dale, P.S. (1976). *Language development: Structure and function* (2nd ed.). New York: Holt, Rinehart & Winston.

Dawkins, R. (1982). *The extended phenotype: The gene as the unit of selection*. Oxford: Oxford University Press.

Edney, E.B., & Gill, R.W. (1968). Evolution of senescence and specific longevity. *Nature, 220*, 281–282.

Falconer, D.S. (1960). *An introduction to quantitative genetics*. Edinburgh: Oliver & Boyd.
Fletcher, P. (1990). Speech and language defects. *Nature, 346*, 226.
Fox, R. (1986). Fitness by any other name. *Behavioral and Brain Sciences, 9*, 192–193. (Peer commentary on Vining, 1986.)
Freedman, D.G. (1967). A biological approach to personality development. In Y. Brackbill (Ed.), *Infancy and early childhood*. New York: Free Press.
Freedman, D.G., King, J.A., & Elliot, O. (1961). Critical period in the social development of dogs. *Science, 133*, 1016–1017.
Fromkin, V., Krashen, S., Curtiss, S., Rigler, D., & Rigler, M. (1974). The development of language in Genie: A case of language acquisition beyond the "critical period". *Brain and Language, 1*, 81–107. Reprinted in Lahey, M. (Ed.) (1978). *Readings in childhood language disorders* (pp. 287–309). Melbourne, FL: Krieger.
Givon, T. (1979). *On understanding grammar*. New York: Academic Press.
Goldin-Meadow, S. (1979). Structure in a manual communication system developed without a conventional language model: Language without a helping hand. In H. Whitaker & H.A. Whitaker (Eds.), *Studies in neurolinguistics* (Vol. 4, pp. 125–209). New York: Academic Press.
Gopnik, M. (1989). A featureless grammar in a dysphasic child. Unpublished typescript. Department of Linguistics, McGill University.
Gopnik, M. (1990a). Feature-blind grammar and dysphasia. *Nature, 344*, 715.
Gopnik, M. (1990b). Genetic basis of grammar defect. *Nature, 347*, 26.
Haldane, J.B.S. (1941). *New paths in genetics*. London: Allen & Unwin.
Halliday, M.A.K. (1973). *Explorations in the functions of language*. London: Edward Arnold.
Hamilton, W.D. (1966). The moulding of senescence by natural selection. *Journal of Theoretical Biology, 12*, 12–45.
Harwerth, R.S., Smith, E.L., III, Duncan, G.C., Crawford, M.L.J., & von Noorden, G.K. (1986). Multiple sensitive periods in the development of the primate visual system. *Science, 232*, 235–238.
Hess, E.H. (1973). *Imprinting*. New York: Van Nostrand.
Hinton, G.E., & Nowlan, S.J. (1987). How learning can guide evolution. *Complex Systems, 1*, 495–502.
Hubel, D.H., & Wiesel, T.N. (1963). Receptive fields of cells in striate cortex of very young, visually inexperienced kittens. *Journal of Neurophysioloigy, 26*, 994–1002.
Hubel, D.H., & Wiesel, T.N. (1965). Binocular interaction in striate cortex of kittens reared with artificial squint. *Journal of Neurophysiology, 28*, 1041–1059.
Hurford, J.R. (1989). Biological evolution of the Saussurean sign as a component of the language acquisition device. *Lingua, 77*, 187–222.
Jerison, H.J. (1973). *Evolution of the brain and intelligence*. New York: Academic Press.
Johnson, J.S., & Newport, E.L. (1989). Critical period effects in second language learning: The influence of maturational state on the acquisition of English as a second language. *Cognitive Psychology, 21*, 60–99.
Kirkwood, T.B.L., & Holliday, R. (1979). The evolution of ageing and longevity. *Proceedings of the Royal Society, B205*, 531–546.
Kirkwood, T.B.L., & Holliday, R. (1986). Ageing as a consequence of natural selection. In A.H. Bittles & K.J. Collins (Eds.), *The biology of human ageing* (pp. 1–16). Cambridge: Cambridge University Press.
Koehler, O. (1952). "Wolfskinder", Affen im Haus und vergleichende Verhaltensforschung. *Folia Phoniatrica, 4*, 29–53.
Lazar, R.M., Tamaroff, M., Nir, Y., Freund, B., O'Reilly, R., Kirkpatrick, D., & Kapoor, N. (1983). Language recovery following isolation for severe combined immunodeficiency disease. *Nature, 306*, 54–55.
Lenneberg, E.H. (1967). *Biological foundations of language*. New York: Wiley.
Lenneberg, E.H. (1972). On explaining language. In M.E.P. Seligman & J.L. Hager (Eds.), *Biological boundaries of learning* (pp. 379–396). New York: Appleton–Century–Crofts.
Lenneberg, E.H., Nichols, I.A., & Rosenberger, E.F. (1964). Primitive stages of language develop-

ment in mongolism. *Disorders of communication* (Vol. XLII). Baltimore, MD: Williams & Wilkins.

Ludlow, C.L., & Cooper, J.A. (1983). Genetic aspects of speech and language disorders: Current status and future directions. In C.L. Ludlow & Cooper, J.A. (Eds.), *Genetic aspects of speech and language disorders*. New York: Academic Press.

Marcotte, A.C., & Morere, D.A. (1990). Speech lateralization in deaf populations: Evidence for a developmental critical period. *Brain and Language, 39*, 134–152.

Marler, P. (1972). A comparative approach to vocal learning: Song development in white-crowned sparrows. In M.E.P. Seligman & J.L. Hager (Eds.), *Biological boundaries of learning* (pp. 336–376). New York: Appleton-Century-Crofts.

Mayr, E. (1970). *Populations, species, and evolution*. Cambridge, MA: Harvard University Press.

Medawar, P.B. (1952). *An unsolved problem in biology*. London. Reprinted in Medawar P.B. (1957). *The uniqueness of the individual*. London: Methuen. (Page references are to reprinted version.)

Milner, E. (1976). CNS maturation and language acquisition. In H. Whitaker & H.A. Whitaker (Eds.), *Studies in neurolinguistics* (Vol. 1, pp. 31–102). New York: Academic Press.

Monod, J. (1972). *Chance and necessity*. London: Collins.

Newport, E.L., & Supalla, T. (1987). A critical period effect in the acquisition of primary language. University of Illinois, manuscript under review.

Nottebohm, F. (1969). The "critical period" for song learning. *Ibis, 111*, 386–387.

Nottebohm, F. (1984). Vocal learning and its possible relation to replaceable synapses and neurons. In D. Caplan, A.R. Lecours, & A. Smith (Eds.), *Biological perspectives on language* (pp. 65–95). Cambridge, MA: MIT Press.

Oyama, S. (1976). A sensitive period for the acquisition of nonnative phonological system. *Journal of Psycholinguistic Research, 5*, 266–283.

Oyama, S. (1978). The sensitive period and comprehension of speech. *Working Papers on Bilingualism, 16*, 1–17.

Partridge, L., & Harvey, P.K. (1988). The ecological context of life-history evolution. *Science, 245*, 1343–1350.

Piattelli-Palmarini, M. (1989). Evolution, selection, and cognition: From "learning" to parameter setting in biology and the study of language. *Cognition, 31*, 1–44.

Pinker, S., & Bloom, P. (1990). Natural language and natural selection. *Behavioral and Brain Sciences, 13*, 707–784.

Ploog, D. (1984). Comment on J. Leiber's paper. In R. Harre & V. Reynolds (Eds.), *The meaning of primate signals* (p. 88). Cambridge: Cambridge University Press.

Rose, M., & Charlesworth, B. (1980). A test of evolutionary theories of senescence. *Nature, 287*, 141–142.

Rovee-Collier, C.K., & Lipsitt, L.P. (1982). Learning, adaptation, and memory in the newborn. In P. Stratton (Ed.), *Psychobiology of the human newborn* (pp. 147–190). New York: Wiley.

Scarr-Salapatek, S. (1976). An evolutionary perspective on infant intelligence: Species patterns and individual variations. In M. Lewis (Ed.), *Origins of intelligence: Infancy and early childhood* (pp. 165–197). London: Wiley.

Scott, J.P. (1978). Critical periods for the development of social behavior in dogs. In J.P. Scott (Ed.), *Critical periods*. Stroudsberg, PA: Dowden, Hutchinson & Ross.

Singh, J.A.L., & Zingg, R.M. (1942). *Wolf children and feral man*. New York: Harper.

Singleton, D. (1989). *Language acquisition: The age factor*. Clevedon, UK: Multilingual Matters.

Sober, E. (1984). *The nature of selection: Evolutionary theory in philosophical focus*. Cambridge, MA: MIT Press.

Spuhler, J.N. (1979). Genes, molecules, organisms, and behavior. In J.R. Royce & L. Mos (Eds.), *Theoretical advances in behavior genetics* (pp. 9–40). Alphen aan den Rijn, Netherlands: Sijthoff & Noordhoff.

Tinbergen, N. (1972). The innate disposition to learn. In M.E.P. Seligman & J.L. Hager (Eds.), *Biological boundaries of learning* (pp. 245–250). New York: Appleton-Century-Crofts.

Vargha-Khadem, F., & Passingham, R.E. (1990). Speech and language defects. *Nature, 346*, 226.

Vining, D.R., Jr. (1986). Social versus reproductive success: The central theoretical problem of

human sociobiology. *Behavioral and Brain Sciences, 9*, 167–187.

Wachs, T.D., & Gruen, G.E. (1982). *Early experience and human development*. New York: Plenum Press.

Wells, G. (1979). Variation in child language. In P. Fletcher & Garman, M. (Eds.). *Language acquisition: Studies in first language development* (pp. 377–395). Cambridge: Cambridge University Press.

Wexler, K., & Culicover, P. (1980). *Formal principles of language acquisition*. Cambridge, MA: MIT Press.

Wiesel, T.N., & Hubel, D.H. (1963a). Effects of visual deprivation on morphology and physiology of cells in the cat's lateral geniculate body. *Journal of Neurophysiology, 26*, 978–993.

Wiesel, T.N., & Hubel, D.H. (1963b). Single-cell response in striate cortex of kittens deprived of vision in one eye. *Journal of Neurophysiology, 26*, 1003–1017.

Wiesel, T.N., & Hubel, D.H. (1965a). Comparison of the effects of unilateral and bilateral eye closure on cortical unit responses in kittens. *Journal of Neurophysiology, 28*, 1029–1040.

Wiesel, T.N., & Hubel, D.H. (1965b). Extent of recovery from the effects of visual deprivation in kittens. *Journal of Neurophysiology, 28*, 1060–1072.

Williams, G. (1957). Pleiotropy, natural selection, and the evolution of senescence. *Evolution, 11*, 398–411.

Williams, G.C. (1966). *Adaptation and natural selection*. Princeton, NJ: Princeton University Press.