Table 1 Age records as percentages of world records

Age	Shot-put	Discus	200 m	Marathon
10	61	60	79	68
20	91	90	98	94
30	100	100	99	100
40	98	95	93	96
50	76	78	84	88
60	49	53	74	76

Percentages based on fitting the exponential model (see text), to published data.

Thus, a speed of 8.12 m s^{-1} for the 200 m race is 80% of the world record speed (10.15 m s^{-1}), whereas a shot-put of 17.46 m is 80% of the corresponding world record (21.82 m). These comparisons (Table 1) lend further support to the hypothesis that strength deteriorates faster with age than does stamina.

Fewer data are available on women's age records (current records only include running events up to 3,000 m, and rarely are there records for women over 45 yr of age). A comparison with those for men suggests that girls mature faster than boys (with respect to running events) in the age range 8–12 yr. For example, at ages 8, 9 and 11 women's records are faster than those for males in the 400 m run (see Fig. 1*b*). It also appears that beyond age 30, speed deteriorates at a faster rate for women than for men. For example, the fit curves show that in the 100 m sprint the women's record at age 45 is 85% of the women's world record, whereas the corresponding figure for men is 90% of the men's world record.

The records analysed here comprise the marks of many individual athletes but they can be thought of as those set by a 'super' runner; one who is in top condition throughout his life span. The ordinary runner is one whose speed is slower, perhaps by some fixed amount over the entire age span, than that of the super runner. The speed curve for the ordinary runner may have the same shape as that of the super runner and the rates of change of speed with age would be the same. This hypothesis could be tested by following up several runners over their life spans, recording annually their running times over measured distances. This could be done retrospectively, by contacting those who have run for a number of years and determining their best times at various distances each year.

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DAN H. MOORE II

Biomedical Division,
Lawrence Livermore Laboratory,
Livermore, California 94550

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The capacity for joint visual attention in the infant

LITTLE is known about how visual attention of the mother–infant pair is directed jointly to objects and events in the visual surround during the first year of the child's life. To what extent does the child follow the mother's lead and the mother the child's, and what are the processes involved? The ability of the infant to respond successfully to such signals allows the mother to isolate and highlight a much wider range of environmental features than if the infant ignores her attention-directing efforts. We report a preliminary investigation of the extent of the infant's ability to follow changes in adult gaze direction during the first year of life.

Mothers who had volunteered as subjects in response to newspaper advertisements were asked to bring their infants into our laboratory at a time when the infant was 'usually active'. Thirty-four infants, 2–14 months old, were tested in a small, sparsely-furnished room (15 ft by 10 ft) with a one-way screen at one end and a window with drawn blinds at the other. The infant was placed in a highchair, appropriate to his age, in an upright position. The mother played with him until he seemed settled and was then replaced by the experimenter—either male or female in their twenties, unknown to the baby. It was not practicable to have the mothers as experimenters with the rather strict testing requirements used here although many observations were made prior to this on other mothers and infants in a similar, though less controlled, situation. The experimenter first played with the infant for a short period and, if the latter showed no signs of distress, the mother left the room. The experimenter remained seated in front of the infant, eyes at the same level, about 0.5 m away. The infant was then given two trials in a prearranged order.

On each trial the experimenter first made eye-to-eye contact then silently turned his (her) head through 90° to fixate a small (concealed) signal light, 1.5 m away, for 7 s. He then turned back to interact with the infant. Two trials were given, one involving a head turn to the right, one a turn to the left, with inter-trial intervals varying from 20 to 50 s, depending on difficulties in establishing subsequent eye-to-eye contact. The infant's behaviour was recorded by two concealed video cameras set at 45° to the experimenter–infant face-to-face axis, giving a split-screen 45° profile of the infant from two sides. The experimenter was not visible on the screen. The infants were also filmed for calibration looking at experimenter in fixed positions of known angular displacement.

Trials were scored from the videorecord by the experimenter, based on infant head movement only. A positive response was scored if the infant looked (a) in the same direction (right/left) but not down at the floor or up at the ceiling; (b) without an intervening look elsewhere (ignoring short looks down during postural adjustment); (c) within 7 s; and (d) appeared to be looking for or at something (involving halting the head turn for 0.5 s or more with a cessation of limb movement). The infant did not have to appear to be fixating exactly the same point. Scoring reliability was ascertained from three naive observers uninformed as to experimenter's judgment. They produced 96% agreement on trials scored as positive, 90% on those scored as negative.

The proportion of infants judged as having produced a positive response on one or both trials increases steadily with age (Table 1). The form of the response, that is, how soon and where the infants look, did not show systematic change with age. Latencies in response were very variable—any point up to the end of the trial, and the infants would look anywhere from about 20° to 90° away from the midline. Labelling responses as positive does not, of course, mean that infants were looking for something to look at. In the upper age groups, however, there was strong evidence to suggest this may be so with infants often looking away, looking back at the experimenter and then looking away again. This had been even more marked when the mother was the experimenter in earlier observations. Of the negative trials over 80% were comprised of responses where the infant either kept his eyes on the experimenter or looked down at the highchair table top.

The proportion successfully responding may well be depressed by the strangeness of the setting although the incidence

Table 1 Percentage of children judged as following line of regard in one or both trials

Age (months)	No. infants	% Showing positive response
2–4	10	30
5–7	13	38.5
8–10	6	66.5
11–14	5	100

of following line of regard is enhanced by pointing and exclamatory vocalisations such as "Oh look". By 11 months, however, some children gave unequivocal positive responses to eye movement alone. Some (from 6 months) even followed line of regard when the experimenter and mother, not interacting with the infant, conversed a metre away from the child.

It is possible that the ability to orient with respect to another has implications for Piaget's[1] more complex notions of the egocentric child. In so far as mutual orientation implies a degree of knowledge in some form about another person's perspective then the child in its first year may be considered as less than completely egocentric. The source of such abilities (for example, imitation) remains to be investigated but utilisation of another's gaze direction may be a very basic process. Cooke's Hartebeeste (*Alcelaphus buselaphus*) on hearing a danger signal raise their heads, look at the signaller and orient in the direction he is facing (M. Stanley-Price, personal communication). Such observations need careful investigation but it may not be entirely unexpected that human infants should also have greater abilities than has been supposed (for example, Schaffer[2]).

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M. Scaife
J. S. Bruner

Department of Experimental Psychology,
South Parks Road,
Oxford, UK

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Role of juvenile hormone esterases and carrier proteins in insect development

The discovery of a binding protein, specific for juvenile hormone (JH)[1] in the haemolymph of the tobacco hornworm, *Manduca sexta*, suggested that this protein carries JH molecules from the secretory organs (corpora allata) to the target sites. We report here that the binding protein provides the hormone total protection from degradative enzymes present in the haemolymph throughout early larval life[2,3]. Just before the start of pupal differentiation, however, a new esterase appears in the haemolymph which is able to hydrolyse the protein-bound hormone. We have also found that the binding protein at concentrations of 10^{-8} M strongly enhances the development-inhibiting action of JH on wing disk tissue *in vitro*. These properties, taken together, demonstrate a carrier role for the binding protein, and give new insights into the control of hormone levels in the haemolymph.

Two closely related forms of the binding protein (CP-1 and CP-2) were purified to homogeneity by gel filtration on Sephadex G-100 (0.005 M Tris-HCl, *p*H 7.3, 0.1 M NaCl), followed by ion-exchange chromatography on Biogel DEAE (0.005 M Tris-HCl, *p*H 8.3, 0.1–0.14 M NaCl gradient) and preparative isoelectric focusing (*p*H 4–6, 1% Ampholine, 500 V, 72 h). The binding proteins elute as a single peak from the Sephadex column but are separated on the ion-exchange column and are finally purified by isoelectric focusing. Under these experimental conditions CP-1 (*p*I = 4.8) was the major binding protein (> 75% of the total hormone binding capacity) and was used for subsequent studies. The hormone–protein complex formed with pure CP-1 and that formed in crude haemolymph showed that same dissociation constant ($K_D \simeq 10^{-7}$ M)[1] and slow dissociation rate ($t_{1/2}$ = 15 min).

The JH hydrolytic activity in the haemolymph of fifth instar larvae can be separated into two distinct peaks by gel filtration on Sephadex G-100 (0.005 M Tris-HCl, *p*H 7.3, 0.1 M NaCl). The activity in peak I (molecular weight $\simeq 10^5$) is totally inhibited by 10^{-4} M diisopropylphosphorofluoridate (DFP) (*p*H 7.0, 15 min) whereas that in peak II (molecular weight $\simeq 6 \times 10^4$) is unaffected under the same conditions. Using this concentration of DFP, the crude haemolymph can be assayed for the two types of esterases. Enzyme class II has been further purified by preparative isoelectric focusing (*p*H 4-6, 1% Ampholine, 500 V, 72 h) and three closely related forms (JH-esterases A, B and C) were separated. All three forms show equal rates of hydrolysis of JH, and JH-esterase A was routinely used for our experiments.

Table 1 Carrier protein protection of JH against hydrolysis by esterase fractions isolated from haemolymph of fifth instar *M. sexta* larvae

Esterase fraction	Hydrolysis of JH (%) −CP*	Hydrolysis of JH (%) +CP†	Protection (%)
Peak I	43	0	100
JH-esterase A	74	70	5

Pure synthetic *Hyalophora cecropia* JH [(methyl *trans,trans,cis* 3, 11-dimethyl-7-ethyl-10,11-epoxytrideca-2,6-dienoate) labelled in the chain (7-ethyl-1,2-^{3}H,67 mCi mg^{-1} (New England Nuclear Corp.) was used. Reaction products were separated and identified by thin-layer chromatography according to the method of Slade and Zibitt[2] and quantitated by liquid scintillation counting.

*Incubation for 15 min at room temperature of 20 µl esterase and 10 µ JH (8×10^{-7}M) followed by addition of 10 µl carrier protein (4×10^{-5}M), incubation for 2 min and then addition of 10 µl charcoal (4.5 µg) to remove unbound JH. Charcoal removed by centrifugation at 8,000*g* for 2 min. Supernatant applied immediately to thin-layer chromatographic plate for analyses.

†Same procedure as described for −CP but CP and JH were pre-incubated for 2 min before addition of esterase.

The hydrolysis of the hormone by JH-esterase A was measured in the presence and absence of binding protein CP-1. Table 1 shows that none of the enzymes of family I can hydrolyse JH complexed to CP-1. On the other hand, JH-esterase A hydrolyses the hormone even in the presence of a large excess of CP-1. In an attempt to correlate the presence of JH-esterases with the levels of JH in the insect, we examined the haemolymph from fourth instar larvae and found that esterases of type II are virtually absent. In view of these results it was of interest to study the level of type II esterases during the critical premetamorphic period of the fifth instar. Figure 1 shows that the level of the JH-esterases increases considerably and reaches a peak on day 5 of the fifth instar while type I esterases show relatively little variation during the same period.

These results suggest important functions of both the binding protein and JH-esterases in the regulation of the JH level in the haemolymph during larval development. In the early instars, where high levels of JH are required, the binding protein is necessary to protect JH from degradation by type I esterases. Because of the continuous presence of these esterases, only JH complexed to the binding protein can reach the target tissues and thus the binding protein is the true carrier in the haemolymph. In the fifth instar, on the other hand, type II esterases could constitute the most important factor in lowering the JH level in the haemolymph, thus permitting metamorphosis.

To test further the participation of the carrier protein in the inhibitory action of JH in insect development, we used the technique of Oberlander and Tomblin[4]. This method measures the action of JH in terms of the inhibition of ecdysone-induced cuticle deposition in tissue cultures of imaginal wing disks from the Indian meal moth, *Plodia interpunctella*. Table 2 shows the considerable synergistic action exerted by low levels ($< 10^{-8}$ M) of *M. sexta* carrier protein on JH inhibition of *Plodia* wing disk metamorphosis. The protein alone has no effect in this system. Thus the carrier protein acts not only in haemolymph during transport of the hormone but also at the target issue. Because of the high level of hormone and the very low level of carrier protein (well below K_D) required to evoke a large synergistic effect, it is unlikely that this is simply a protective effect of the carrier against degradative enzyme, which is known to occur in disk tissue[5]. Rather, the