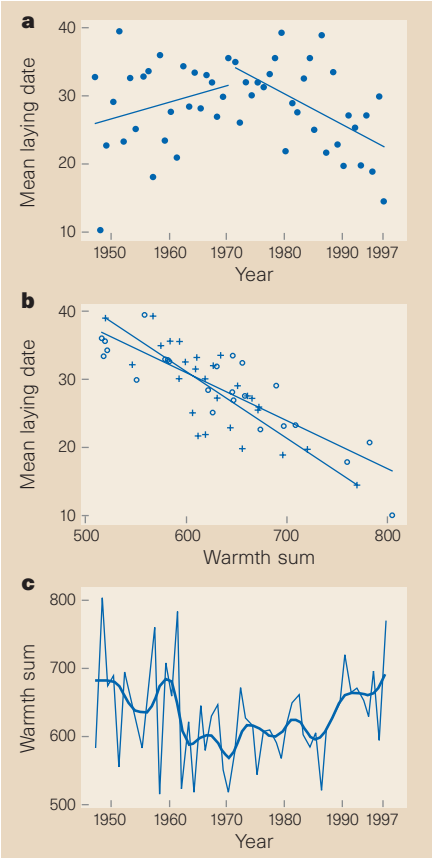

Figure 1 Laying date of Marley great tits and spring temperatures. **a**, Mean laying date (1 represents 1 April) plotted against time. Lines are regressions fitted to the two groups of data. The curvature of the best-fit model was confirmed by polynomial regression, which showed the quadratic term in time to be highly significant ($F = 11.37$, d.f. = 1,47, $P = 0.002$). The upward slope in 1947–70 is not significantly different from 0 (slope = 0.2035, s.e.m. = 0.2054, $t = 0.99$, 21 d.f., $P = 0.333$; if the exceptionally hot and early 1948 is removed the slope becomes 0.01). The tendency for laying to become earlier in 1971–97 is highly significant (slope = −0.4387, s.e.m. = 0.1331, $t = 3.30$, 25 d.f., $P = 0.003$). **b**, Regression of mean laying date for great tits in Marley wood against warmth sum. The data are classified as 1947–70 and 1971–97. The warmth sum is calculated as the sum of the daily maxima from 1 March to 25 April in each year. The difference in slopes for 1947–70, −0.069, and 1971–97, −0.097, is not significant ($F = 3.06$, d.f. = 1,47, $P = 0.087$). The overall slope of −0.083 ± 0.008 s.e.m. is significantly different from 0 ($t = -10.42$, $P < 0.001$). **c**, Temperature trends at Oxford. The thick line is the resistant smoother "4253H, twice"[7].

markedly from about 1985 onwards.

We know that there is considerable advantage to the birds to breed as early as they can, because the earliest breeders tend to produce the most surviving offspring[3]. However, the high energetic demands of breeding[4] might constrain the time at which the females can start to lay if sufficient food is not available. Experiments involving the provision of artificial food in the spring[5,6], causing the birds in the experimental area to breed earlier than controls, support the idea that the timing of breeding is at least to some extent food-limited. Presumably, the early warm weather enables the birds to breed earlier because the food supply they need for breeding becomes available earlier.

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Synchronized courtship in fiddler crabs

The apparent paradox posed by the synchronization of mating displays by males competing to attract females has provoked considerable interest among evolutionary biologists[1–3]. Such synchronized sexual signalling has only been documented for communication using light flashes (bioluminescence) or sound. It has been suggested that the "fundamental reasons that might favour precise adjustments in signal timing relative to that of a particular neighbour could only be compelling for signallers using these two channels"[1]. Here we provide the first quantitative evidence for synchronous production of a conventional visual courtship signal, the movement of a body part.

The fiddler crab (*Uca annulipes*) lives in mixed-sex colonies on inter-tidal mudflats. When their habitat is exposed at low tide, crabs emerge from their burrows, feed and interact socially. Gravid females leave their own burrows and mate in the burrows of males[4]. Females find these burrows by sequentially entering ('visiting') the burrows of several males. Males attract females to their burrows by repeatedly raising and lowering their single greatly enlarged claw (97 ± 6% of 'waving' is directed at females, $n = 30$ males). Several waving males usually cluster around a female. Before she mates, she visits one male from each of several clusters.

Preliminary field observations suggested that males in clusters wave synchronously[5]. To test this, we made video recordings of females and clustered waving males and documented the timing (0.04 s precision) of waves by each male in the cluster relative to the visited male ($n = 45$ clusters, each with 2–6 males) . We define a wave cycle as the interval between the onset of successive claw waves of the visited male (wave cycle duration = T_v). Each wave of each of the other males in the cluster ('neighbours') was assigned to the wave cycle of the visited male in which it began. We calculated the difference in the onset time of the first wave of the visited male (t_v) and that of a neighbour (t_n), and the phase angle, $\alpha = ((t_n - t_v)/T_v) \times 360°$ (Fig. 1a).

The phase angle is a measure of wave synchrony. If $\alpha = 0°$ or 360° there is perfect synchrony, at $\alpha = 180°$ waves are produced alternately. The mean T_v was 1.70 s (s.d. = 0.67); whereas the mean $(t_n - t_v)$ was only 0.20 s (s.d. = 0.31; for both means, $n =$ 328 wave cycles by 45 visited males). Using circular statistics, we found the mean α (±s.e.m.) for 328 pairs of waves was 4.4 ± 2.9°. We then calculated the mean α for each of the 45 clusters and tested whether these were uniformly distributed, as expected if males in clusters wave independently of one another. The distribution was not uniform. Instead, these means were significantly concentrated around phase angles indicating close synchrony (Rayleigh test, $P < 0.01$; mean ± s.e.m. per cluster = 6.8 ± 18.7°). Moreover, in 23 of the 29 clusters where sample sizes were large enough to permit statistical testing, the phase angles were significantly clumped ($P < 0.05$; Fig. 1b). Therefore, most neighbours, in most clusters, waved in close synchrony with the visited male.

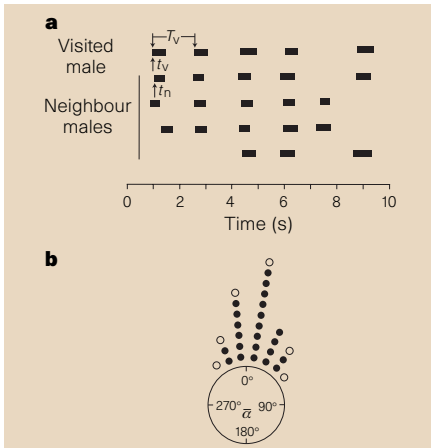

Figure 1 Synchronized courtship waving in clusters of male *Uca annulipes*. **a,** Waves (black bars) by five males in one of 45 clusters. The phase angle, α, is the difference in onset time of a wave of a neighbour male (t_n) and a wave of the male the female visited (t_v) as an angular proportion of the interval between onsets of the visited male's waves (T_v). **b,** Mean phase angles for 29 clusters. Filled circles: clusters with significantly non-uniform distributions; open circles: non-significant ($P > 0.05$). Fieldwork was carried out in Durban Harbour, South Africa.

None of the proposed cooperative explanations for synchrony[1] seem to apply to waving in *U. annulipes*. First, there are no predators who would be confused by group synchrony thereby reducing each male's predation risk. Second, synchrony does not increase group conspicuousness to distant females because males wave synchronously only when a female is <10 cm away (P. B., unpublished data). The most plausible explanation is one recently proposed for synchronous chorusing by a katydid[2]. Modelling shows that competition between males to call before their neighbours can lead to synchrony. Males compete to call first because females prefer leading calls.

This so-called 'precedence effect', whereby signal receivers show greater responsiveness to the earlier signal in a pair, is found in many acoustic situations, including sound localization in humans. It has not, however, been reported in a visual communication system. In *U. annulipes*, the visited male produced leading waves significantly more often than his neighbours (4.5±3.5 compared with 3.2±3.3 waves; Wilcoxon test, $n=45$, $P<0.02$). Female *U. annulipes* may prefer leading signals and synchrony may arise as an incidental effect of competition between males to signal first.

This is the first example of synchronous production of a non-bioluminescent visual signal. Mechanisms of visual signal perception and processing must possess the properties that were previously thought to limit synchronized courtship signalling to acoustic and bioluminescent channels[1].

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Connexin mutations and hearing loss

Kelsell *et al.*[1] provide convincing evidence that mutations in the gene encoding the gap-junction protein connexin 26 (Cx26) are responsible for autosomal recessive non-syndromic hearing loss at the DFNB1 locus on chromosome 13q12. They also report a small family with apparent autosomal dominant congenital hearing loss and autosomal dominant palmoplantar keratoderma (PPK) in which two siblings with profound hearing loss are heterozygous for a single base-pair substitution resulting in a methionine-to-threonine (ATG-to-ACG) change in codon 34 (M34T) of Cx26 (refs 1, 2). The authors conclude that the M34T change is the genetic basis of profound hearing loss in this family and suggest, on this basis, that the Cx26 gene is responsible for autosomal dominant non-syndromic hearing loss (ADNSHL) at the DFNA3 locus chromosome 13q12 (ref. 3). We have identified a family in which the M34T variant is not associated with hearing loss, suggesting that this conclusion might be premature.

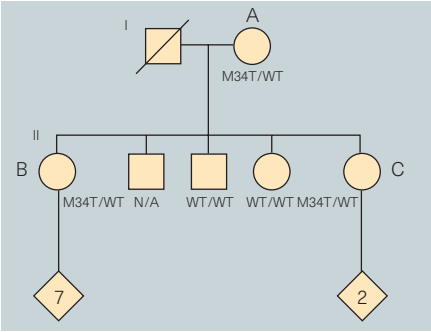

Figure 1 Pedigree structure in a family with the M34T variant of Cx26. Individual A (I:2) is 83 years old, individual B (II:1) is 57 years old and individual C (II:5) is 55 years old. The numbers in diamonds represent numbers of children. WT, wild-type; N/A, not available.

In a Cx26 mutation screen of 100 random individuals from the midwestern United States, we discovered one person who carries the M34T allele. DNA sequencing confirmed this person and two other family members to be heterozygous for this mutation. All three individuals have excellent hearing (Figs 1, 2)[4]. Individuals B and C report that their children, the youngest of whom is 24, all have normal hearing.

There are various explanations for these data. First, the M34T variant might be responsible for a form of ADNSHL that is not expressed in certain individuals. But the identification of multiple heterozygous individuals with no evidence of inherited hearing loss argues against this suggestion. Second, individuals in this family might carry a compensatory change that nullifies the effects of the M34T variant. However, single-stranded conformational polymorphism (SSCP) and sequence analysis of the Cx26 coding region of individual C show no evidence of additional sequence variants. The most likely explanation for these data is that the M34T variant represents a simple polymorphism that does not cause autosomal dominant hearing loss and is present in a small percentage of the general population.

Determining whether a particular DNA variant represents a disease-causing mutation or a simple polymorphism requires information about the inheritance pattern of the DNA variant within affected families and the frequency of the variant allele within the general population. Although Kelsell *et al.* show that M34T segregates with the profound hearing-loss phenotype in one affected family, the few individuals available for study and the presence of a second deafness-associated disorder make it difficult to draw any clear conclusions based on this family alone[2,5,6]. Kelsall *et al.* did not see the M34T variant in their screen of 80 chromosomes from non-related individuals, but a wider search might have revealed it.

Our results suggest that the M34T variant does not cause autosomal dominant hearing loss and emphasize the need for caution when interpreting mutation data based on a single affected family.

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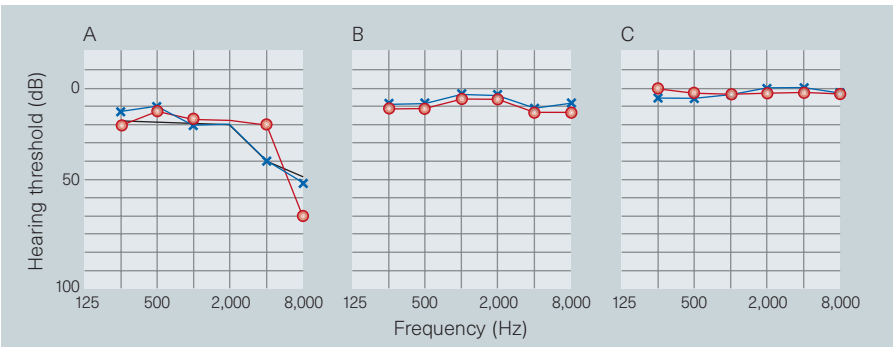

Figure 2 Pure-tone thresholds in the individuals identified in Fig. 1. Circles, right-ear thresholds; crosses, left-ear thresholds. The broken line in A represents the mean pure-tone thresholds in the better ear of women aged 80–84 (ref. 4).