zero-crossing structure of the stimuli, we do not mean to imply that zero-crossings are explicitly represented. Other descriptions of the pattern structure are possible (including the centroids of zero-bounded regions<sup>5</sup>), and we have no doubt that the positions of peaks of local energy<sup>14</sup> would give a similar picture. The critical question is whether  $d_{\text{max}}$  is determined by the statistical structure of the pattern, and if so, at what spatial scale or scales. Bex et al. do not provide a quantitative model of  $d_{\rm max}$  based on 'motion energy', and until one is available it would be premature to reject an existing model that has made some successful predictions.

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## Mariner transposons in humans

SIR — The mariner-Tc1 gene superfamily represents a diverse collection of DNAbased transposable elements with broad phylogenetic distribution<sup>1</sup>. We have now identified two mariner transposons in the human genome which share both structural and sequence similarity to elements previously described in arthropods, nematodes and planaria<sup>1-3</sup>. The two human elements are members of divergent mariner subfamilies, suggesting multiple instances of horizontal transfer of these elements into the human genome.

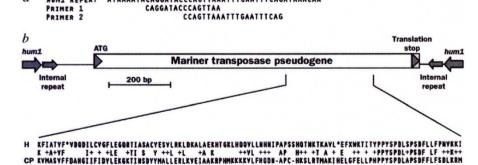
Mariner elements, first described in Drosophila<sup>4</sup>, consist of a transposase gene flanked by two short terminal inverted repeats. This structure is similar to a variety of other DNA-based transposable elements<sup>5</sup>, including the Ac element in maize. The autonomous Ac elements are associated with a family of nonautonomous Ds elements<sup>6</sup>. Whereas Acand Ds elements are defined by the same 11-base-pair (bp) inverted repeats, the Ds elements vary considerably in both size and internal sequence. Small, divergent, non-autonomous elements that share sequence similarity with the autonomous forms only within the terminal inverted repeats are a commonly observed feature in DNA-based transposons5.

The human mariner transposons were detected by identifying two families of putative non-autonomous transposons in

NUM1 REPEAT ATAAAATACAGGATACCCAGTTAAATTTGAATTTCAGATAAACAA

the genome. These elements were identified as short, conserved, inverted repeated domains present in multiple loci<sup>7</sup>. The members of the first of these families are defined by a 45-bp inverted repeated domain designated hum1 (a in the figure). Small putative transposable elements can be easily identified in a variety of human genes by screening the available databases with the consensus huml repeat<sup>8</sup>. Two oligonucleotide primers were designed using this repeat, and used to direct two rounds of polymerase chain reaction (PCR) amplification with human genomic DNA as a template. A 1.4-kilobase amplification product from these reactions was sequenced and found to be a mariner transposable element, designated humar1 (Gen-Bank locus HSU38613; b in the figure). The same element has also been cloned from a human genomic library using humarl PCR product sequences as probes.

The structure of *humar1* is similar to previously described mariner elements. As shown in the figure (b), the hum1 repeats defining the element flank smaller (29 bp) imperfect inverted repeated domains. We have sequenced two additional genomic clones containing humar1 elements (GenBank loci HSU38614 and HSU38615) which are defined only by the internal 29-bp repeats and lack the hum1 domains. This suggests that the internal repeats represent the authentic terminal



Sequence of the hum1 repeat and structure of the humar1 transposon. a, Consensus sequence of the hum1 inverted repeated domain. The oligonucleotide PCR primers used to amplify the humar1 transposable element are indicated. b, Structure and partial transposase deduced amino-acid sequence of the humar1 element. The relative positions of the hum1 and internal 29-bp inverted repeats are indicated by arrows. A portion of the carboxy-terminal domain of the transposase was translated (asterisks indicate stop codons) and submitted to the BLAST Network Service of the National Center for Biotechnology Information<sup>8</sup>. The sequence alignment of the humar1 (H) and C. ploribunda (CP) transposase was returned. Positions of identity are shown; + indicates a conservative change.

repeats of this transposon. The transposase pseudogene is interrupted by four frameshifts and numerous stop codons. The deduced amino-acid sequence of the transposase is similar to that of the mariner transposase from lacewing (Chrysoperla plorabunda; PIR locus \$36925), indicating that this element is a member of the irritans subfamily of mariner elements9.

We have identified a second human mariner element, a member of a second subfamily. Similar to humar1, this element was found by first identifying a second family of small, putative, non-autonomous transposons defined by inverted repeated domains (designated hum7, consensus inverted repeated domain sequence: TGGTGCAAAAGTAATGCAGTTTT-TGCCA)\*. When this sequence was used as a database query<sup>8</sup>, one of the returned sequences (GenBank locus HUMTCRB, positions 495,300-497,513) was found to contain a mariner element defined by two hum7 repeats. The DNA sequence of the transposase pseudogene at this locus is interrupted by four frameshifts, numerous stop codons and a 950-bp insertion. The deduced amino-acid sequence of the putative transposase is similar to that of a planarian (Dugesia tigrina) mariner transposase (GenBank locus DTPM9), suggesting that this element is derived from the cecropia subfamily of mariner elements9.

The two mariner elements described here represent two divergent subfamilies, consistent with multiple horizontal transfer events involving mariner transposons1. We have not yet identified intact (mobile) human mariner elements, which would represent potential sources of transformation vectors.

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\*During the preparation of this manuscript, we became aware that the humar7 element described here was also identified by Hugh Robertson, University of Illinois, using mariner-transposase-specific sequences (H. M. Robertson, personal comm unication), and that partial mariner transposase sequences have been reported<sup>1</sup>

a