Corrigenda

Thyroid hormone receptor α isoforms generated by alternative splicing differentially activate myosin HC gene transcription

Seigo Izumo & Vijak Mahdavi
Nature **334,** 539-542 (1988).
IN the typing of the derived amino-acid sequence of rTRα1 and rTRα2 cDNAs, the sequence for amino acids 401-410 were inadvertently swapped between the two isoforms. The correct sequence reads: FLEVFEDQEV for rTRα1 and QLLGMHVVQG for rTRα2.

T-cell antigen receptor genes and T-cell recognition

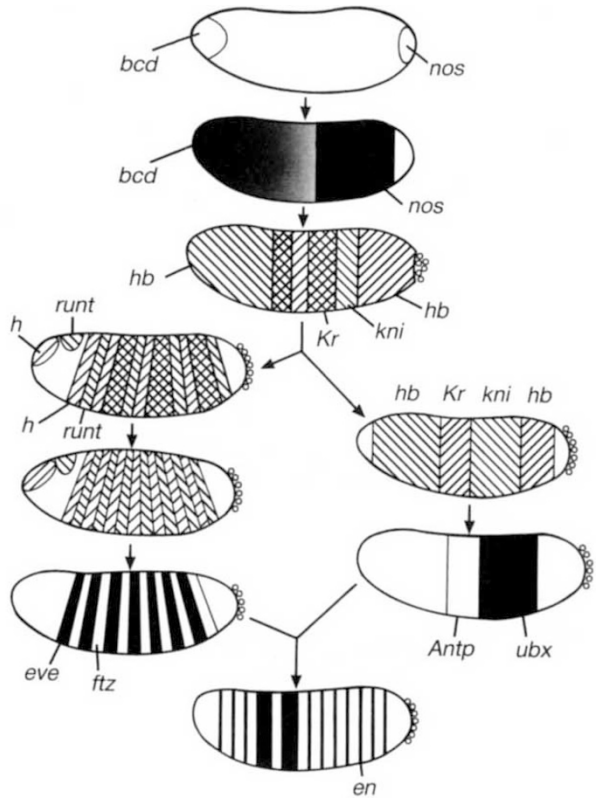
Mark M. Davis & Pamela J. Bjorkman
Nature **334,** 395-402 (1988).
IN this Review Article, the colour-coding given in the legend to Fig. 4 relates to an earlier version of the figure, not the one in the final version of the manuscript. The legend is reprinted here in full with the correct colour coding:

Fig. 4 Representations of the structures of *a*, the immunoglobulin combining site; *b*, the peptide-binding site of an MHC molecule; and *c*, the alignment of CDRs in a hypothetical TCR over a peptide-MHC complex. *a*, The arrangement of CDRs in an immunoglobulin antigen-binding site (Fab J539)[122] viewed from above (from the position of the antigen). The carbon-α backbone of V_H and V_L is shown in blue with van der Waals' surfaces highlighting the three CDRs from each domain (CDR1: blue, CDR2: yellow, CDR3: pink). The first and second CDRs from one variable domain (for example, immunoglobulin V_L or TCR V_α or V_γ) are separate from their counterparts on the partner variable domain (immunoglobulin V_H or TCR V_β or V_δ). The space between them is occupied by the third CDR from each *V* domain. The similarities between immunoglobulins and TCRs suggest that TCRs may have a combining site that preserves these same general features (see text for details). *b*, Top surface of an MHC molecule with a (hypothetical) bound peptide. The carbon-α backbone of the α_1 and α_2 domains of HLA-A2 (ref. 40) is shown in blue with van der Waals' surfaces highlighting the two α-helices (yellow). Van der Waals' surface of a hypothetical bound peptide (a 12-mer polyvaline α-helix) that has been fitted between the HLA α-helices is shown in pink. The putative recognition site for processed foreign antigens is located on the top surface of the molecule between two α-helices for the human class I histocompatibility molecule HLA-A2[40,41]. The N-terminal α_1 and β_1 domains of class II MHC molecules are predicted to have a similar tertiary structure and peptide-binding site[42]. Note that the distance between the MHC α-helices, and the separation of the first and second CDRs of each V-region in the combining site shown in part *a* are very similar. (Figures are to scale with respect to each other). *c*, Model for TCR interaction with a peptide-MHC complex. The combining site CDRs (*a*) are shown aligned over the peptide-MHC complex (*b*). The molecules in this figure are rotated ~90° with respect to their orientations in (*a*) and (*b*). *V* domains of Fab J539 (ref. 122; top of figure) here represent the *V* domains of a TCR bound to a peptide-MHC complex (bottom of figure). The carbon-α backbone of V_L and V_H (blue) is shown with main and side-chain atoms of CDR1 and CDR2 (yellow) from each domain. The main and side-chain atoms of the CDR3s of each domain are shown in pink. The carbon-α backbone of the α_1 and α_2 domains of HLA-A2 (blue) is shown with the main and side-chain atoms of the two α-helices in yellow and the main and side-chain atoms of a hypothetical peptide bound between the two helices in pink. The relatively flat surface of the V-region combining site is complementary to the flat surface of the MHC-peptide complex, with CDR1 and CDR2 of each V domain 'fitting' over an MHC α-helix. The CDR3s from each *V* domain are then aligned over the peptide (see text for details). Because the antibody (and presumably TCR) V regions pair with approximate twofold (dyad) symmetry[9,10], a similar interaction of CDR1 and CDR2 with the MHC α-helices and CDR3 with peptide would be accomplished if the V_H-V_L dimer shown in this figure were rotated by 180° about the pseudo-dyad axis between the two V regions (axis is vertical in this figure). Note that the relative sizes of the combining site (*a*) and the peptide-binding site of the MHC molecule (*b*) would allow TCRs to be bound in different registers along the MHC α-helices, depending on the particular peptide bound to that MHC protein. Thus the *V* genes employed by different T cells restricted to the same MHC molecule would not be expected to be identical. Furthermore, the C-terminal few amino acids encoded by a *V* gene are part of the *V-J* or *V-D-J* junction, and would be predicted by this model to interact primarily with peptide rather than MHC determinants.

In addition, in Table 1 on page 397, the penultimate column should be headed γ rather than ν.

The molecular genetics of embryonic pattern formation in *Drosophila*

P. W. Ingham
Nature **335,** 25-34 (1988).
THE labels were omitted from Fig. 2 in this Review Article, and are shown below in a black-and-white version of the figure.



Errata

Sensory transmitters regulate intracellular calcium in dorsal horn neurons

M. D. Womack, A. B. MacDermott & T. M. Jessell
Nature **334,** 351-353 (1988).
IN this letter, the two sentences beginning on line 29 on page 353 should read: "Both classes of transmitters appear to regulate $[Ca^{2+}]_i$ in these neurons, albeit by many different mechanisms. Substance P increases $[Ca^{2+}]_i$ in many dorsal neurons by mobilizing intracellular Ca^{2+} stores.

Global fire at the Cretaceous–Tertiary boundary

W. S. Wolbach, I. Gilmour, E. Anders, C. J. Orth & R. R. Brooks
Nature **334,** 665-669 (1988).
ON page 666, the third line from the bottom should read: "The soot content rises even more sharply across the boundary: 11, 1930 and 97 p.p.m."