

a common role in a process whose only feature may be an axial arrangement of tissues. It would thus also be premature to surmise that, if such homoeobox sequences are truly ancestral, the conservation of such a sequence reflects a common ancestral lineage for invertebrates and vertebrates in the development of segmentation controlled by a common, homologous DNA sequence.

However, alternative possibilities exist. Leaving aside convergent evolution, an ancestral homoeobox sequence may have been usurped or recruited for different functions in different morphogenetic processes in separate evolutionary lineages, and although invertebrates and vertebrates possess a common homoeodomain, it may possibly be involved with quite different aspects of development. Given a possible DNA-binding role for the homoeobox sequence¹² the homoeo-product may have a common function within different morphogenetic processes in separate evolutionary lineages, thus explaining its continued conservation.

Therefore, it seems to us somewhat premature to associate homoeobox sequences wholeheartedly with vertebrate segmentation and the control thereof. There is clearly an inherent danger in establishing such a model. It is too soon to rule out the role of homoeoboxes in morphogenetic processes other than segmentation.

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T-cell receptor genes and ataxia telangiectasia

SIR — The nature of the T-cell antigen receptor has been recently discussed in *News and Views*¹. The genes for the β chain of the T-cell receptor have been cloned and mapped and human chromosome 7, bands 7p13-21², while the α -chain genes still await chromosomal assignment. We wish to draw attention to some observations which might indirectly bear on the latter issue as well as on the pathogenesis of immunodeficiency in human disease.

It is known that in lymphocytes from individuals with the chromosomal instability syndrome ataxia telangiectasia (AT), 7-14 translocations appear to be a nonrandom event (their frequency is more than 40 times higher than expected)³⁻⁶. Break-

points on these chromosomes are also highly nonrandom, involving bands 7p14 (consistently with the position of the T-cell receptor β -chain genes), 7q35, 14q12 and 14q32 (consistently with the known position of the immunoglobulin heavy-chain genes). The relative frequencies of breaks at 7p14 versus 7q35, and at 14q12 versus 14q32 are 1:1 and 3:1, respectively³⁻⁶. It is reasonable to speculate that the spontaneous breakages at bands 7p14 and 14q32 in AT lymphocytes originate from faulty rearrangements of the T- and B-cell receptor genes which map there. This also suggests that these genes are normally rejoined, during rearrangement, by the same repair mechanism which is defective in AT, thus providing a basis for the T- and B-cell immunodeficiency characteristic of this syndrome⁷. The fact that 7-14 translocations are found in T-lymphocytes might indicate that both chromosomes contain genes which rearrange in these cells. The nonrandom breakpoints at bands 7q35 and 14q12 may reflect the positions of two different sets of rearranging T-cell receptor genes, for example the α -chain and the so-called non- α genes¹.

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Medical versus animal antibiotics in resistance

SIR — In his letter on "animal antibiotics", A. H. Linton¹ says that "the present debate [on resistance] . . . hinges on whether or not subinhibitory, rather than prophylactic or therapeutic levels of antibiotics play the major role". I believe that subtherapeutic, rather than subinhibitory, levels fed to animals are central to the present debate. Even though there is laboratory work on subinhibitory levels, these low levels are not added to animal feeds.

As cited by Linton¹, there is a large reservoir of R plasmid-carrying indigenous flora in the intestines of animals², but these flora are not necessarily a significant source of resistance for human pathogens. For example, ampicillin-resistant *Neisseria gonorrhoeae* probably derives its resistance from human origins³.

The suggestion that the energy cost to bacteria of carrying R plasmids places them at a disadvantage has been negated in some studies, but has found support in others, as discussed by Timoney and Linton⁴. Rough cultures of *Salmonella choleraesuis* var.

kunzedorf, with associated reduced virulence, were found to be better recipients of R factors than were their smooth virulent parental counterparts.⁵

Richmond and (K.B.) Linton⁶ found that sewage from hospitals contained more resistant organisms than did domestic sewage. They concluded that the main selective pressure for tetracycline-resistant organisms was from medical rather than veterinary use of antibiotics.

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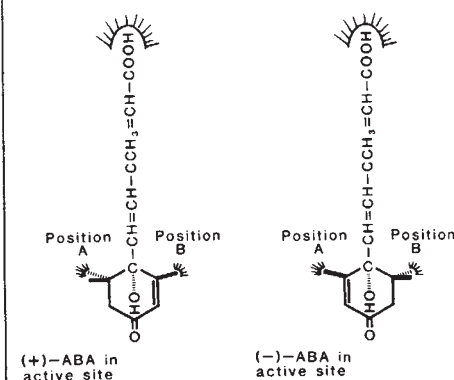
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Globulin flexibility worked out by hand

SIR — Pirie's recent note¹, about the flexibility of globulin molecules and the comfort of transposing left and right shoes, misses the main point of the original model². It proposes that (-)abscisic acid can fit into the normal active site of a receptor (affecting growth inhibition) which is normally filled by natural (+)ABA, provided that the 2'-methyl group of the (-) can occupy the site of a 6'-methyl group of the (+), and a 6'-methyl of the (-) fits into the place which is occupied by the 2'-methyl of the (+). This transposition could occur without distortion.

It is analogous to putting the little finger of a left hand into the thumb of a right glove and one's thumb into the little finger's position. The procedure can be carried out without discomfort and provides an appropriate mode for the similar action of the two enantiomers of ABA.

Pirie puts his foot in; a more even-handed approach is suggested:



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