TROELSTRA *ET AL.* REPLY—We agree with McCoy and Stanley's view that the age at the bottom of the Tyro Basin core must be considerably younger than we had assumed in our papers, which were mainly based on shipboard observations. Shore-based analyses, still in progress, have produced different data. Because of the different bottom water regimes in the Tyro and Kretheus basins, we would like to consider each basin separately.

First, we want to stress that, at present, conditions suitable for sapropel formation, namely an anoxic and reducing environment due to halocline water stratification, do exist in the Tyro basin. Laboratory studies on core P46 indicate that sediments were not deposited in exclusively pelagic conditions. The most important argument for this is the presence of fining-upward sequences in the core, indicating transport by mass-flows. Furthermore, faunal and floral composition through the core is rather uniform and suggests a relatively warm climate. If the age at the bottom of the core were 80,000 yr, a fluctuating faunal and floral pattern would be expected, due to climatic changes. Moreover, the presence at the bottom of the core of well-preserved algae, one species still in possession of bright green-coloured chlorophyll, indicates that little or no diagenesis took place. This is contrary to what one would expect if the sediments were deposited at 80,000 yr BP.

In contrast to the Tyro Basin, bottom waters in the Kretheus Basin are well-ventilated at present. Core P45 consists of alternate dark-coloured (? resedimentated sapropel) and lighter-coloured sediments. The latter contain abundant sand-sized grains of metamorphic rock indicating that sedimentation in the Kretheus Basin is also governed by mass-flow transport. Planktonic foraminiferal composition through the core is comparable with that in core P46.

We conclude that sedimentation rates in both basins must be high. At this stage, pending ^{14}C analyses, an entire Holocene age for both cores cannot be excluded. Resedimented S1 may be one of the major contributors to the sapropelic intervals in the cores, as correctly assumed by McCoy and Stanley.

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Sequence similarity among retroviruses—erratum*

RECENTLY Toh *et al.*[1] reported the existence of amino acid sequence similarity between the reverse transcriptase of certain retroviruses and the DNA polymerases of cauliflower mosaic virus (CaMV) as well as a region of hepatitis B virus (HBV) that probably encodes a DNA polymerase. For completeness, we draw attention to other regions of protein similarity between retroviruses (human T-cell leukaemia virus, HTLV; Moloney murine leukaemia virus, Mo-MuLV; and Rous sarcoma virus, RSV), HBV and CaMV that were not reported by Toh *et al.*

The sequences are presented in Table 1 and the relative positions of the similar regions indicated in Fig. 1. The common set of amino acids discussed by Toh *et al.* is conserved in the amino-terminal region of all retrovirus reverse transcriptases studied (regions I–III). These common sequences are located near the amino-terminus of the HBV protein and the centre of the CaMV polymerase. Another region of similarity, IV, not discussed by Toh *et al.*, is also found in all retroviruses and is present in the CaMV protein. Region V, reported by Toh *et al.*, to be present in Mo-MuLV and RSV, is also found on HTLV. We also draw attention to sets of amino acids that are conserved with respect to sequence and relative position between the reverse transcriptases of HTLV and Mo-MuLV (M1–M4) and HTLV and RSV (R1–R4).

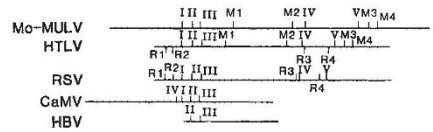

Fig. 1 Alignment of the polymerase gene products among five different viruses, depicting the regions shown in Table 1.

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Table 1 Alignment of the similar regions in polymerase gene products among different viruses

REGION	VIRUS		SEQUENCE
I	HTLV	114	DLRDAFFQTPL
	Mo-MuLV	270	DLKDAFFCLRL
	RSV	107	DLKDCFFSIPL
	CaMV	355	DCKSGFWQVTL
II	HTLV	150	WKVLPQGFKNSPTLF
	Mo-MuLV	305	WTRLPQGFKNSPTLF
	RSV	143	WKVLPQGMTCSPTIC
	CaMV	384	WNVVPFGLKQAPSIF
	HBV	25	FRKIPMGVGLSPFLL
III	HTLV	184	IL.QYMDDILLASPSH
	Mo-MuLV	338	ILLQYVDDLLLAATSE
	RSV	177	ML.HYMDDLLLAASSH
	CaMV	413	CC.VYVDDILVFSNNE
	HBV	57	LAFSYMDDVVLGAKSM
IV	CaMV	331	DAYNLPNKDELLTLIRGKKIFSSF
	Mo-MuLV	727	EGKEIKNKDEILALLKALFLPKRL.S
	HTLV	541	LALGTFQGRSSQAPFQALL.LP.RLLS
	RSV	556	FFTEGNDVADSQATFQAY.PL.REAK
V	HTLV	677	LHVWVDTFSGAISA
	Mo-MuLV	930	LLVEIDTFSGWIEA
	RSV	650	LAVTVDTASSAIVV
M1	HTLV	263	QALL
	Mo-MuLV	469	QALL
M2	HTLV	499	PPHKSAQRAELLGL
	Mo-MuLV	673	PAGTSAQRAELIAL
M3	HTLV	720	TDNGPAYIS
	Mo-MuLV	974	TDNGPAFVS
M4	HTLV	745	HVPYNPTSSGLVERSN
	Mo-MuLV	799	HCAYRPQSSGQVERMN
R1	HTLV	37	LQALQHLVRKALEAGHIEP
	RSV	31	LVALTQLVEKELQLGHIEP
R2	HTLV	61	NNPVFPVKKANGTWRFIHDLRATN
	RSV	55	NTPVFVIRKASGSYRLLHDLRAVN
R3	HTLV	572	HVRSH
	RSV	546	HVRSH
R4	HTLV	660	IWQGDLT
	RSV	633	IWQTDFT

Molony murine leukaemia virus, Mo-MuLV[2]; human T-cell leukaemia virus, HTLV[3]; Rous sarcoma virus, RSV[4]; cauliflower mosaic virus, CaMV[5]; and hepatitis B virus, HBV[6]. Common amino acids are boxed and conservative substitutions with respect to HTLV are underlined. For each sequence, the first amino acid is numbered.

* This paper was published in the issue of 17 May without title and Fig. 1 due to printing errors.