clear that it was even in existence at that time, as the sea level would have been about 45 m below its present level. The angular target for a voyage directly from New Ireland to the Buka–Bougainville group would have been 30°, and using the island stop-overs, some stages would have had targets as little as 8°.

Once on Buka, the crossings to most of the main islands of the northern Solomons would have been easy as far south as San Cristobal, 1,100 km away. The gap to Santa Cruz probably marked the boundary of Pleistocene occupation. Waisted axe-like tools similar to those dated to 40 kyr ago[4] at the Huon peninsula on the New Guinea coast opposite New Britain have been found on several of the islands of the group. The diversity of human genetics on Bougainville indicates that the islands must have been occupied at least 10 kyr ago[5], a figure that until recently seemed impossible. The colonization of the large islands of the Bismarck and northern Solomons chains occurred soon after that of the Australian–New Guinea continent itself[6]. A sea-crossing capacity, probably denied to the south-east Asian *Homo erectus* populations, was one of the hallmarks of modern man in the region, the events occurring at about the same time that modern *H. sapiens* groups colonized western Europe.

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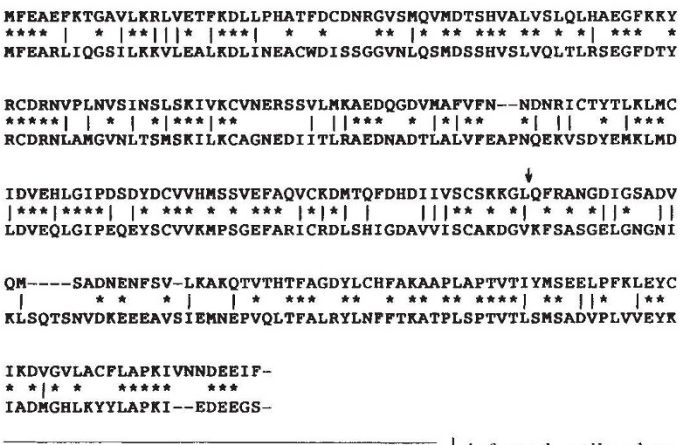
Viral proliferating cell nuclear antigen

SIR—We have identified in the genome of the baculovirus *Autographa californica* nuclear polyhedrosis virus (AcMNPV), a gene that encodes a protein similar to proliferating cell nuclear antigen (PCNA), also termed cyclin[1] or the polymerase-δ auxiliary protein[2,3], which is associated with DNA replication in mammalian cells.

The gene is the previously characterized[4] ETL, which is similar in size to rat PCNA and has 42 per cent amino-acid identity. Conserved sequences are distributed throughout the protein and very few insertions or deletions are required to generate the optimal match (see figure).

Evidence that this viral PCNA is involved in DNA replication is provided by the mutant of AcMNPV, vETLβgal[4], which contains the *Escherichia coli* β-galactosidase gene (*lacZ*) fused to the amino-terminal 165 amino acids of the viral PCNA. This mutant is viable in both insect cell cultures and in perorally infected larvae, but all late viral gene expression, which requires DNA replication, is 4–6 hours later in vETLβgal-infected cells than in wild-type virus-infected cells[4].



Comparison of the amino-acid sequence of ETL[4] (top lines) with rat PCNA[9]. Asterisks, identical residues; lines, substitutions which occur at greater than average frequency among related proteins. Vertical arrow, point at which the ETL sequence is disrupted in vETLβgal.

We have recently confirmed that vETLβgal DNA replication is delayed. Because the mutant virus is viable, however, viral PCNA does not appear to be absolutely required for DNA replication, at least in proliferating cell cultures. Perhaps, viral PCNA is essential for virus replication in nonproliferating, quiescent cells.

In addition to inducing the host DNA polymerase-α[5,6], baculoviruses encode a DNA polymerase[7] which is sensitive to aphidicolin[5,8] and has an associated 3′-to-5′ exonuclease[8], properties shared with DNA polymerases-δ. The induction of these replication-related enzymes may contribute to the ability of AcMNPV to establish an S-phase environment and thereby replicate efficiently in proliferating and quiescent host tissues.

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Dystrophin digest

SIR—We have recently reported[1] the localization of a protein of relative molecular mass 210,000 (210K) sharing an antigenic determinant with the 400K dystrophin on the surface membrane of normal human skeletal muscle fibres. Hoffman *et al.*[2] have since suggested that the 210K protein may be a degradation product of dystrophin, and our own data now support this suggestion.

In previous experiments, we found the 210K protein but not the 400K protein in crude and DEAE-cellulose purified low-ionic-strength extracts of skeletal muscle. But when we isolated the protein samples from frozen (−80 °C) or fresh muscle specimens by direct solubilization of tissues in a gel-loading buffer, containing 10 per cent SDS as described by Hoffman *et al.*[3], instead of the 1 per cent SDS used in our previous report, an immunoreactive 400K band could be recovered with our antibody. Further, we found that the antibodies produced by Kunkel's group (30K, 60K) crossreacted with the 210K protein in our low-ionic-strength extract of skeletal muscle.

We now believe that the 210K protein is probably a part of 400K dystrophin, and that native dystrophin, which is extremely susceptible to proteolysis *in vitro* [4], can easily be digested during the preparation process. The digestion may occur at fixed break point(s), because the 210K protein is always observed[1] as a sharp, single band. Thus, the 210K protein presumably represents a stable portion of the 400K dystrophin.

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