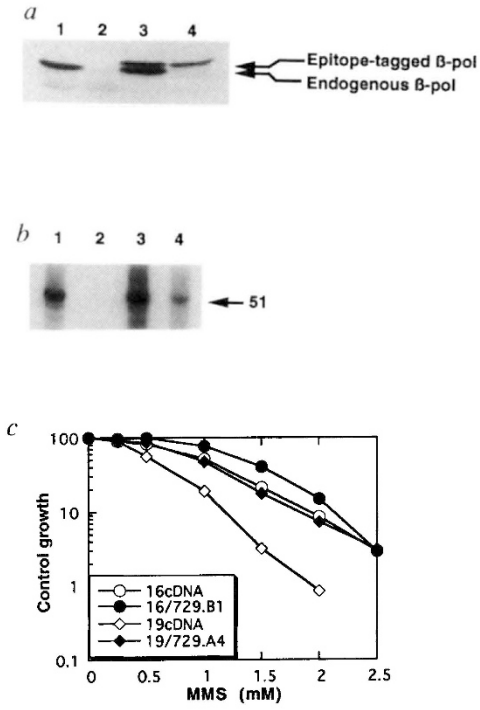

FIG. 4 Genetic complementation of BER deficiency with expression of β-pol minigene. Wild-type and β-pol-deficient cell lines were transfected with pCDNA3 (Invitrogen) or the β-pol expression vector pRS729, respectively. Cells were selected in the presence of 80 $\mu g\,ml^{-1}$ hygromycin and 600 $\mu g\,ml^{-1}$ G418 for 28 days, and further selected for 14 days by limiting dilution (0.3 cells per well) in 96-well microtitre plates. Clones harbouring pRS729 were further screened for expression of β-pol by immunoblot analysis. *a*, Immunoblot analysis of nuclear extract with β-pol-specific polyclonal antibody. 250 μg of nuclear extract from wild-type Mβ16tsA cells (lane 1), β-pol deficient Mβ19tsA cells (lane 2), 16/729.B1 cells (lane 3) or 19/729.A4 cells (lane 4) was resolved by 12.5% SDS-PAGE and probed for β-pol as described[7]. A photograph of the autoradiogram is shown. Epitope-tagged and endogenous β-pol are indicated by the arrows. *b*, Uracil-initiated BER *in vitro*. Nuclear extract from wild-type Mβ16tsA cells (lane 1), β-pol-deficient Mβ19tsA cells (lane 2), 16/729.B1 cells (lane 3) or 19/729.A4 cells (lane 4) were reacted with a 51-base-pair synthetic DNA substrate as described in Fig. 3*a* legend. *c*, Alkylating agent sensitivity. Cells were exposed to the DNA damaging agent MMS as described in the legend to Fig. 3*b*. 16cDNA and 19cDNA cells are Mβ16tsA and Mβ19tsA cells, respectively, stably transfected with the control vector pCDNA3; 16/729.B1 and 19/729.A4 cells are Mβ16tsA and Mβ19tsA cells, respectively, stably transfected with the β-pol expression vector pRS729. The β-pol expression vector pRS729 will be described elsewhere (R.W.S. and S.H.W. manuscript in preparation).

nuclear extract from 19/729.A4 cells was now found to be proficient in BER (Fig. 4*b*, compare lanes 2 and 4). This genetic complementation is also observed *in vivo*, as the hypersensitivity of the β-pol-deleted cells to MMS was completely reversed in the 19/729.A4 cells (Fig. 4*c*). Further, 16/729.B1 cells exhibit an increased resistance to MMS, suggesting that β-pol may be the rate-limiting factor in the BER pathway *in vivo*.

It has been previously thought that β-pol functions in repair synthesis in a manner analogous to the *Escherichia coli* PolA encoded DNA polymerase I, which also is required for strand-displacement synthesis following the removal of RNA primers from newly replicated DNA. PolA mutants are sensitive to a wide variety of DNA-damaging agents including MMS and ultraviolet radiation[12]. Our studies with β-pol null mutants, however, provide no support for a role of β-pol in either DNA replication or the repair of damage from ultraviolet light (for example, nucleotide-excision repair), as β-pol mutant cells grow normally and are not sensitive to ultraviolet radiation. Rather surprisingly, we find that mammalian β-pol has a highly specific role in BER *in vivo*. Whereas DNA polymerases-α, -δ and -ε are unable to substitute for β-pol in the BER reaction *in vitro*, similar genetic studies will be required to delineate their role precisely in DNA repair *in vivo*. The apportionment of β-pol to BER presumably reflects the importance of this repair pathway in maintaining the fidelity and integrity of genomic DNA in mammalian cells. □

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ERRATUM

A nesting dinosaur

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