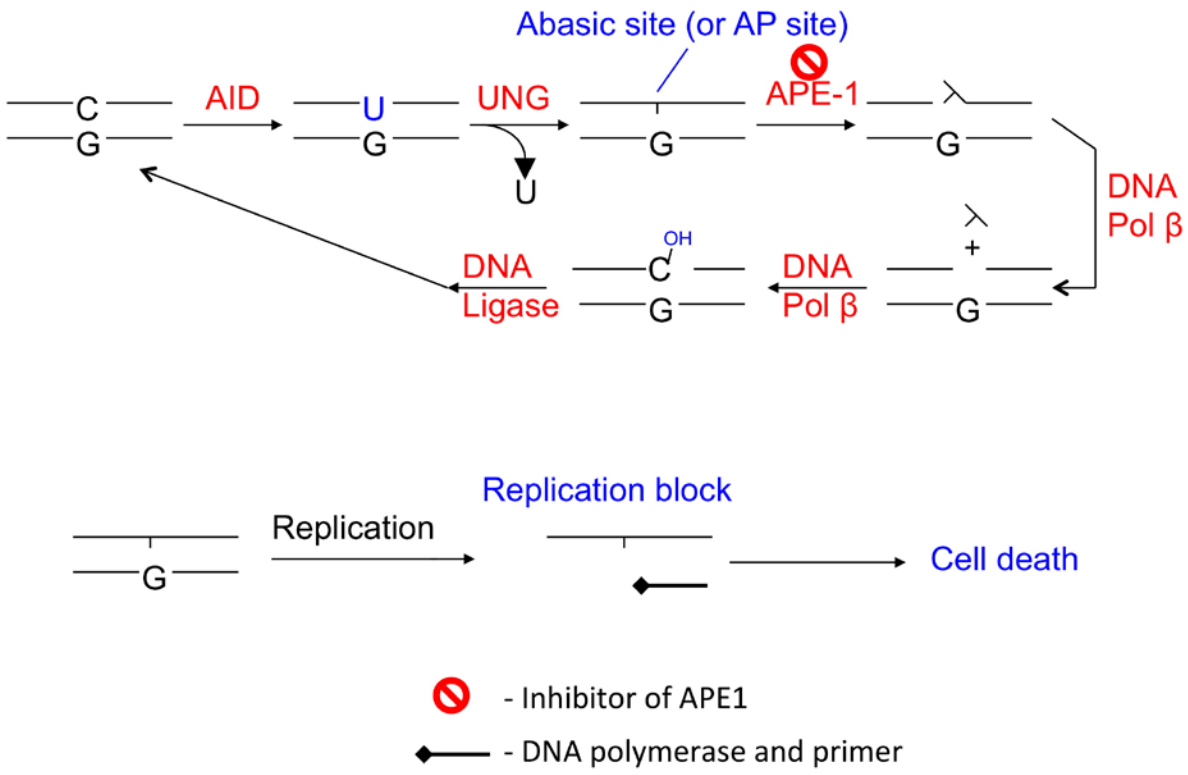

S1 Figure. A model for cell killing by AA3.

Top- Rationale for cell killing by AA3. B-NHL cells overexpress activation-induced deaminase (AID) which converts cytosine into uracil and results in the accumulation of uracils in their genome. These cells also contain uracil-DNA glycosylase (UNG) that removes these uracils generating AP sites. AP sites are further processed by the AP endonuclease (APE-1) and repaired through the actions of DNA polymerase β.

Bottom- AA3 inhibits the activity of APE1 by reacting with AP sites and blocks the progress of replication fork causing cell death.