**Figure S1** Co-precipitation of ING1-ING5 with Lamin A. Lysates from HEK293 cells transfected with ING1-ING5 or GST expression constructs were immunoprecipitated with preimmune non-specific antibody (NS Ab) or anti-lamin A (α-LMNA) and precipitates were blotted with chicken a, α-ING1 b, α-ING2 c, α-ING3 d, α-ING4 e, α-ING5 or f, α–ING1 and visualized with α-IgY-HRP.
Figure S2 Interaction with lamins regulates ING1 induction of apoptosis. GFP and ING1 constructs were cotransfected into HEK293 cells, which were harvested 48h later and stained with propidium iodide. Cells with a sub-G1 DNA content (a measure of apoptosis) are highlighted by the arrows. The LID induced apoptosis by itself at a similar level to wild type ING1, while ING1ΔLID promoted apoptosis less effectively. Induction of apoptosis by increasing linkage between lamin A and HAT/HDAC complexes (ING1 transfection) or by blocking linkage (ING1ΔLID and LID transfections) is consistent with levels of lamin A being important for normal cell metabolism since both decreases21,22 and increases35 in lamin A levels induce sensitivity to apoptosis. Values presented are the average of three independent assays.
Figure S3 Effects of disrupting ING1-Lamin A interactions on nuclear membrane structure. EM analyses of Hs68, HGPS and ING1-transfected cells. a-b, Compared to Hs68 (a) cells, invaginations highlighted by the arrowheads were frequently observed in the nuclear envelope of Prog1 fibroblasts (b) as previously described for HGPS cells, which were also larger in size suggesting that they were entering senescence despite being at a lower passage level than the other primary fibroblast strains. c-h, HEK 293 cells transfected with GFP (c,f) or ING1ΔLID (d,e,g) were fixed and stained 48h after transfection. In contrast to the GFP control which showed morphology similar to untransfected cells with a largely continuous layer of peripheral heterochromatin (compare panels a & c), the LID deletion ING1 construct frequently induced nuclear membrane invagination, lobulation or multiple nuclei, consistent with disruption of the lamin A-ING1 interaction and subsequent altering of the distribution of peripheral heterochromatin contributing to nuclear membrane instability. Panels f-h show higher magnification micrographs that highlight discontinuities in the peripheral heterochromatin near the nuclear envelope (arrowheads) that have been reported in HGPS and in lamin A knockout cells. The arrows in panels d & e compare regions of the NE showing differences in peripheral chromatin density and the horizontal arrow in panel h shows distention of the outer nuclear membrane. All analyses were initially done using a blind experimental protocol in which microscopy was done and recorded for coded samples. Magnifications used in panels a-d are the same and the scale bar equals 2µm. The scale bar = 2µm for panel e and 0.3 µm for panels f-h.
**Figure S4** Subcellular localization of ING1 during mitosis. Fibroblasts were fixed and stained for ING1 proteins (green) and DNA (DAPI, blue). a, ING1 proteins are nuclear during interphase and b, early prophase but start dissociating from nuclear structures in c, pro-metaphase and remain distributed throughout the cell in d, metaphase and e, anaphase. They begin reassociating with nuclear structures in f, telophase. This is consistent with ING1 binding lamin A/C since transit from the nucleus to cytoplasm is similar to the dynamics of lamin A/C depolymerization. Entry into the reforming nucleus appears to slightly precede lamin A/C repolymerization during mitosis which is believed to occur after cytokinesis and staining appears in particulate sites consistent with binding a subpopulation of lamin A. The scale bar represents 5 µm.
Figure S5 The subcellular localization of ING1 by lamin A potentiates ING1 function. Model for how tethering of ING proteins by Lamin A is needed to stabilize ING proteins and localize them in the nucleus for use as stoichiometric components of HAT and HDAC complexes. Nuclear transport and chromatin tethering by interaction with lamin A is followed by recruitment of acetylation and/or deacetylation complexes and altering histone acetylation levels and chromatin structure. If this function is altered by loss of Lamin A interaction and subsequently decreased levels of ING and Rb proteins, the expected phenotype would be an altered state of chromatin compaction and distribution, with subsequent altered susceptibility to apoptosis, both of which are seen in HGPS cells and in response to altered levels of both Rb and ING proteins. ING binding to trimethylated histone H3, stoichiometric residence in HAT and HDAC complexes, ING1 phosphorylation, binding to 14-3-3 and exit from the nucleus and transport by karyopherins have all been previously described (reviewed in reference 8).
Figure S6 Disrupting ING1-Lamin A interactions alters histone acetylation.  

a. Intact ING1-Lamin A

b. Disruption by excess ING1

c. Disruption by LID expression

d. Disruption by LID mutation

Increased acetylation of both histone H3 and histone H4 has been reported upon overexpression of ING1 by needle microinjection. Expression of the LID region blocks the ability of ING1 to interact with lamin A, resulting in loss of HDAC targeting. ING1 lacking the LID partially localizes to the nucleus and that fraction that does partially prevents interaction between ING1 and lamin A/HDAC complexes resulting in a similar but less obvious phenotype.