SUPPLEMENTAL TABLE 1. Cell lines evaluated in this study and relevant mutations.

Cell line	Mutation
IMR-05	neurofibromatosis type 1
IMR-32	N/A
NBL-S	anaplastic lymphoma kinase
NB-EBc1	tumor protein p53
CHP-134	N/A
CHP-212	anaplastic lymphoma kinase
SMS-SAN	tumor protein p53
NB-69	tumor protein p53
KELLY	N/A
NGP	N/A
NB-1691	N/A
SK-N-SH	N/A
SK-N-BE(2)-C	N/A
NLF	N/A
SK-N-DZ	N/A
SK-N-BE(2)	N/A
SK-N-AS	N/A
NB-SD	N/A
SK-N-FI	N/A

*N/A=not applicable

SUPPLEMENTAL TABLE 2. EC_{50} values of radionuclide therapy in IMR-05 and NLF

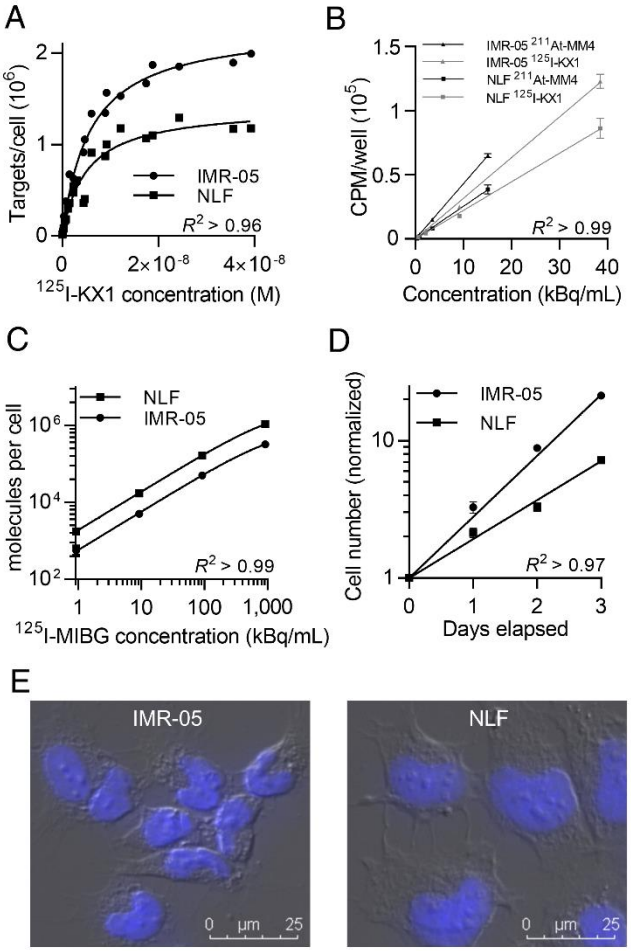
	IMR-05	NLF
[^{211}At]MM4	620 ± 30 pCi/mL	20 ± 4 nCi/mL
[^{125}I]KX1	12.5 ± 0.9 nCi/mL	980 ± 60 nCi/mL
[^{211}At]NaAt^{x}	22.6 ± 1.4 nCi/mL	2.1 ± 0.2 μCi/mL
[^{131}I]KX1	82.8 ± 0.8 nCi/mL	3.8 ± 0.1 μCi/mL
[^{125}I]MIBG	2.2 ± 0.2 μCi/mL	8.2 ± 0.5 μCi/mL

*Values are reported as mean ± SEM.

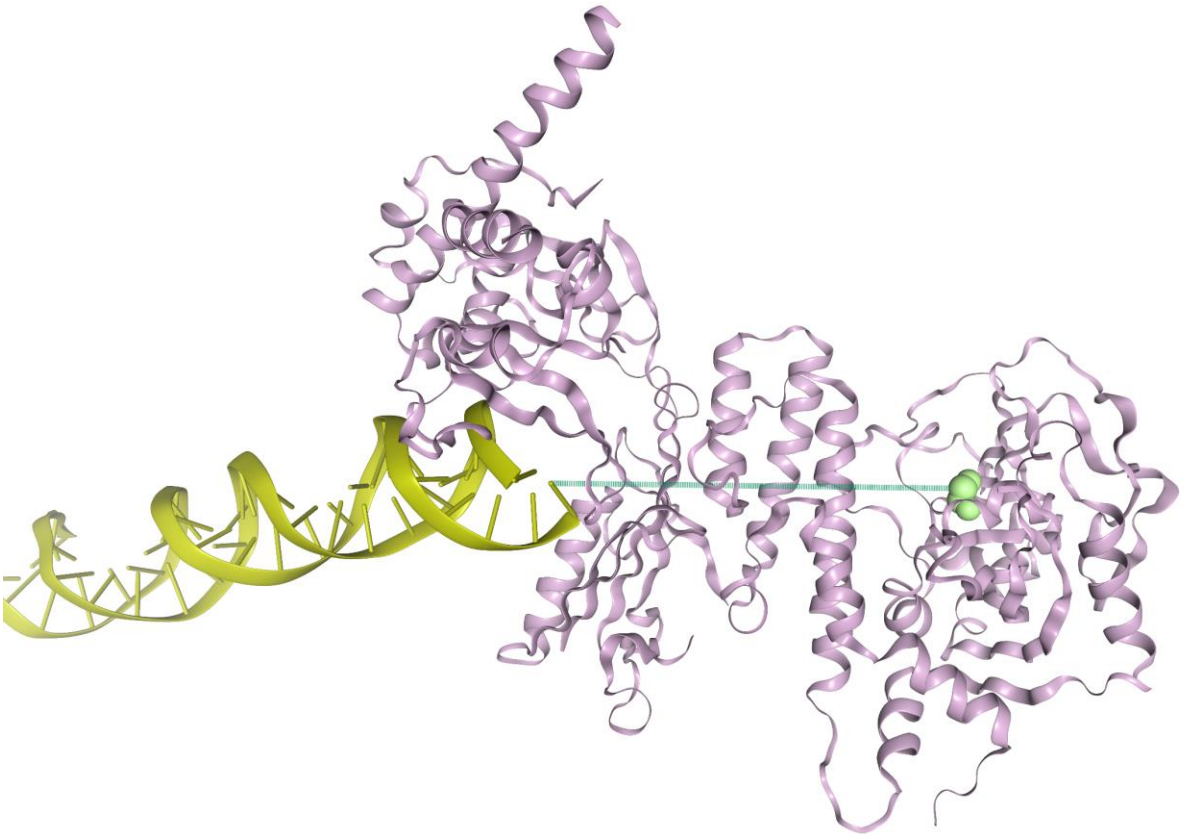
SUPPLEMENTAL TABLE 3: Parameters of IMR-05 and NLF in the linear-quadratic model

	IMR-05		NLF	
	α	β	α	β
[^{125}I]KX1	3.8 ± 0.2	N/A	0.42 ± 0.03	N/A
[^{131}I]KX1	2.00 ± 0.03	N/A	0.25 ± 0.01	N/A
[^{125}I]MIBG	0.65 ± 0.09	N/A	0.23 ± 0.02	N/A
[^{211}At]MM4	13.6 ± 0.6	N/A	1.3 ± 0.2	N/A
[^{211}At]NaAt^{x}	3.7 ± 0.2	N/A	0.86 ± 0.08	N/A
External gamma	0.81 ± 0.06	0.22 ± 0.06	0.25 ± 0.01	N/A

*Values are reported as mean ± SEM. N/A=not applicable (approximately zero)



SUPPLEMENTAL FIGURE 1. (A) Radioligand saturation binding study with [^{125}I]KX1 revealed higher B_{max} in IMR-05 (2.30 ± 0.07 x 10^6 targets/cell) than NLF (1.41 ± 0.07 x 10^6 targets/cell) but similar K_d (5.8 ± 0.5 nM in IMR-05; 5.1 ± 0.8 nM in NLF). **(B)** Comparison of target binding affinity between [^{125}I]KX1 and [^{211}At]MM4 under non-saturating conditions yielded similar K_d of [^{211}At]MM4 in IMR-05 (4.3 ± 0.5 nM) and NLF (4.5 ± 0.9 nM). **(C)** Direct measurement of cellular [^{125}I]MIBG uptake at cytotoxic concentrations showed 3.29 ± 0.07 times greater uptake in NLF compared to IMR-05. **(D)** IMR-05 and NLF cell lines demonstrated exponential growth pattern with doubling times of 16.2 ± 0.1 hours and 25.5 ± 0.5 hours, respectively. **(E)** Bright field and fluorescence microscopy with DAPI staining allowed measurements of nuclear and cellular radii in IMR-05 (6 µm and 8 µm) and NLF (9 µm and 12 µm) cells.



SUPPLEMENTAL FIGURE 2. Three-dimensional structure of PARP1 (purple) bound to DNA (yellow). The distance from the PARP1 active site (green) to DNA was measured at 50.0 Å (dotted line) (*16-18*).

SUPPLEMENTAL EQUATION 1. Calculation of the binding affinity (K_d) of [^{211}At]MM4 under non-saturating conditions

When [Ligand] << K_d (less than 1%),

$$(Specific\ binding) = \frac{B_{max}[Ligand]}{K_d + [Ligand]} \approx \frac{B_{max}[Ligand]}{K_d}$$

Then, solving for B_{max} yields

$$B_{max} = \frac{(Specific\ binding)(K_d)}{[Ligand]} = \alpha \cdot K_d$$

where α is the slope of the specific binding vs. [Ligand] plot.

Since B_{max} is shared between [^{125}I]KX1 and [^{211}At]MM4,

$$\alpha_{(MM4)} \cdot K_{d(MM4)} = \alpha_{(KX1)} \cdot K_{d(KX1)}$$

Therefore, solving for $K_{d(MM4)}$ yields

$$K_{d(MM4)} = \frac{\alpha_{(KX1)} \cdot K_{d(KX1)}}{\alpha_{(MM4)}}$$

SUPPLEMENTAL EQUATION 2. Calculation of cumulated activity

Let

B = number of bound molecules per cell at equilibrium
A_s = specific activity (Bq/moles)
N_A = Avogadro's number
$t_{1/2}$ = physical half-life
T = duration of treatment
t = time

Then, the activity of bound molecules in the cell can be represented as a function of time:

$$A(t) = \frac{B}{N_A} \cdot A_s \cdot e^{-\lambda t}$$

where $\lambda = \ln(2)/t_{1/2}$.

Then, the cumulated activity $\tilde{A}$ can be obtained by integrating A(t) over the duration of treatment:

$$\tilde{A} = \int_0^T A(t)dt = \int_0^T \frac{B}{N_A} \cdot A_s \cdot e^{-\lambda t} dt = \frac{B}{N_A} \cdot A_s \cdot \left(\frac{1 - e^{-\lambda T}}{\lambda}\right)$$