**Supplementary Figure Legends: Ladjohounlou *et al.***

**SUPPLEMENTARY FIG. S1.**(A) 212Pb (red) decays into 212Bi by emitting a beta minus particle. Then, 212Bi decays into 208Tl or 212Po by α or β decays, respectively. Finally, 208Tl and 212Po decay into stable 208Pb through α or β disintegration. The energy of α particles emitted by 212Bi and 212Po are 6.2 MeV and 8.8MeV, while the energy of β particles ranges between 0 and 2.2 MeV. 212Pb display the longest physical half-life in the chain (10.64h *vs* 60min or less for its daughters). After 3h, the decay products are in transient equilibrium with the parent radionuclide. (B)213Bi (red) decays into 209Tl (through alpha decay) or 213Po (beta decay). Both 209Tl and 213Po decay to 209Pb by β or α disintegration, respectively. Finally, 209Pb decays into the stable 209Bi. The physical half-life of 213Bi is 45min, and that of its daughters ranges from seconds to 3.2h for 209Pb.The energy values of α particles emitted by 213Bi and 213Po are 5.9MeV and 8.4MeV, while the energy of β particles ranges between 0 and 1.9 MeV. The energy of α and β particles emitted by 212Pb, 213Bi and daughters in tissues ranges from a few tens of micrometers for α particles, to centimeters for β particles. (C) 125I (red) decays to stable 125Te (through electronic capture decay). 125I has a physical half-life of 59.4 days. Electronic capture is associated with the emission of a mean number of 21 Auger electrons with energy lower than 36 keV. Decay also produces 3.8 to 31.8 keV of soft X-rays.

**SUPPLEMENTARY FIG. S2. Clonogenic cell survival of donor and recipient cells after exposure to unlabeled mAbs**. (A) Clonogenic cell survival of A-431CEA donor cells exposed to 0-27 µg/mL of unlabeled anti-CEA or anti-HER2 mAbs for 90min and in the corresponding recipient cells. (B) Clonogenic cell survival of SK-OV-3MISII donor cells exposed to 0-27 µg/mL of unlabeled anti-MISRII mAb and in the corresponding recipient cells. Results are the mean ± SD of four experiments performed in triplicate. \*p<0.05, \*\*p<0.01, \*\*\*p<0.001 compared with untreated cells.

**SUPPLEMENTARY FIG. S3**. **DNA double strand break formation in SK-OV-3MISRII and A-431CEA cells after RIT**. (A) Foci were classified in large, medium and small according to their size, as reported in the table and shown in the representative immunofluorescence images (left and lower panels). (B) The average number of53BP1 foci per cell was determined by immunofluorescence in 100 A-431CEA cells exposed to 0.5MBq/mL 212Pb-anti-CEA mAb, 212Pb-anti-HER2 mAb, or untreated. (C) The number of micronuclei per binucleated cell was determined in A-431CEA cells after exposure to 0.5MBq/mL 212Pb-anti-CEA mAb, 212Pb-anti-HER2 mAb, or untreated. Results are the mean ± SD of three experiments performed in triplicate. \*p <0.05, \*\*p <0.01, \*\*\*p <0.001, compared with untreated cells.

**SUPPLEMENTARY FIG. S4. Signaling pathways activated in donor and recipient cells after exposure to 213Bi-anti-MISRII mAb alone or with MBCD**. (A) The phosphorylation level of the 46 activated kinases was assessed in SK-OV-3MISRII recipient cells incubated with medium from donor cells exposed or not (untreated) to 0.5MBq/mL 213Bi-anti-MISRII mAb alone, or 0.5MBq/mL 213Bi-anti-MISRII mAb + MBCD using the Human Phospho-Kinase array (Proteome Profiler Array; R&D Systems, Minneapolis, MN). For each kinase, the ImageJ software was used to determine the pixel intensity. (B) Analysis of phosphorylated p38 and NF-ĸB by western blotting in AN3CA donor and recipient cell extracts. CM, conditioned medium from donor cells.

**SUPPLEMENTARY FIG. S5. Signaling pathways activated in donor and recipient cells after exposure to 213Bi-anti-MISRII mAb alone or with MBCD**. (A) Apoptosis (green) was measured in SK-OV-3MISRII donor cells pre-incubated with SP600125 (JNK inhibitor), SPB203580 (p38 inhibitor), imipramine (ASMase inhibitor) or MBCD (lipid raft disruptor) using the TUNEL Detection Kit (Promega, France). Images show results at 48h post-treatment. Nuclei are stained with Hoechst (blue). (B) The phosphorylation level of the 46 activated kinases was assessed in SK-OV-3MISRII recipient cells incubated with medium from donor cells exposed or not (untreated) to 0.5MBq/mL 213Bi-anti-MISRII mAb alone, or 0.5MBq/mL 213Bi-anti-MISRII mAb + MBCD using the Human Phospho-Kinase array (Proteome Profiler Array; R&D Systems, Minneapolis, MN). For each kinase, the ImageJ software was used to determine the pixel intensity.

**SUPPLEMENTARY FIG. S6. Signaling pathways activated in SK-OV-3MISRII donor cells exposed to 213Bi-anti-MISRII mAb + filipin and in recipient cells**. The phosphorylation level of 46 kinases was assessed in (A) SK-OV-3MISRII donor cells after incubation with medium alone (untreated), 0.5MBq/mL 213Bi-anti-MISRII mAb, or with 0.5MBq/mL 213Bi-anti-MISRII mAb + filipin, and (B) in the corresponding SK-OV-3MISRII recipient cells using the Human Phospho-Kinase Array (Proteome Profiler Array; R&D Systems, Minneapolis, MN). For each kinase, the ImageJ software was used to determine the pixel intensity.

**SUPPLEMENTARY FIG. S7. Oxidative stress proteins activated in SK-OV-3MISRII and HCT116 cells after RIT.** Oxidative stress-related proteins activated in donor and recipient cells after incubation of: A) SK-OV-3MISRII donor cells with culture medium (untreated) or with 0.5 MBq/mL 213Bi-anti-MISRII mAb and in the corresponding recipient cells; and B) HCT 116 donor cells with culture medium (untreated) or with 4 MBq/mL 125I-anti-CEA mAb and in the corresponding recipient cells. The human Cell Stress Array (Proteome Profiler Array; R&D Systems, Minneapolis, MN) was used. C) Analysis of phosphorylated NF-ĸB by western blotting using extracts of donor and recipient SK-OV-3MISRII, AN3CA cells exposed to 213Bi-anti-anti-MISRII mAb or not (untreated) or of HCT116 donor cells exposed to 125I-anti-CEA mAb and the corresponding recipient cells. CM, conditioned medium from donor cells.

**SUPPLEMENTARY FIG. S8. *In vivo* effects of MBCD on tumor growth in mice treated with low activities of 213Bi-anti-MISRII mAb.** Mice bearing intraperitoneal (i.p.) AN3CA tumor cell xenografts were treated with: i) NaCl, ii) daily i.p. injections of 300 mg/kg MBCD from day 7 to 13 post-graft, iii) one injection of 213Bi-anti-MISRII mAb at day 11, or (iv) both 213Bi-anti-MISRII mAb and MBCD. At day 14 post-graft, mice were sacrificed, tumors collected, and the total tumor mass was determined.