Figure 1. Confocal microscopy of LS180 cancer cells incubated with FITC-BSA-PCL and FITC-EGFR-BSA-PCL for 4 h.

(A) FITC-BSA-PCL (B) FITC-EGFR-BSA-PCL.The scale bars represent 20 μm (A) and 40 μm (B). Confocal microscopy showed that EGFR-BSA-PCL was significantly internalized in EGFR-overexpressing LS180 cells and exhibited strong green fluorescence. Compared with EGFR-BSA-PCL, BSA-PCL could also bind to cells, but its tumor retention was minimal and the green fluorescence was weak.

**Figure 2.** 131I uptake in LS180 cells.

(A) Time-dependent uptake of 131I in LS180 cells. The maximum 131I uptake for LS180 cells was obtained after 4 h of incubation with 131I-EGFR-BSA-PCL and 131I-BSA-PCL, but their respective maximum CPM levels were different. The 131I uptake of 131I-EGFR- BSA-PCL was higher than that of 131I-BSA-PCL. (B) 131I uptake for LS180 cells after 4 h of incubation withnanoparticles. The radioactivity count in EGFR-BSA-PCL and BSA-PCL groups was much higher than that of 131I group. As the dose of 131I-labeled nanoparticles increased, the CPM increased; in the 131I group, the radioactivity was linear and was maintained at a low level. Mean values and standard deviations are shown.

Figure 3. Changes in the animal body weight.

The animal weight change of the LS180 xenografted mice subjected to various treatments during the observation period is shown (*n* = 5). The tumor growth of the mice was monitored over 33 d after injection. Significant reduction of the body weight was found in the 131I and normal saline groups. Data was expressed as mean ± S.E.

Figure 4. Changes in tumor volume.

The tumor volume curves of LS180 colorectal cancer xenografted mice with the various treatments during the experiment period are shown in this map(*n* = 5). Rapid tumor growth was observed in the xenografted colorectal cancer nude mice from groups treated with saline or 131I. By contrast, tumors grew slowly in mice treated with 131I-EGFR-BSA-PCL or 131I-BSA-PCL.

Figure 5. **Tissue distribution of 131I-EGFR-BSA-PCL, 131I-BSA-PCL, and 131I in LS180 cells on 4h, 24h, and 72h after injection.**

(A)131I-EGFR-BSA-PCL group; (B) 131I-BSA-PCL group; (C) 131I group.**The biodistribution was counted as percentage of the injected dose per gram (% ID/g). Tissue distribution of 131I-EGFR-BSA-PCL, 131I-BSA-PCL, and 131I in nude mice with human** colorectal cancer **xenograft-bearing nude mice at 4, 24, and 72 h after injection of the different drugs (*n* = 5).**

**Figure 6. Immunohistochemical staining** of LS180 colorectal cancer.

Immunohistochemical staining of the LS180 xenograft lines with cetuximab to assess EGFR (staining of the cell membrane) expression.

**Figure 7. Histopathology of the liver, spleen, and tumor tissue at 3 d after the end of the treatment (**100×**).**

(A)131I-EGFR-BSA-PCL group; (B) 131I-BSA-PCL group; (C) 131I group; **(D) normal saline group. (A to D) No significant morphological damage occurred in liver and spleen. (A to D) Tumor necrosis was found in all the** 131I-EGFR-BSA-PCL, 131I-EGFR-BSA-PCL, 131I**,** and **normal saline groups.**