**Supporting Tables S1-S3, and Figures S1-S7.**

**Table S1.** Zeta potentials and hydrodynamic diameters of AuNPs, DNA-Au NPs and nanocomplexes.

| Sample | Zeta-potential (mV) | Hydrodynamic diameter (nm) |
|---|---|---|
| Pure AuNPs | –14 ± 2.2 | 21 ± 1.1 |
| Non-targeted DNA-Au NPs | –30 ± 2.0 | 51 ± 1.6 |
| Non-targeted Nanocomplexes | –23 ± 1.6 | 49 ± 1.8 |
| Targeted DNA-Au NPs | –28 ± 2.7 | 53 ± 2.9 |
| Targeted Nanocomplexes | –21 ± 2.1 | 52 ± 2.7 |
| Targeted ctrl DNA-Au NPs | –27 ± 2.3 | 53 ± 2.6 |
| Targeted ctrl Nanocomplexes | –22 ± 1.9 | 51 ± 2.9 |

**Table S2.** Determination of the average RNase A, DNA and ctEGF loading number per AuNP in the synthesis of DNA-Au NPs, nanocomplexes and NC-Ls.

| Sample | Initial mole ratio NP:RNase:DNA | DNA sequence | The number of RNase A per NP | The number of DNA strands per NP |
|---|---|---|---|---|
| DNA-Au NP | 1:300 | Anti-MDR1 | 0 | 106 ± 5 |
| Nanocomplex | 1:60:300 | Anti-MDR1 | 13.9 ± 0.7 | 85 ± 4 |
| NC-L | 1:60:60 | Anti-MDR1 | 14.1 ± 1.1 | 45 ± 6 |
| Control DNA-Au NP | 1:300 | Control DNA | 0 | 110 ± 3 |
| Control Nanocomplex | 1:60:300 | Control DNA | 14.1 ± 0.8 | 81 ± 3 |

For EGFR-targeted DNA-Au and nanocomplex, the loading number of ctEGF per AuNP was calculated to be 6 ± 1.

**Table S3.** $IC_{50}$ values of OVCAR8 and OVCAR8/ADR cells after sequential treatment of nanocomplexes and doxorubicin.

| Cell types | Treatments | $IC_{50}$ (μM) | |
|---|---|---|---|
| | | **Doxorubicin (DOX)** | **Hydroxyurea** |
| OVCAR8 | Control | 0.26 ± 0.02 | 130 ± 6.1 |
| | Targeted nanocomplex (0.12 nM) | 0.24 ± 0.03 | 134 ± 4.7 |
| OVCAR8/ADR | Control | 88.9 ± 3.3 | 143 ± 5.8 |
| | Non-targeted nanocomplex (0.12 nM) | 85.5 ± 4.8[a] | ------ |
| | Targeted ctrl nanocomplex (0.12 nM) | 89.3 ± 5.2[a] | ------ |
| | Targeted nanocomplex (0.01 nM) | 32.3 ± 1.3[b] | ------ |
| | Targeted nanocomplex (0.04 nM) | 15.6 ± 0.3 | ------ |
| | Targeted nanocomplex (0.12 nM) | 3.9 ± 0.2 (23)[c] | 148 ± 7.2[a] |

[a] non-significance. $P > 0.05$ versus control.

[b] $P < 0.01$ versus control.

[c] Number in the parenthesis represents fold-reversal.

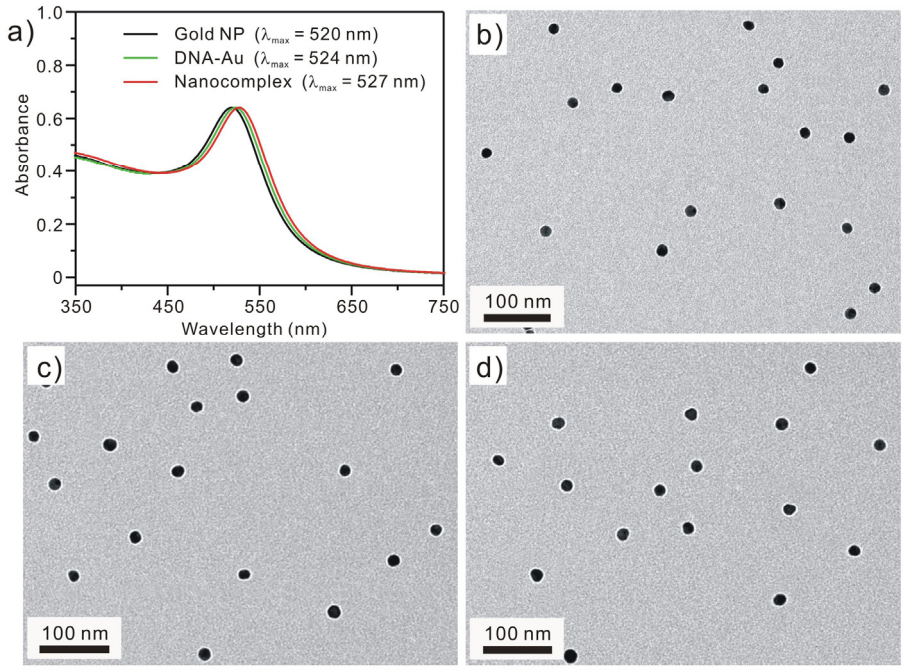



**Figure S1.** a) UV-Vis spectra of unmodified gold nanoparticles (black), DNA-Au NPs (green) and nanocomplexes (red). TEM images of b) unmodified gold nanoparticles, c) DNA-Au NPs, and d) nanocomplexes.

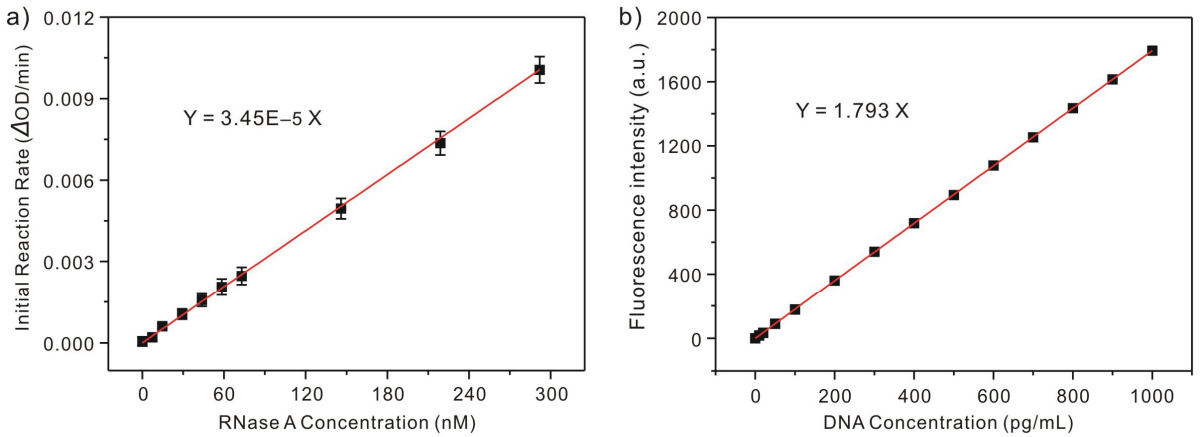



**Figure S2.** a) Plot of initial reaction rate as a function of RNase A concentration for hydrolysis of cytidine 2',3'-phosphate. The substrate concentration was 0.1 mg/mL. b) Plot of fluorescence intensity as a function of DNA concentration.

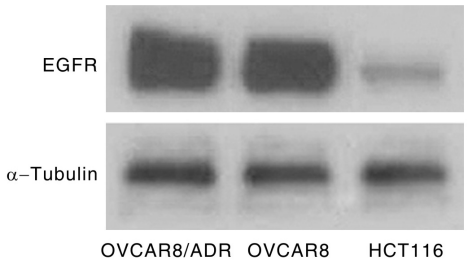



**Figure S3.** Western blot analysis of EGFR expression levels in OVCAR8/ADR cells, OVCAR8 cells and HCT116 cells.

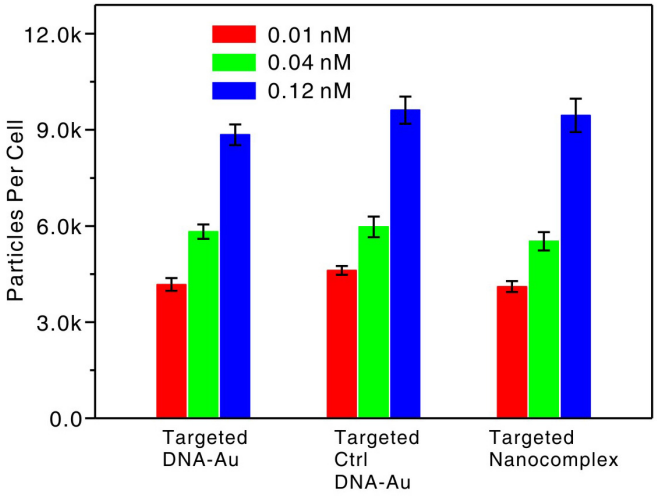



**Figure S4.** Cellular uptake of EGFR-targeted nanoparticles (DNA-Au, Ctrl DNA-Au and Ctrl nanocomplex) after 5 h incubation at 37 $^{o}$C, which are similar with that of targeted nanocomplex.

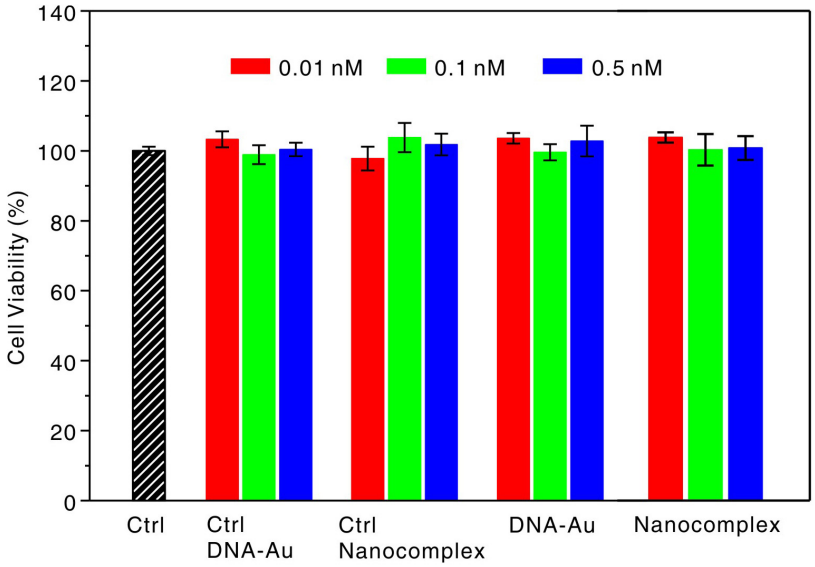



**Figure S5**. Viability of OVCAR8/ADR cells treated with targeted nanoparticles (ctrl DNA-Au, ctrl nanocomplexes, DNA-Au and anti-MDR1 nanocomplexes) at concentrations of 0.01, 0.1 and 0.5 nM after 48 h incubation at 37 $^{o}$C.

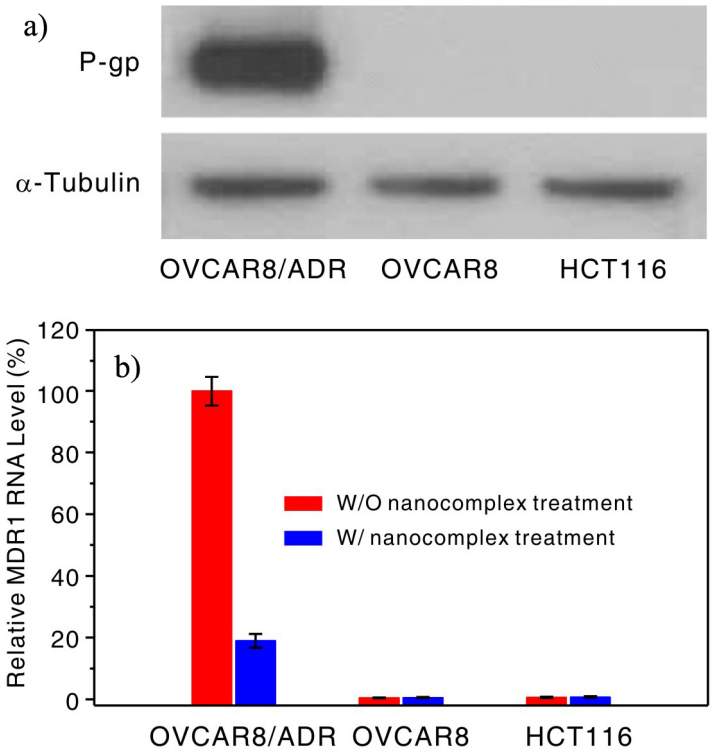



**Figure S6.** a) Western blot analyses of Pgp expression levels in the OVCAR8/ADR cells, OVCAR8 cells and HCT116 cells. b) qRT-PCR analyses of MDR1 mRNA level in the OVCAR8/ADR cells, OVCAR8 cells and HCT116 cells (Red), as well as those after treatment with targeted anti-MDR1 nanocomplexes at a concentration of 0.12 nM (Blue).

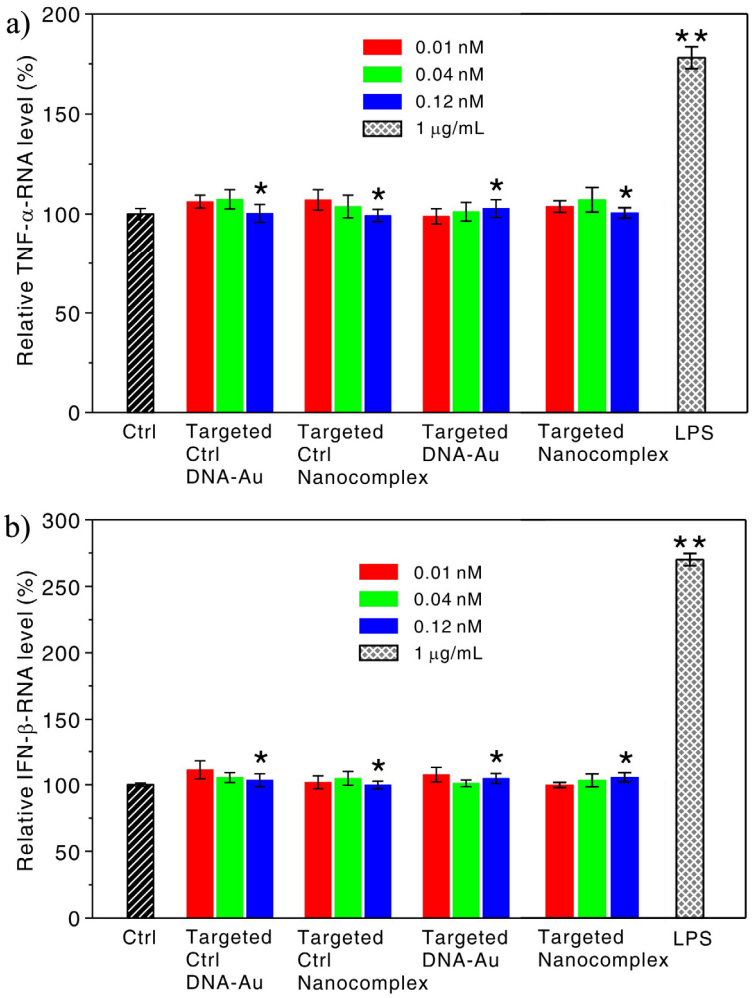



**Figure S7.** TNF-α mRNA (a) and IFN-β mRNA (b) levels in the OVCAR8/ADR cells treated with the targeted nanoparticles (ctrl DNA-Au, ctrl nanocomplex, DNA-Au and anti-MDR1 nanocomplex at concentrations of 0.01, 0.04 and 0.12 nM, and LPS at a concentration of 1 μg/mL. TNF-α and IFN-β mRNA levels were relative to that of human GAPDH. Each bar presents the mean and standard deviation derived from three independent experiments; Student's t test, * for non-significance, $P > 0.41$; ** for $P < 0.0001$.

## Experimental sections:

### 1. Chemicals.

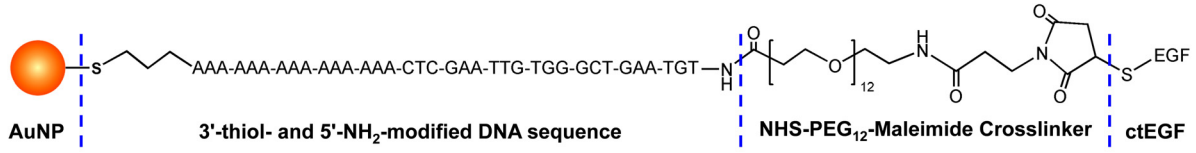
RNase A (ribonuclease A from bovine pancreas), RNase H (ribonuclease H from Escherichia coli recombinant), RNase-free buffer, and chemicals were ordered from Sigma-Aldrich. Anti-MDR1 and control ssDNA oligonucleotides were purchased from IDT. [C4]-Monothiol Cys-tagged EGF (ctEGF) was purchased from Sibtech, Inc (Complete amino acid sequence of ctEGF: Met Lys Glu Ser **Cys** Ala Lys Lys Phe Gln Arg Gln His Met Asp Ser Gly Gly Gly Gly Ser Gly Gly Gly Gly Ser Gly Gly Gly Gly Ser Met GlyAsn Ser Asp Ser Glu

Cys Pro Leu Ser His Asp Gly Tyr Cys Leu His Asp Gly Val Cys Met Tyr Ile Glu Ala Leu Asp Lys Tyr Ala Cys Asn Cys Val Val Gly Tyr Ile Gly Glu Arg Cys Gln Tyr Arg Asp Leu Lys Trp Trp Glu Leu Arg).

**The DNA sequences are the following list:**

| Names of ssDNA | Sequence |
|---|---|
| Anti-MDR1 DNA | 5'-TGTAAGTCGGGTGTTAAGCTC-$A_{15}$-alkanethiol-3' |
| Amino-modified Anti-MDR1 DNA for conjugation to ctEGF | 5'-$NH_2$-TGTAAGTCGGGTGTTAA-GCTC-$A_{15}$-alkanethiol-3' |
| Control DNA | 5'-TG***A***A***C***GTC***AC***GT***CA***TAAG***G***TC-$A_{15}$-alkanethiol-3' |
| Amino-modified control DNA for conjugation to ctEGF | 5'-$NH_2$-TG***A***A***C***GTC***AC***GT***CA***TAA-G***G***TC-$A_{15}$-alkanethiol-3' |

**2. Synthesis of ctEGF-modified DNA oligonucleotides (ctEGF-DNA).** ctEGF was conjugated with amino-modified DNA oligonucleotides using NHS-PEG-Maleimide crosslinker according to the manufacturer's protocol. In a typical experiment, DNA oligonucleotides (1 nmol) were dissolved in 100 uL PBS buffer, and then a NHS-$(PEG)_{12}$-Maleimide solution (10 nmol) was added to the above solution. After incubation for 30 min at room temperature, the solution was desalted to remove excess crosslinkers using a desalting column. Then, ctEGF (1.2 nmol) was mixed with activated DNA oligonucleotides solution, and incubated for 30 min at room temperature. After the reaction, the conjugates were purified with HPLC and analyzed by mass spectrometry.



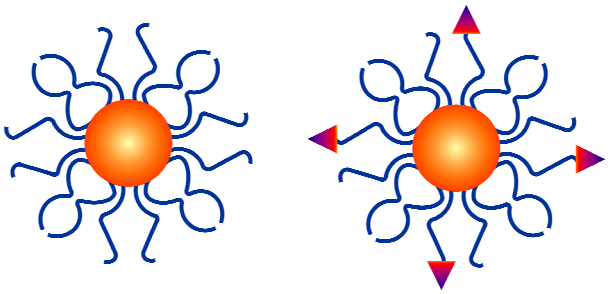

**Scheme S1.** Schematic representation describing the structures of 5'-$NH_2$ modification of DNA oligonucleotide with ctEGF and its 3'-SH conjugation with AuNPs.

**3. Synthesis of Gold Nanoparticles, DNA-Au NPs, and Nanocomplexes.**

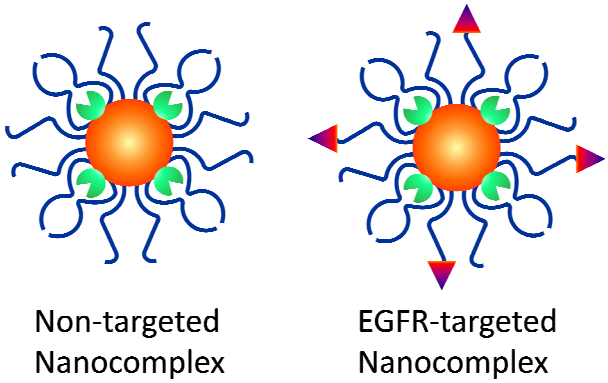
**3.1. Gold nanoparticles (AuNPs) synthesis.** Citrate-stabilized gold nanoparticles (15 ± 0.7 nm, Figure S1A) were prepared according to literature procedures.[S1, S2]

**3.2 Synthesis of DNA-functionalized gold nanoparticles (DNA-Au NPs).** Gold nanoparticles (3 nM, 15 ± 0.7 nm in diameter) were mixed with alkanethiol-modified oligonucleotides (1.8 nmol) and phosphate buffer (1.0 M, pH 7.4) was added to bring the mixture solution with 10 mM phosphate. After 8 h, a sodium chloride solution (1.5 M solution in RNase-free water) was added to bring the NaCl concentration gradually to 0.3 M

during a period of 32 h. The solution was further shaken for another 8 h. Then the resulting DNA-Au NPs were purified using centrifugation (11000 rpm, 10 min, for three times), and were redispersed in PBS buffer for use. PBS buffer components used in this paper: 10 mM phosphate, pH 7.4, 138 mM NaCl, 27 mM KCl and 5 mM $MgCl_2$. For the EGFR-targeted DNA-Au NPs, the mixture of DNA and ctEGF-DNA oligonucleotides at a molar ratio of 15:1 was added to Au NPs solution. DNA loading number per AuNP was determined in Table S1.




**Scheme S2.** Schematic representation describing the structures of non-targeted and EGFR-targeted DNA-Au NPs.




**Scheme S3.** Schematic representation describing the design of non-targeted and EGFR-targeted nanocomplexes.

**3.3. Nanocomplex synthesis.** In a typical synthesis, Au NPs (3 nM, 15 ± 0.7 nm in diameter) were mixed with RNase A (150 nM) in a carbonate buffered solution (2 mL; carbonate, 10 mM; pH 9.6).[S3] Under shaking for 30 min, alkanethiol-modified DNA oligonucleotides and phosphate buffer (1.0 M, pH 7.4) were added to bring the mixture solution with 10 mM phosphate. After 8 h shaking, a sodium chloride solution (1.5 M solution in RNase-free water) was added to bring the NaCl concentration gradually to 0.3 M during a period of 32 h. The solution was further shaken for another 8 h. Then the resulting nanocomplexes were purified using centrifugation (11000 rpm, 10 min, for three times), and were re-dispersed in PBS buffer for use. For the EGFR-targeted nanocomplexes, the mixture

of DNA and ctEGF-DNA oligonucleotides at a molar ratio of 12:1 was added to RNase-Au conjugates solution. Note that all the vials and tubes used herein were modified by silane for minimizing the nonspecific binding of RNase A onto the surface of these glass containers. The loading number of DNA and RNase A per AuNP was determined in Table S1.

## 4. Determination of the Number of RNase A, Oligonucleotide and ctEGF Loaded onto Gold Nanoparticles.

**4.1. RNase A Loading Determination**. The average number of RNase molecules loaded onto a single nanocomplex was determined by a subtraction method. The total amount of RNase molecules loaded onto AuNPs in a synthesis batch was determined by subtracting the amount of unloaded RNase molecules from the amount of RNase molecules added initially. This total loading amount was then divided by the total number of nanocomplexes in the solution, yielding the average number of RNase A per single nanocomplex. The number of nanocomplexes was determined by using UV-Vis absorption spectroscopy ($\lambda$ = 524 nm, $\varepsilon$ = $3.64 \times 10^8$ $M^{-1}cm^{-1}$). The amount of unloaded RNase in a reaction solution was determined by measuring the RNase activity of the supernatant after removal of nanocomplexes. A typical RNase activity measurement was performed according to the literature method, in which cytidine-2':3'-phosphate was used as the substrate.[S4] Then, the amount of RNase molecules was obtained using a standard RNase activity curve (see Figure S2). Note that all the vials and tubes used in this experiment were modified by silane for minimizing the nonspecific binding of RNase A onto the surface of these glass containers.

**4.2. DNA Loading Determination.** The number of nanocomplexes or DNA-Au NPs were determined by using UV-Vis spectroscopy ($\lambda$ = 524 nm, $\varepsilon$ = $3.64 \times 10^8$ $M^{-1}cm^{-1}$).[S5] DNA oligonucleotides were released from nanocomplexes or DNA-Au NPs by dissolving their AuNPs backbones in 0.1 M KCN solution. The number of DNA molecules per nanoparticle was determined using an oligonucleotide quantification kit (Oligreen; Invitrogen) following the manufacturers' recommendations. The DNA loading number was calculated by dividing the concentration of oligonucleotides by the concentration of gold nanoparticles (Figure S3).

**4.3. ctEGF loading determination.** The number of targeted nanocomplexes or DNA-Au NPs were determined by using UV-Vis spectroscopy ($\lambda$ = 524 nm, $\varepsilon$ = $3.64 \times 10^8$ $M^{-1}cm^{-1}$). ctEGF ligands were released from nanocomplexes or DNA-Au NPs by dissolving their AuNPs backbones in 0.1 M KCN solution. The number of ctEGF ligands per nanoparticle was determined *via* SDS-PAGE by comparing the band intensity of each sample to a standard curve using Image J Image.[S6] The average ctEGF loading number was calculated by dividing the concentration of ctEGF ligands by the concentration of Au NPs.

**5. Characterization of Nanocomplexes using TEM, UV Spectra and DLS.** Transmission electron microscopy (TEM) measurements were performed on a JEOL 200X operated at 200 kV. UV-Vis absorption spectra were measured using a Shimadzu UV1701. Dynamic Light Scattering (DLS) analysis was carried out using a Zetasizer Nano ZS (Malvern Instruments Ltd, UK).

**6. Determination of Nanocomplex Activity.**

**6.1. Synthesis of RNA substrates using *in vitro* transcription.** The complete cDNA segment coding P-glycoprotein/MDR1 gene was obtained from vector pGEM3Zf(-)Xba-MDR1.1 purchased from ATCC. MDR1 DNA fragment in length of 970 bp and firefly luciferase DNA (Fluc DNA) in length of 1653 bp were cloned into pcDNA 3.1(-) vector (Invitrogen) with standard molecular cloning method. The cloning primers for MDR1 are: 5'-GTCAGCTAGCCTGTTCGTTTCCTTTAGG-3' (Forward); 5'-GCCCGGATC-CAGCTTTCTTTATCCC-3' (Reverse). The primers for Fluc mRNA: 5'-GACCGCTAG-CGCCACCATGGAAGACGCCAAAAAC-3' (Forward), 5'-GGCGCGGATCCTTACA-CGGCGATCTTTCC-3' (Reverse). Endonuclease restriction cutting sites Nhe1 and BamH1 were incorporated in the forward and reverse primers, respectively. The expression vectors were sequenced correctly. Both vectors were digested by BamH1. The resulting linearized DNA plasmids were purified and used as the templates for *in vitro* transcription to make the MDR1 mRNA segment (nucleotides 400–1369) or the Fluc mRNA (nucleotides 1-1653) using the MEGAscript® Kit (Invitrogen) following the manufacture's protocol.

To evaluate the target selectivity of the anti-MDR1 nanocomplex, 970-nt MDR1 mRNA fragment was used as the target substrate. 1,653-nt Fluc mRNA was served as the control substrate, which does not contain complementary sequences to the nanocomplex-bearing anti-MDR1 DNA oligonucleotides. Moreover, RNase H showed no cleavage activity towards the mixture of Fluc mRNA and the anti-MDR1 DNA oligonucleotide (Figure 1 in the main text), which experimentally confirmed that Fluc mRNA does not have complementary sequences to the anti-MDR1 DNA oligonucleotide.

**6.2. RNase A activity assay.** In a typical test, mRNA substrates (1 μg) were incubated with nanocomplexes, DNA-Au NPs, control nanocomplexes, control DNA-Au NPs or NZ-Ls at a concentration of 0.02 nM or unbound RNase A (0.28 nM) in a PBS solution (10 μL) for 15 min. Then 10 μL of the formaldehyde loading buffer (Londa Rockland, Inc.) was added to denature the RNA products, and the resulting solution was heated at 65 °C for 12 min, and then immediately placed on ice for 2 min before loading onto a 2% agarose/formaldehyde denaturing gel (10× MOPS buffer, 5 mL: RNase free water, 45 mL; agarose, molecular

biology grade, 1.0 g; and 37% formaldehyde solution, 1 mL). Gel electrophoresis was performed at 60 V for approximately 90 min or until the front line of bromophenol blue dyes migrated about 6 cm in the gel. Afterwards, the gel was stained by SYBR Green II for visualization.

**6.3. Proteinase K resistance tests.** In a typical proteinase K resistance test, nanocomplexes (0.02 nM), NZ-Ls (0.02 nM) or unbound RNase A (0.28 nM) were first incubated with proteinase K (10 nM) in a PBS solution at 37 $^{o}$C for 1 h. Then the product of this proteinase K treatment was divided into two parts and further incubated with the MDR1 (or Fluc) mRNA (1 μg) in a PBS buffer (10 μL) at 37 $^{o}$C for 15 min. The products were analyzed by using electrophoresis in a 2% formaldehyde agarose gel as described above.

**7. Evaluation of Cellular Uptake of Non-targeted and EGFR-Targeted Nanocomplexes.** To evaluate the specific delivery of ctEGF-modified nanocomplex, we chose three cell lines including EGFR-negative HCT116 cells, EGFR-positive OVCAR8 and OVCAR8/ADR cells. In a typical experiment, targeted or non-targeted nanoparticles (anti-MDR1 nanocomplexes, DNA-Au NPs, ctrl nanocomplexes and ctrl DNA-Au NPs) at concentrations of 0.01, 0.04, and 0.12 nM in RPMI 1640 media were added into the cells (70% confluence) cultured in 6-well plates, respectively. After incubation for 5 h, culture media were removed, and the treated cells were washed with 1× PBS three times. Afterwards, the cells were detached from the 6-well plates using trypsin (0.3 mL, 0.25%) and transferred into glass vials. The number of cells in each well was counted using a hemocytometer. Then, the cells were lysed under sonication (37 $^{o}$C) for 1 hour, and clear solutions were obtained. Au nanoparticles in these solutions were dissolved using freshly made aqua regia (0.3 mL), and resulting solutions were then diluted by 50-fold using a solution containing 2% $HNO_3$, 1% HCl, and 8 ppb Rhenium (internal standard), and the concentrations of gold ions in these solutions were determined using inductively coupled plasma mass spectrometer (ICP-MS, Element 2 ICP-MS, Thermo Scientific, USA). The concentration of Au nanoparticles in each well was calculated using the Au ion concentration and the size of gold nanoparticles determined using TEM. Finally, the number of gold nanoparticles per cell in each well was calculated using the number of cells in the corresponding well.[S7, S8]

**8. Cell Proliferation Assay.**

**8.1. Cytotoxicity of targeted nanocomplexes on OVCAR8/ADR cells.** OVCAR8/ADR cells were dispensed into 96-well plates at a final concentration of $1 \times 10^4$ cells/well in a culture medium (100 μL), and incubated overnight before treatment. The culture medium was

then removed and replaced with new medium containing the targeted anti-MDR1 nanocomplexes at varying concentrations of 0.01, 0.1 and 0.5 nM. After 48 h incubation, cell viability was measured using the MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide) assay (Figure S5).

**8.2. Effect of targeted nanocomplexes on drug sensitivity of OVCAR8/ADR cells.** OVCAR8 and OVCAR8/ADR cells were seeded into 96-well plates at a final concentration of $1 \times 10^4$ cells/well in a culture medium (100 μL), and incubated overnight before treatment. The culture medium was then removed and replaced with new medium containing the targeted anti-MDR1 nanocomplexes at different concentrations of 0.01, 0.04 and 0.12 nM. After 5 h incubation, the solution was replaced with fresh medium containing incremental doxorubicin concentration, and the cells were allowed to grow for another 48 h. After that, cell viability was measured using the MTT assay.

**9. Anti-MDR1 Effects of Nanocomplexes in OVCAR8 and OVCAR8/ADR Cells.** OVCAR8 and OVCAR8/ADR cells were grown in RPMI 1640 media supplemented with 10% fetal bovine serum and antibiotics at 37 ºC in 5% $CO_2$. Cells were seeded onto 35-mm wells of a 6-well plate and cultured overnight. The cells were treated with fresh media containing targeted anti-MDR1 nanocomplexes or targeted controls (DNA-Au NPs, control nanocomplexes, control DNA-Au NPs) using different concentrations of 0.01, 0.04 and 0.12 nM or non-targeted anti-MDR1 nanocomplexes (0.12 nM) for 5 h, followed by incubation with fresh growth medium for another 48 h. Control cells were incubated only with the culture medium. After that, cells were lysed and assayed by quantitative real-time PCR analyses (qRT-PCR) and western blot.

**10. RNA Extraction and qRT-PCR Analysis for the RNA Levels of MDR1, TNF-α, IFN-β and GAPDH**. RNA samples were extracted from OVCAR8, OVCAR8/ADR and HCT116 cells using a TriPure isolation reagent (Roche). To prevent DNA contamination, total RNA was treated with RNase-free DNase II (Invitrogen). Total RNA samples (1 μg per reaction) were reversely transcribed into cDNAs by SuperScript™ First-Strand Synthesis System (Invitrogen). Then, the cDNAs were used as templates in quantitative real-time PCR. The amplification reactions were performed using Fast SYBR Green Master Mix on a StepOne Plus real-time PCR system (Applied Biosystems, Foster City, CA) according to the manufacturer's protocol. The Primers for MDR1 gene were 5'-AAATTGGCTTGACAAGTT-GTATATGG-3' (Forward) and 5'-CACCAGCATCATGAGAGGAAGTC-3' (Reverse). The primers for TNF-α gene were 5'-CAAGCTTAACCGTTTACTCGCTGCTGT-3' (Forward)

and 5'-TGCGGCCGCTGCTGGCTACTCCTCATCCT-3' (Reverse). The primers for TNF-α gene were 5'-CGAGTCTGGGCAGGTCTA-3' (Forward) and 5'-CGAAGTGGTGGTCTTG-TTG-3' (Reverse). The primers for IFN-β gene were 5'-AAGAGTTACACTGCCTTTG-CCATC-3' (Forward) and 5'-CACTGTCTGCTGGTGGAGTTCATC-3' (Reverse). The human glyceraldehyde-3-phosphate dehydrogenase (GAPDH) was used as an internal control in PCR amplification, and its primers were 5'-GGTCTCCTCTGACTTCAACA-3' (Forward) and 5'-AGCCAAATTCGTTGTCATAC-3' (Reverse).

**11. Western Blot Analysis of Pgp and EGFR proteins.** Cell lysates were prepared by treating samples with ice-cold RIPA buffer (PIERCE) with a protease inhibitor cocktail (Cell Signaling Technology, Inc.) for 5 min on ice followed by centrifugation at 4 ºC for 15 min to sediment particulate materials. Proteins then were separated by SDS-PAGE, transferred to nitrocellulose membranes. The membranes were incubated in blocking solution containing 5% skim milk for 1 h at room temperature, and then immunoblotted with monoclonal anti-Pgp mouse (C219) antibody (EMD Millipore), EGF receptor rabbit mAb, β-Actin (13E5) rabbit mAb (Cell Signaling Technology, Inc.), or monoclonal anti-α-tubulin mouse antibody (Sigma-Aldrich). Immunoreactive bands were visualized using Supersignal$^{TM}$ West Pico chemiluminescent substrate (PIERCE) according to the manufacturer's directions.

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