## *Erratum* Two engineered site-specific antibody-drug conjugates, HLmD4 and HLvM4, have potent therapeutic activity in two DLL4-positive tumour xenograft models: Am J Cancer Res. 2020; 10(8): 2387-2408

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The labels "Q3 1.62%" and "Q4 96.7%" of the control group in **Figure 3D** were not in the correct position, and their positions were switched.

The two pictures in **Figure 6E** are the same. Because these two pictures are very similar, we mistakenly put the same picture twice.

The correct **Figures 3D** and **6E** are provided as shown below.

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## Engineered site-specific ADCs have potent therapeutic activity



Figure 3. In vitro cytotoxicity and mechanism of anti-DLL4 ADCs. A. The cytotoxicity of ADCs was assessed by MTT assay. The percentage of cell inhibition relative to untreated control HUVEC cells was calculated for each drug concentration. The three ADCs, especially the conjugates with DM1, induced potent anti-proliferative ef-

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fects in HUVEC. The IC50 value of Docetaxel, HLmD4, JmD4, HLvM4 and DM1 was 10.57 nM, 37.45 nM, 36.12 nM, 40.53 nM and 11.72 nM, respectively. B. ADCs for 24 h examined by caspase 3/7 activity. C. Western blotting for  $\alpha$ -tubulin and  $\beta$ -tubulin. ADCs conjugated with DM1 or MMAE inhibit the formation of  $\alpha \& \beta$ -tubulin dimmer. Lane 1: Negative control; Lane 2: Docetaxel; Lane 3: DM1; Lane 4: MMAE; Lane 5: H3L2; Lane 6: HLmD4; Lane 7: HLvM4; and Lane 8: JmD4. D. ADCs induce apoptosis of HUVEC cell. The cells were separately treated with corresponding concentrations of Docetaxel, DM1, H3L2 analyzed by flow cytometry following staining with Annexin V-FITC and PI. E. Quantitative analysis of apoptosis assay. F. Cell cycle analyzes HUVEC cells which were incubated with certain concentrations of drugs for 24 h and stained with PI. The percentage of cells in each phase was indicated. G. Quantitative analysis of cell cycle assay. Data were presented as the mean  $\pm$  SD, n=3, \*P<0.05, \*\*P<0.001, \*\*\*P<0.005. NS: no significance.





**Figure 6.** Engineered anti-DLL4 ADCs show lower toxicity than conventional ADC and small molecule drug in mouse safety studies. A. The plasma stability of ADCs was tested in BALB/c nude mouse (n=6 animals/group, single i.v. dose on day 1). Changing values in blood over time relative to study day 1 were plotted. B, C. ICR mice (n=5 animals/group, single i.v. dose on day 1) were given HLmD4, JmD4, HLvM4, H3L2, DM1 or Docetaxel at the indicated dose levels. Blood was drawn from mice on study days 6 and 12 for clinical chemistry (serum AST levels) and hematology (platelet counts). D, E. MDA-MB-231 or A549 xenograft BALB/c nude mouse models in different groups (n=6) were weighed daily after dosing, and changes in body weight over time relative to study are plotted from day 1 to day 24. F. Survival rates of tumour-bearing mice in different groups (n=6). In the MDA-MB-231 model and the A549 model, 5 mg/kg HLmD4 caused an effect to prolong evidently the length of survival. The arrows indicate dosing days except H3L2 group. Data are presented as the mean ± SD, \*\*P<0.01, \*\*\*P<0.0001. NS: no significance.