

↳ LNCaP cells / non PCA cells

I.V.

- conc. of plasmid
→ find optimal amt for detection
- conc. of cells
→ confirm ↑ cell conc. ⇒ ↑ GFP brightness

D.V.

- brightness of GFP
→ measured by plate reader

Control:

- Set-up without plasmid
 - Set-up without cancer cells
- } -ve control

* Set-up that only contains GFP (no promoter/enhancer)
To ensure GFP can be expressed



↳ LNCaP cells / non PCA cells

- confirm Gluc works the same as GFP.

D.V.

- brightness of Gluc
→ also w/ plate reader

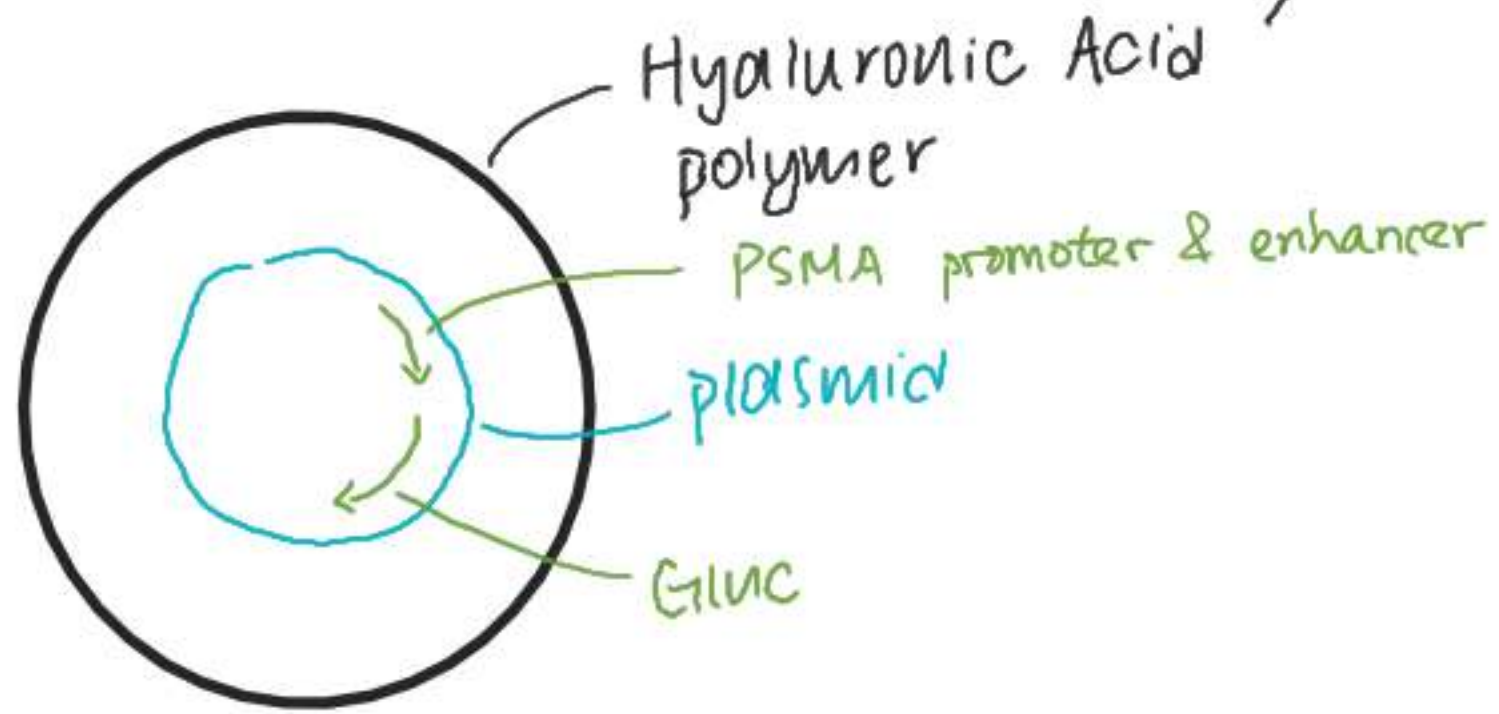
Control

- Set-up without promoter & enhancer
- set-up only Gluc



DETECT

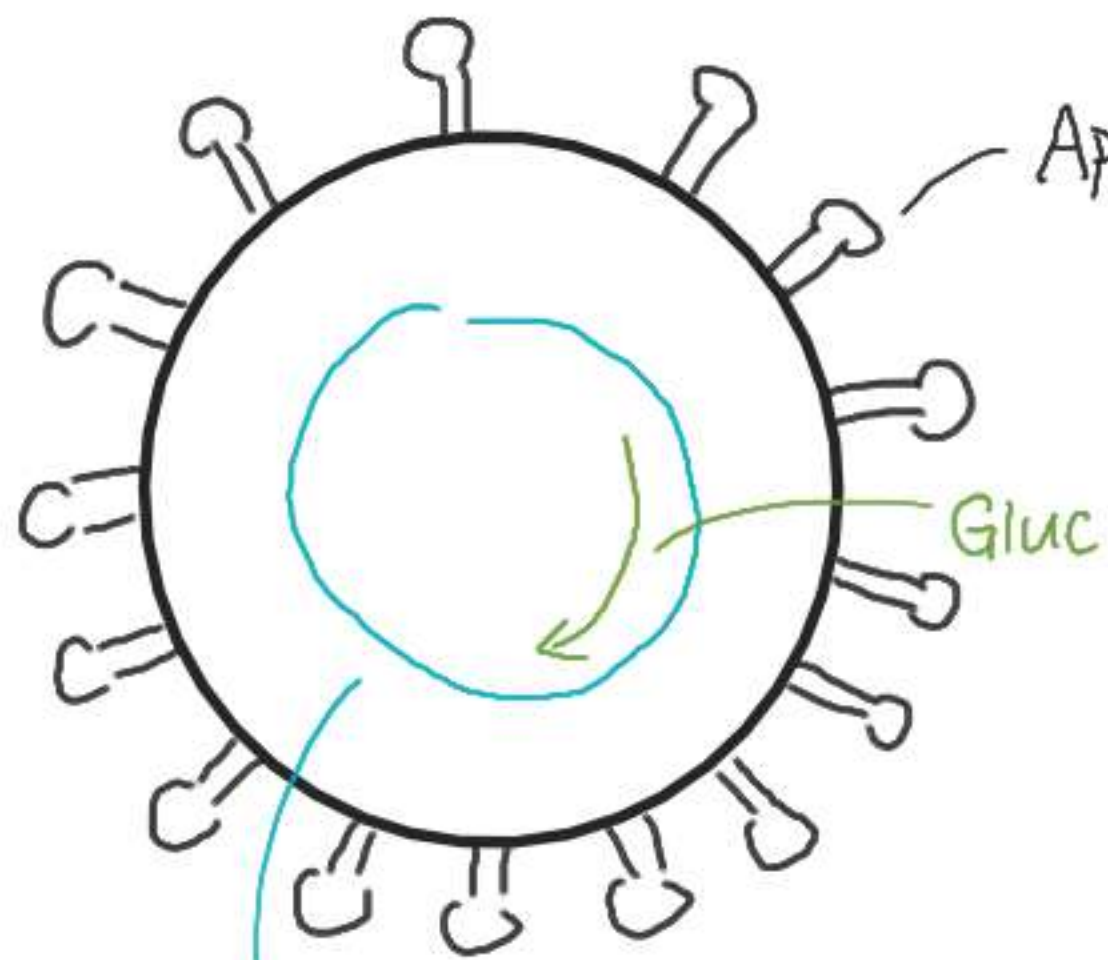
Approach A



natural ability: bind to
CD-44 (protein), overexpressed
at tumours

DETECT

Approach B

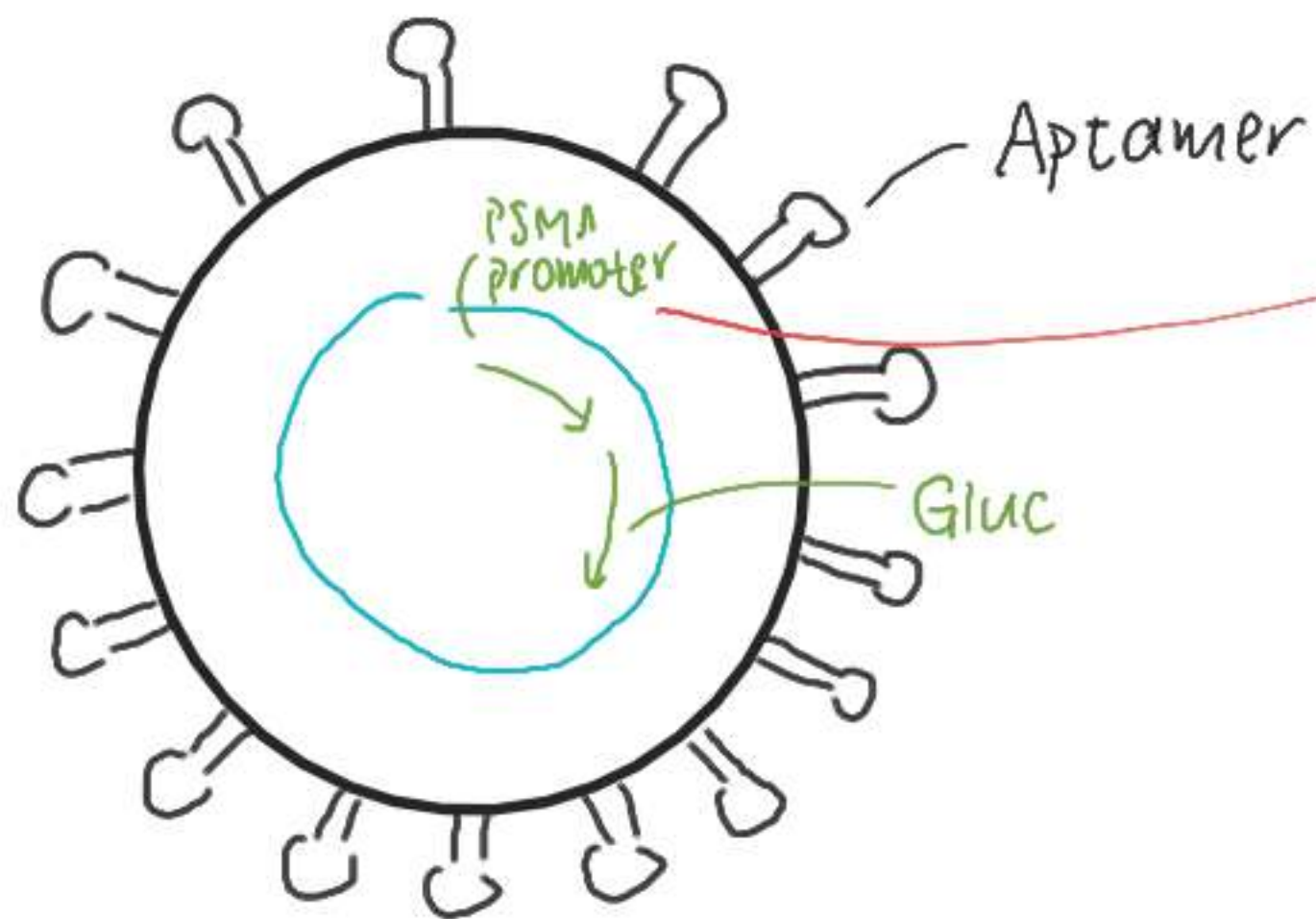


Aptamer: sequence of DNA/RNA that binds to certain proteins
recall: "key" / "lock"

no PSMA promoter → directly expresses Gluc in cell
→ using constitutive promoter

DETECT

Approach C



note that both protein of Aptamer & PSMA must be present for Gluc to be expressed

↳ \approx AND gate: \Rightarrow

↳ \neq specificity

DETECT

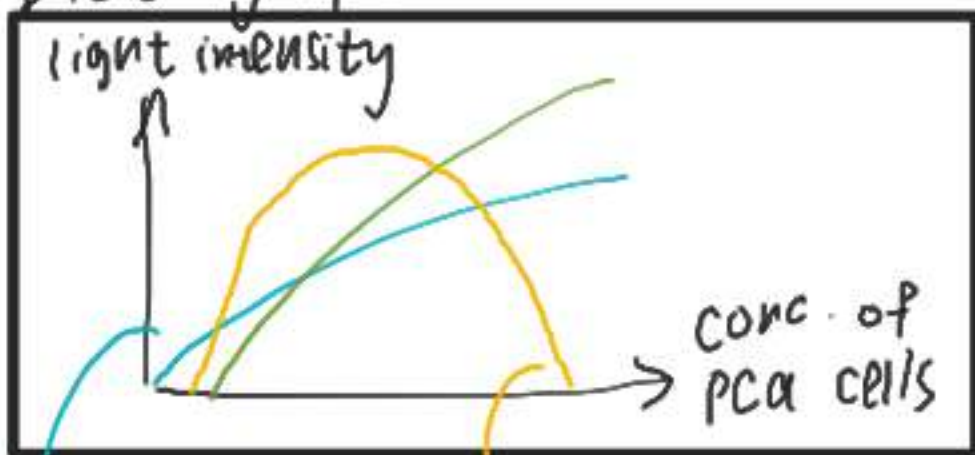
Measurements

I.V. - amt. of cells

- > all PCA cell
- > all non-PCA cell
- > (multiple) PCA & non-PCA mixed together

D.V. - lowest conc. of PCA cells that is detectable

- plot graph

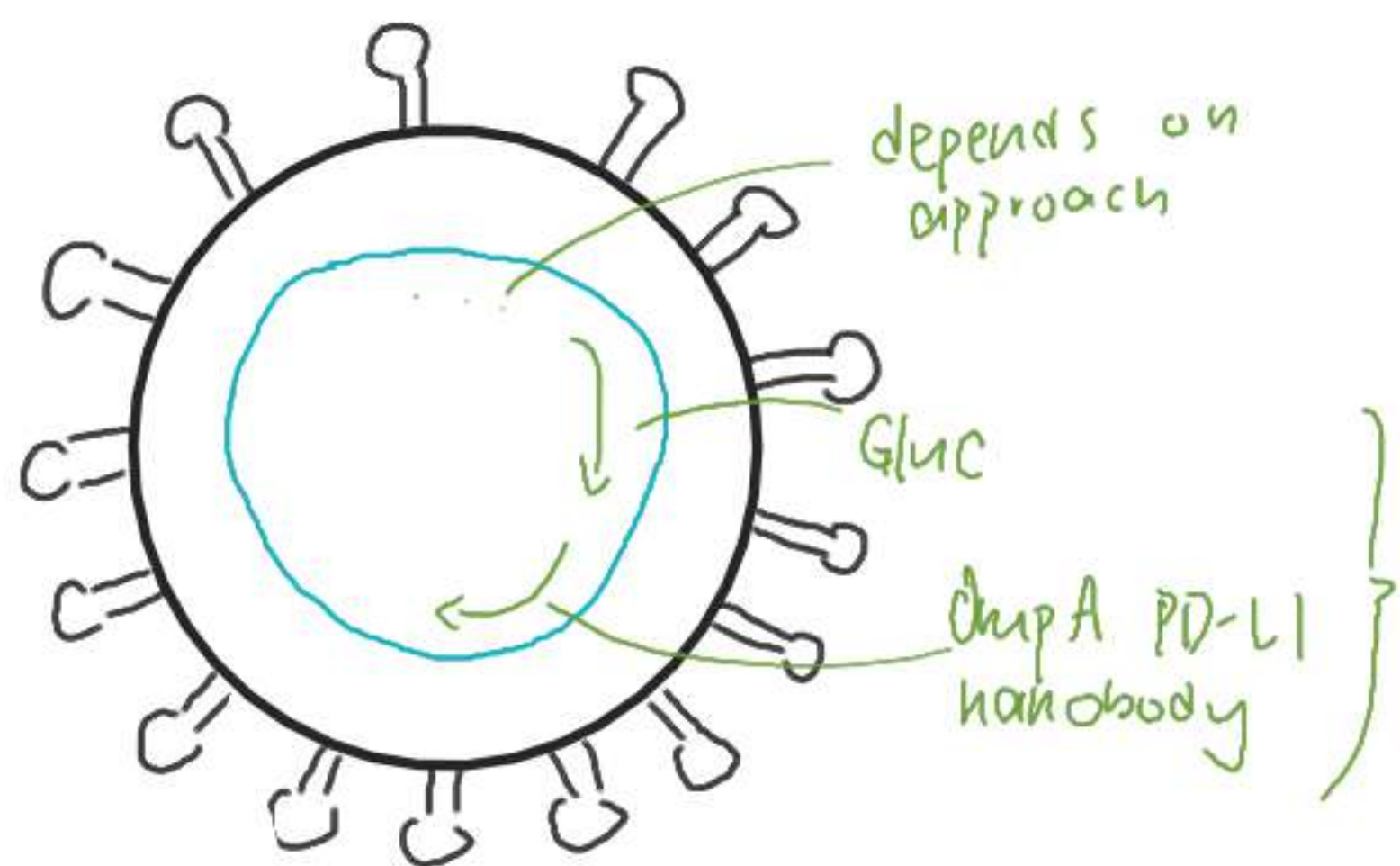


取捨: high sensitivity / larger rate of increase w/ PCA increase

DETECT

	Approach A	Approach B	Approach C
+ve		<ul style="list-style-type: none"> - simplest & easiest implementation - highest sensitivity 	<ul style="list-style-type: none"> - highest specificity
-ve	<ul style="list-style-type: none"> - HA polymer may be activated at other tumours / disease sites → ↓ conc. when it arrives at prostate → ↓ sensitivity 	<ul style="list-style-type: none"> - "走火" 	<ul style="list-style-type: none"> - lower sensitivity than B

DETECT



OmpA... will be expressed
alongside Gluc

→ OmpA brings
PD-L1 nanobody
outside cell

↓
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KILL