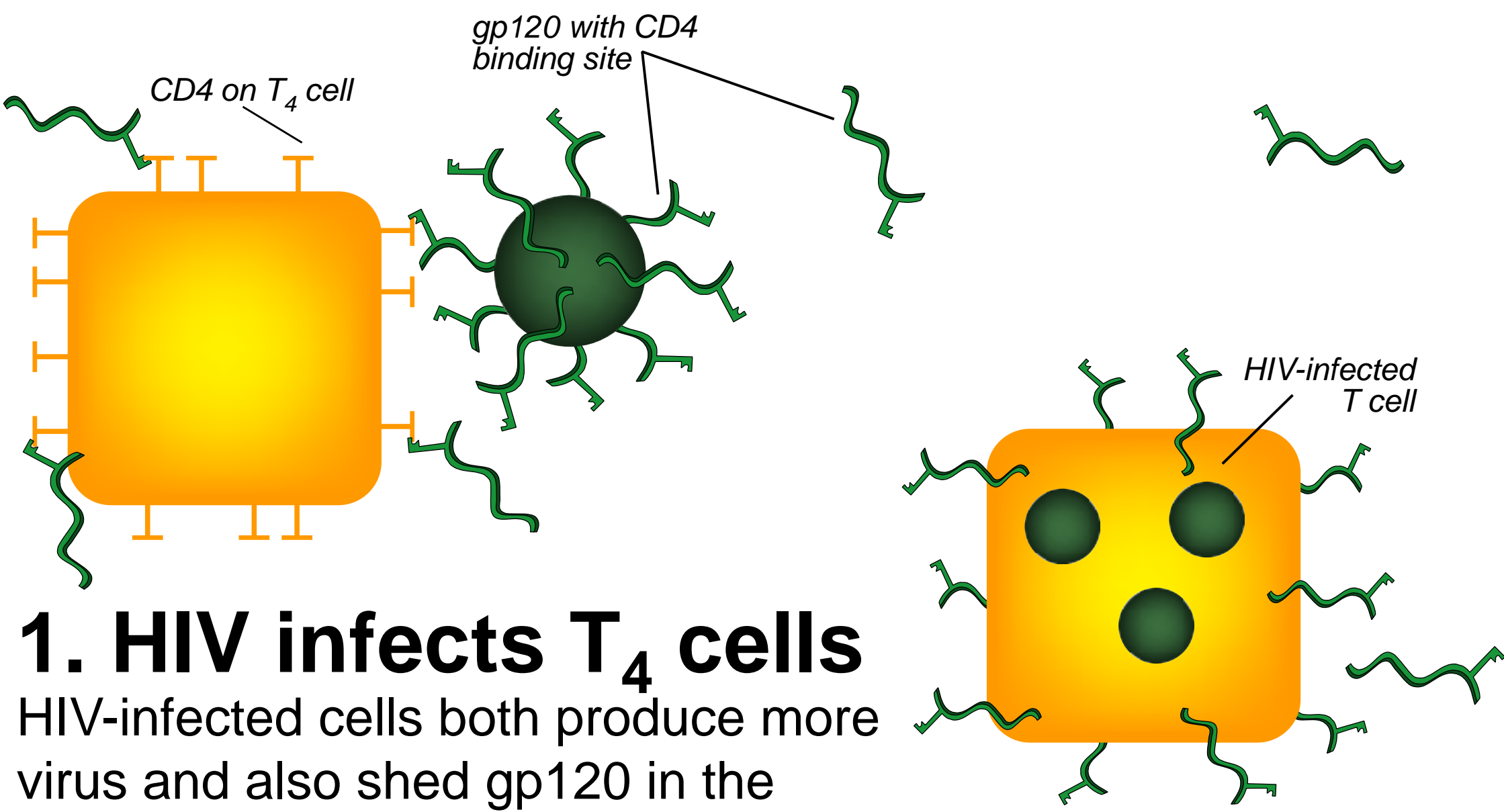
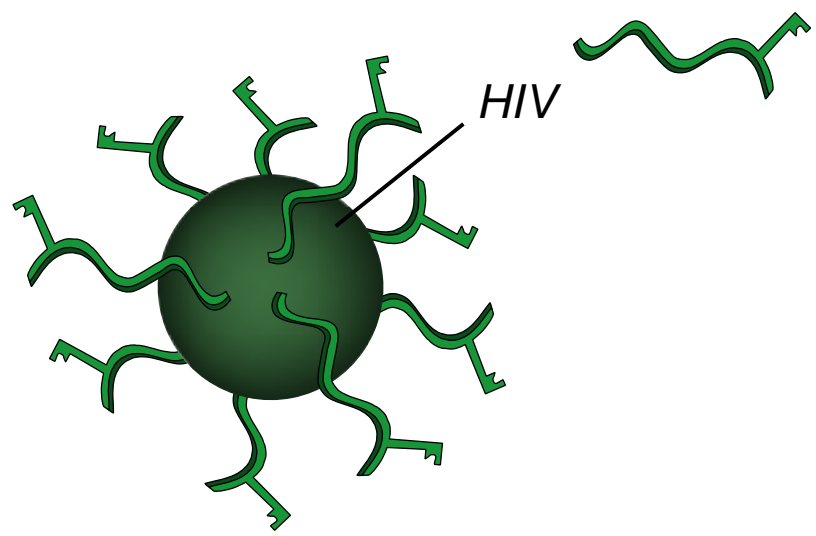


# HIV's Interaction with the Immune System



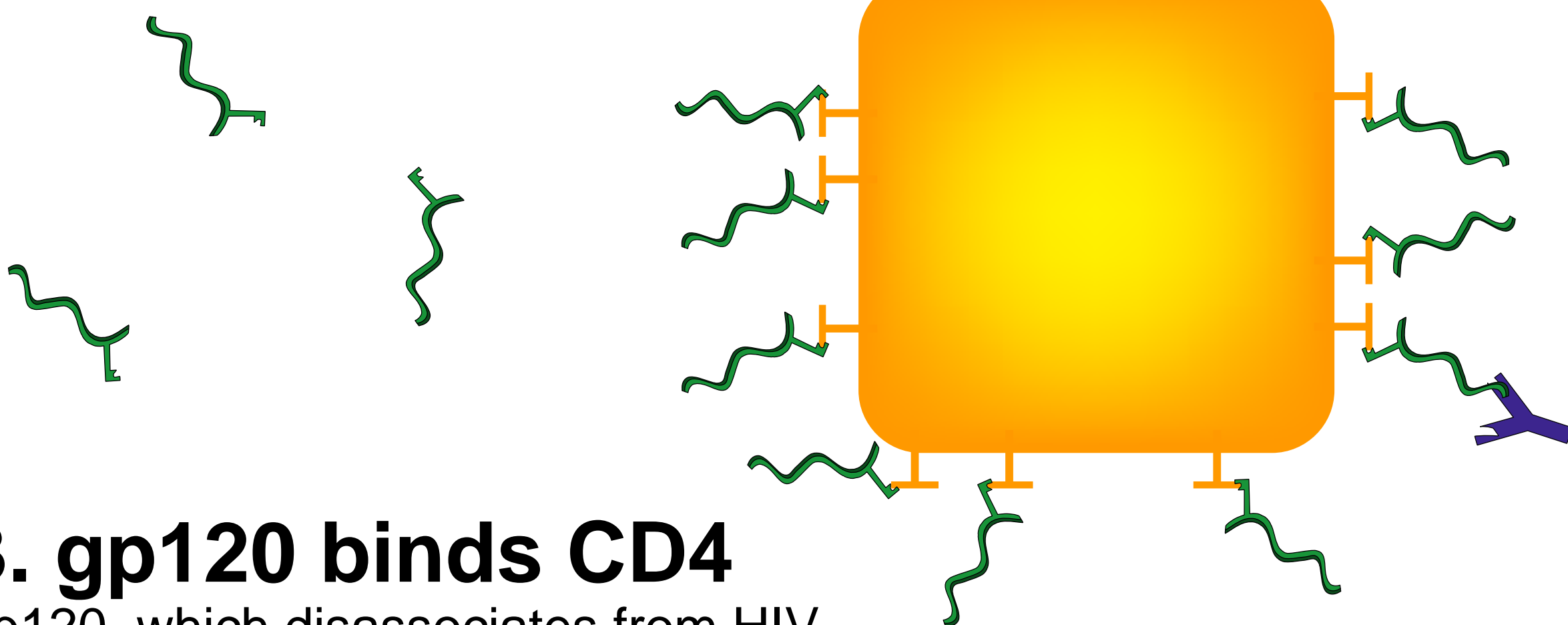
## 1. HIV infects T<sub>4</sub> cells

HIV-infected cells both produce more virus and also shed gp120 in the bloodstream.



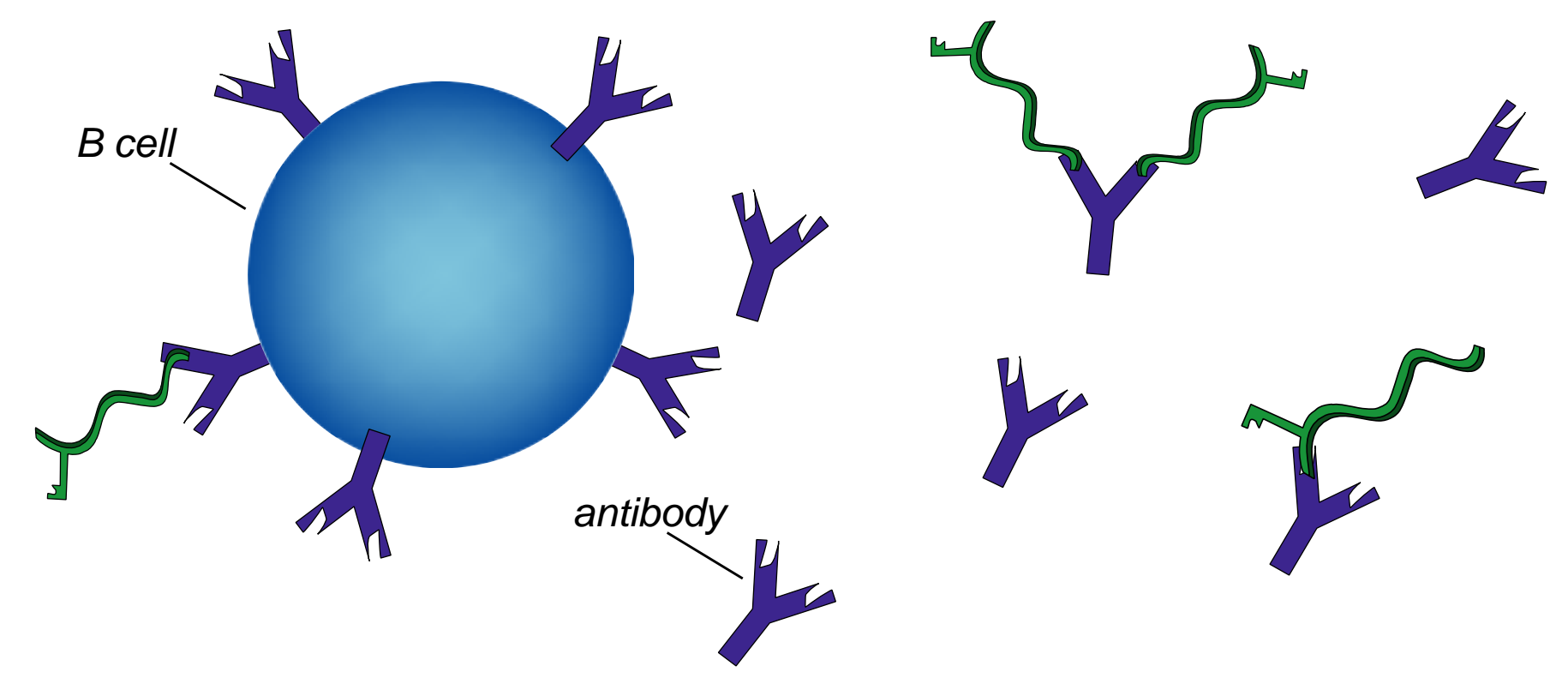
## 2. gp120 is everywhere

After an infection has been established for two years, free gp120 is present at 40 nM concentration, making it one of the most common proteins in the bloodstream.



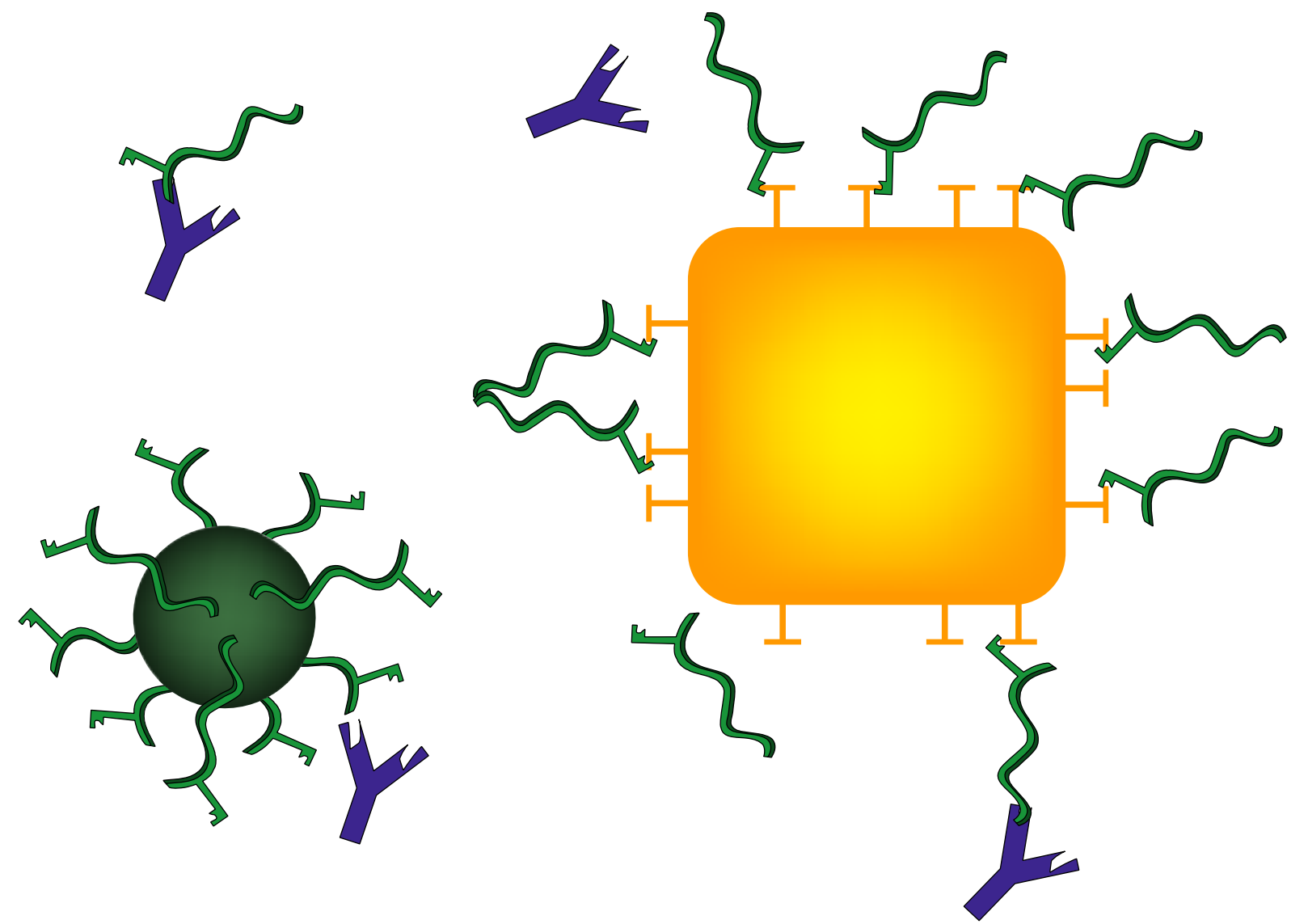
## 3. gp120 binds CD4

gp120, which disassociates from HIV easily, binds to CD4 independent of attachment to the virus. Most T<sub>4</sub> cells have 80% of their CD4 receptors bound by gp120, which causes mild anergy.



## 4. B cells react to gp120

When activated, gp120-reactive B cells produce antibodies against the viral coat protein. Activated B cells produce 200 antibodies per second and divide every 90 minutes, but the antibody response in a new HIV infection can be delayed up to six months by gp120 binding to and being internalized by CD4 receptors on T<sub>4</sub> cells.

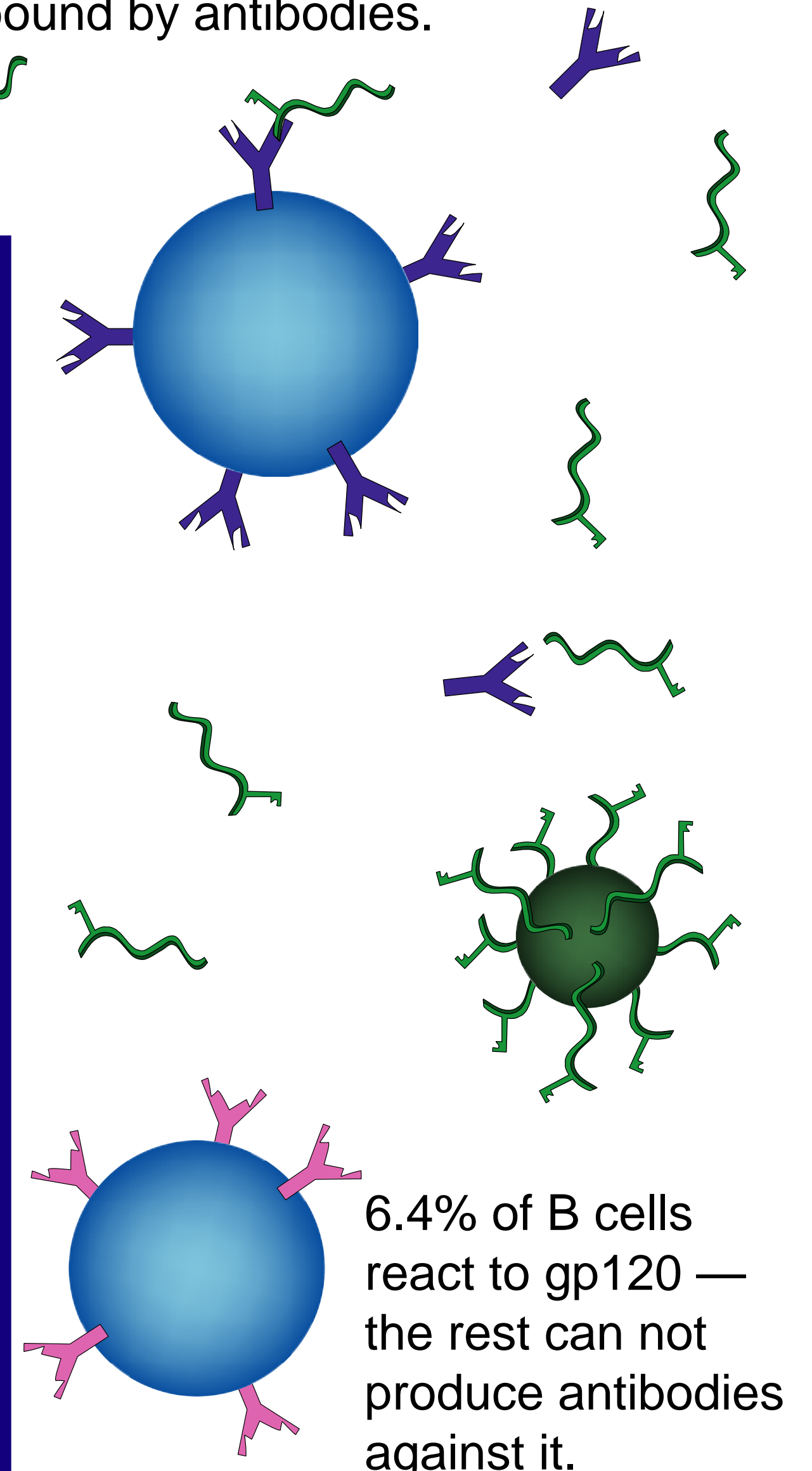


## 5. Antibodies bind gp120

Antibody targeting of uninfected T cells, whose CD4 receptors are covered with gp120, is 100 times more effective than targeting of infected T cells or free virus, whose gp120 simply disassociates upon being bound by antibodies.

## Bystander Cell Death Causes AIDS:

If T cell death was caused exclusively by direct infection by HIV, clinical studies should show large fractions of T cells infected by the virus (at left). Instead, studies show that no more than 1% of T cells are infected by HIV — but up to 80% are uninfected and dying (at right).



6.4% of B cells react to gp120 — the rest can not produce antibodies against it.