Solutions

Marking: **M1** a correct method, **A1** the correct answer, **B1** an independent mark.

Each answer shows the steps, then the **result**.

2.1

S: $8/24 \approx$ **33**%

M: $1/24 \approx$ **4**%

2.2

$2^{30} \approx 1.07 \times 10^{9}$

30 doublings

10 more, since $2^{10} \approx 1000$

2.3

apoptosis is programmed and uses energy; necrosis follows injury

apoptosis leaves neat fragments that are eaten with no inflammation; necrotic cells burst and cause inflammation

2.4

oncogenes drive division; a change that makes them more active is a gain of function

one changed copy is enough

tumour suppressors brake division; both copies must be lost (two hits)

3.1

(a) 30 doublings $\times$100 days = 3000 days $\approx$ **8.2** years

(b) 10 doublings = 1000 days $\approx$ **2.7** years

(c) most of a tumour's life passes before it is 1 cm; finding it smaller gives years to treat it before it is large or has spread

(d) many tumour cells die, or are short of blood supply, so the net growth is slower

3.2

(a) every cell starts with one hit already, so one more chance hit in any cell removes p53 completely

(b) carrier: $10^{8} \times 10^{-6} =$ **100** cells; non-carrier needs two hits: $10^{8} \times 10^{-12} =$ $\mathbf{10^{-4}}$

(c) the carrier's risk is about a million times higher, so cancers come early and often

3.3

(a) tumour A: ten times as many of its cells are dividing at any time, so more are hit by each dose

(b) hair roots, the mouth and gut lining, and bone marrow also divide fast, so their cells are killed too

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(c) normal tissue such as marrow recovers faster than the tumour, so each gap lets the normal cells regrow while the tumour shrinks a little more with each cycle

(d) check blood counts before each cycle and delay the dose if they are low; treat infections early, and use mouth care and anti-sickness medicine