SCN: SDXR8

1.

$$D = \Psi \left(\frac{\mu_{en}}{\rho}\right)$$

energy fluence $= E\Phi$ (due to 2cm glass)

$$= 300\times10^{3}\times1.602\times10^{-19}\times\frac{2\times10^{9}}{4\pi(50)^{2}}\times(0.9)^{2}$$

$$= 2.4783...\times10^{-9}$$

$$D = (2.4782...\times10^{-9})(31.6) = 7.83...\times10^{-8}\ J\,kg^{-1}$$

↑ This is the dose rate

30 minutes × 60 seconds × $D = 1.4096...\times10^{-4}$

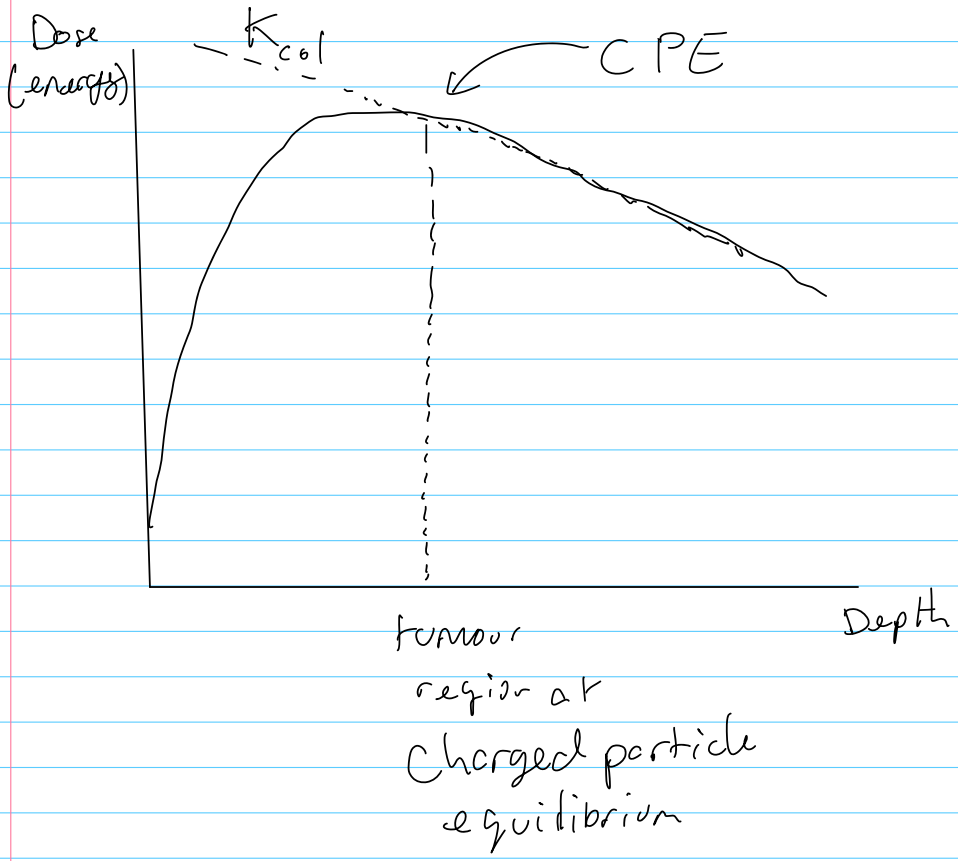
if she works 200 days across the year her annual dose rate is

$200\times1.4096\times10^{-4} = 0.0282$ (3sf)

2. a)

Photons will transfer their energy to the electrons completely, and this will mean that the photon energy is given as kinetic energy to the electrons. Since some of this energy is lost due to radiative energy losses that produce a photon, the dose absorbed is the transferred energy minus these losses. Therefore, the absorbed dose is the total energy in minus the total energy out.

Kerma represents the kinetic energy released per unit mass for the energy transferred from the photons to the electrons. The total kerma is the kinetic energy released for collisional interactions added to radiative interactions, thus the collisional kerma represents the net energy transferred (etot - er). This collisional kerma is the same as absorbed dose in the case of charged particle equilibrium, where equal intensities of electrons are going in as leaving. Thus, the only kerma energy to be considered here would be collisional interactions and thus the collisional kerma is equal to absorbed dose for charged particle equilibrium.



b) Bragg-Gray Cavity theory is that the cavity needs to be small enough to not affect the fluence of charged particles. Also, absorbed dose in the cavity is absorbed entirely by the charged particles crossing it.

This theory is required in combination with Charged particle equilibrium (CPE) to measure the dose at a given depth in the material. This is because:

$D = K_c = \Phi\left(\frac{\mu_{en}}{\rho}\right)$, assuming Bragg-Gray & CPE

This formula can thus be used to calculate the absorbed dose.

3.

A electron beam of 20 MeV can be used for treating deeper tumours in the abdomen using radiotherapy. This area is relatively mobile which is a limitation of this procedure as the abdomen easily moves. Since proton beams have a higher PDD peak as they are more concentrated in one area, as compared to electron beams. They also can reach lower depths than electron beams due to this focusing of the beam.

Proton therapy is often used for young people as they are highly vulnerable to the effects of radiation. Thus, the 200 MeV beam of protons can be used in this case to reduce this exposure. This would be especially true for brain tumours that require lots of care around the region as the PDD of proton beams is more focused (peaker curve) than electron beams. Electron beams would cause too much damage to healthy neighbouring areas.

4. a) PTV – Planned Target Volume – this is a geometrical concept that accounts for patient set-up errors.

CTV – Clinical Target Volume – a volume containing the subclinical microscopic disease

GTV – Gross Tumor Volume – The gross palpable or visible extent and location of malignant growth, (The macroscopic manifestation of the disease).

b) PTV margins are grown based on known random and systematic errors that are inserted into a recipe. The recipe is designed to deliver the clinical target volume minimum dose for a predefined percentage of patients.

PTV Margin Recipe:

$$\text{PTV Margin} = 2.5\Sigma + 1.64\sqrt{(\sigma_p^2 + \sigma^2)} - 1.64\sigma_p$$

σ = quadratic Sum of all treatment errors (random = blur dose)

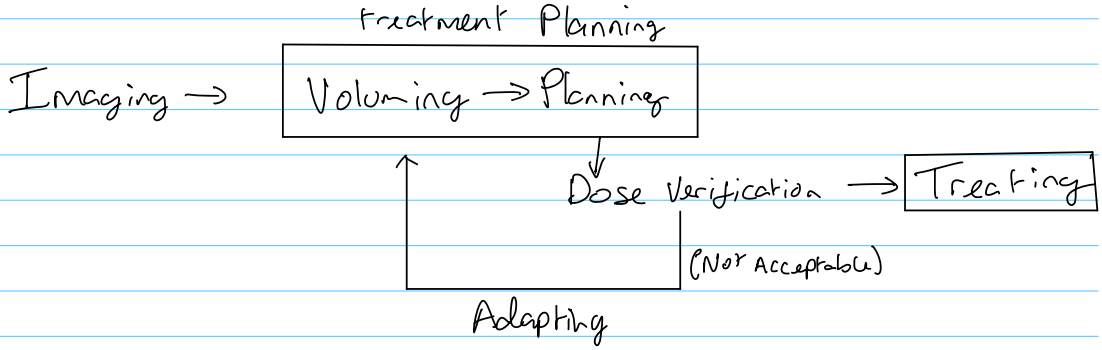
Σ = quadratic Sum of all preparation errors (systematic = Shift dose)

σ_p = beam penumbra (photons → σ_p = 3.2mm over range of 0-5mm)

In order to ensure 90% of patients have CTV coverage of >95% of the prescribed dose. This gives the coefficients of the terms as:

$$\therefore \text{PTV Margin} = 2.5\Sigma + 0.7\sigma$$

c) External Beam Radiotherapy:



- Firstly, the patient would have physical exams, which would lead to the patient undergoing imaging to identify the characteristics of the illness. The imaging modalities can be CT or MRI to identify the location of the tumour in the breasts. The likely modality would be MRI as it has better soft tissue contrast to identify the tumour

- Radiation therapists may use a mask or pad to keep the patient still during imaging

- The scans will be used by dosimetrists, oncologists and medical physicists to determine the location and volume of the tumour/tumours. Computer aid will be used in the form of an algorithm to compute the PTV and CTV to find the optimal spatial and geometric region for irradiation.
  - This is <u>volumning</u> which determines the CTV, GTV and PTV

- Then, the <u>planning</u> stage is when software such as Monte Carlo simulations can be used to determine the treatment parameters like dose, angle of beams, size and shape of beams, and number of treatment sessions.

This stage sets out the objectives of the treatment plan and parameters.

- The verification stage is used to model the treatment using the voluming and planning parameter set. If this is accepted then treatment goes ahead, otherwise it goes back to the planning stage to adjust the treatment.

5. a)

The radiation dose response curve shape is due to 5e fact that as the dose increases the damage done to the cells increase too. Therefore, as the dose increases the cell damage starts of small due to small amounts of double strand breaks, but as the dose increases far more double strand breaks occur and this increases the likelihood of the cell dying. This increases to a maximum and then decreases. The goal is to achieve a high probability of local tumour control while minimising the damage done to normal tissue cells. Thus, this shape of the curve can be used therapeutically as the TCP curve is different to the NTCP curve, so more damage is done to tumour cells and this makes radiotherapy a suitable option for treating cancer tumours as the healthy cells may be able to repair themselves and recover up to a point while the tumour cells are killed.

b) Single Strand breaks:

This is a form of radiation DNA damage that leads to the break of one of the helix bonds of the DNA strand.

In the case of 1Gy of gamma rays being used to irradiate a region, around a 1000 single strand breaks are produced. Therefore, the number of single strand breaks is far higher than double strand breaks during radio therapy.
The number of single strand breaks is linearly related to the dose, so higher doses leads to much more damage.

However, despite this type of DNA damage occuring frequently, the repair of this type of damage is quick and accurate. Therefore, the consequence is that there are few cells killed relative to double strand breaks.

Since this type of damage results in loss of breaks, considering the survival curve of a tumour region would show that with smaller doses the curve follows the optimal linear line initially, but as the dose gets larger and the resulting numbers of single strand breaks get proportionally larger, the repair mechanism becomes saturated. This results in the sub-lethal (βD^2) section of the damage on the survival curve, which is where the cells exponentially begin to drop. So, the single strand breaks cause much of the radiation-induced cell death only with larger doses.

Double-Strand Breaks:

Double Strand breaks are less common, as 1 Gy of Gamma rays will result in 80-100 double Strand breaks. The no of double Strand breaks relative to dose is linear or linear-quadratic.

The repair of these breaks occurs, but due to the extent of the damage, it is far less accurate. Due to the extent of the damage, many cells with this damage are killed as the repair is insufficient.

Thus, this is a far more potent form of DNA damage due to the double Strand breaks in the DNA helix. Considering the Survival curve, lower doses cause double Strand breaks and thus most of the cell death is in the lethal region of the curve (αD), which is caused by these double Strand breaks. As higher doses are used, the combination of more double Strand breaks and far more Single Strand, with Saturation of the repair mechanism leads to exponentially higher cell death. So, the curve decays rapidly. Hence, fractionation Should be used to repeatedly induce cell death in a more consistent fashion or the damage to the healthy cells in the region would be unacceptable.

c)

4Rs - Repair, Reassortment, repopulation, reoxygenation

Repair - during single strand breaks, the repair is quick and accurate, while, in the case of double strand breaks the repair is far less accurate. This is because the repair of double strand breaks often lead to Dicentric and Ring formation, instead of normal chromosomes. Therefore, single strand breaks lead to few cells dying, while the double strand breaks lead to many cells dying. The lethal damage section of the survival curve displays very ineffective repair due to the presence of double strand breaks, as the dose is increased the repair mechanism is saturated and even the single strand breaks begin to result in cell death due to inaccurate repair.

Reassortment - this represents the cell cycle, where there are four stages - mitosis, pre-synthesis gap, DNA duplication, and post-synthesis gap. The times take 1 hour, 8-12 hours, 9-12 hours, 4-6 hours. This is an important note in radiobiology as the mitsosis phase is radio sensitive and the DNA duplication phase is radio resistant, which makes them more and less vulnerable to radiation respectively. Surviving cells will continue the cycle and then may be in the sensitive phase when the next fraction of radiation is given.

Repopulation - this is the cell's response to radiation leading to an increase in the cell division. This is important since in healthy tissues the Acute responding tissues start the process early, while late responding ones start later potentially after radiotherapy. Meanwhile, for malignant tissues the same process applies but for some malignant cells they undergo accerlated repopulation with a decrease in cell cycle time, so some treatments will need to be extended to counter this effect.

Reoxygination - during fractionation, this is the process where the outer cells of the tumour are reoxyginated as the healthy cells around the tumour are initially killed after fractionation. This oxygenates some of the hypoxic and anoxic cells such that they can be killed in the next fraction as cells with oxygen are more sensitive to radiation.

6. i) $Q = 7.5\,\mu C$ $\quad E = 662\,keV$

$$X = \Psi \left(\frac{\mu_{en}}{\rho}\right)_{air} \left(\frac{e}{W_{air}}\right)$$

$$\Psi = E\Phi = (662\times10^{3})(1.602\times10^{-19})\ 7.5\times10^{-6} \times \frac{6.24\times10^{18}}{4\pi(10)^2}$$

$$= 3.9496\ldots\times10^{-3}$$

$$X = (3.9496\ldots\times10^{-3})(29.2)\left(\frac{1}{33.97}\right)$$

$$= 3.395\ldots\times10^{-3} = 3.4\times10^{-3} \quad (2s.f.)$$

ii) $D_{air} = K_C = \Psi\left(\frac{\mu_{en}}{\rho}\right)$

$$= (3.9496\times10^{-3})(29.2) = 0.1153\ldots$$

$$= 0.12$$

$$D_{graphite} = D_{air}\frac{\left(\frac{\mu_{en}}{\rho}\right)_m}{\left(\frac{\mu_{en}}{\rho}\right)_{air}} = (0.1153\ldots)\frac{(29.3)}{(29.2)}$$

$D_{graphite} = 0.116\ J\,kg^{-1}$

iii) if absorbed dose is $0.116\ J kg^{-1}$, then:

$$D = \Psi \left(\frac{\mu_{en}}{\rho}\right) \quad \therefore \quad 0.116 = E \frac{x}{4\pi r^2} \left(\frac{\mu_{en}}{\rho}\right)$$

$$0.116 = (662 \times 10^3)(1.602 \times 10^{-19}) \frac{x}{4\pi (10)^2} (29.3)$$

$$x \simeq 4.6911 \ldots \times 10^{13} \text{ Bq}$$

$$= 4.7 \times 10^{13} \text{ Bq for activity.}$$