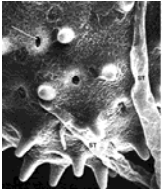


Write succinct answers in the space provided after each question, or, if you need more space, continue your answer on the back of the page. The potential value of each answer is 3 pts. unless otherwise noted.

1. (a) In the image below, what is the structure labeled "ST", and what two molecules interact on the surface of ST that influence whether certain kinds of pollen will germinate?



Ans.: The structure is the stigma (or the papilla cell of the stigma), and the two molecules would be SCR (male factor) and SRK (female factor), which control whether sporophytic self-incompatibility occurs...

(b) What is ARC1 and how it changed when an incompatible reaction occurs?

Ans.: ARC1 is a E3 ligase protein that is phosphorylated by the activated SRK receptor

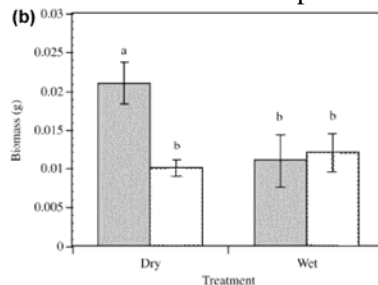
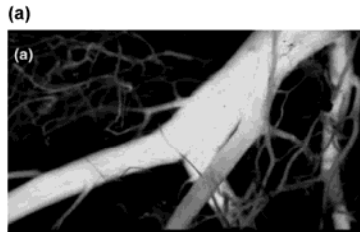
(c) In what cell type and in what subcellular locale is the GCS1 protein expressed, and why are *gcs1* mutants not fertile?

*Ans.: The GCS1 protein is expressed on the surface of pollen sperm cells, and *gcs1* mutants are not fertile because in these mutants the sperm does not fuse with (and fertilize) the egg.*

2. (a) Name three different genes that encode blue light photoreceptors and indicate one blue-light response each one regulates.

Ans.: Cry1, phot1 and phot2 are three different genes that encode blue-light photoreceptors. Cry 1 activation suppresses hypocotyl growth; Phot1 & Phot2 together mediate blue light-induced stomata opening & phototropism.

(b) In the image (a) below, a protein is visualized in roots. What method is used to visualize that protein, and when this method was used, where was the visualized protein preferentially localized in roots?



Ans.: Phototropin is visualized as a phototropin-GFP hybrid protein, which fluoresces green, and it was visualized primarily in the upper part of roots.

(c) In image (b) above, under dry conditions, wild-type plants (dark bar) do better than mutant plants (white bar) that are missing the protein visualized in image (a), but both wild type and mutant plants perform the same in wet conditions. What is the most plausible explanation for these results?

Ans.: Wild-type plants, unlike mutant plants, express the normal level of phototropin, and this photoreceptor induces negative phototropic responses in roots, allowing them to grow straighter and deeper into the soil and thus better harvest deeper water sources during a drought. When water is plentiful, the presence of root phototropin does not confer any particular advantage for growth.

(d) What is a major unanswered question about the function of the photoreceptor in the signal transduction chain that connects the light signal to stomatal opening?

Ans.: It is not clear what target protein other than phototropin itself is phosphorylated by phototropin to mediate blue-light-induced stomatal opening

(4 pts.) 4. (a) If you measured the quality of light on the jungle floor of a dense tropical forest what color of light would dominate and why?

Ans.: Far- red light would dominate, because the green leaf canopy of the jungle would absorb most of the other colors of sunlight except far-red, which it would transmit to the jungle floor.

(4 pts.) (b) Unbeknownst to most historians, Tarzan was a botanist, and he noticed that certain seeds did not germinate on the jungle floor. He wondered whether the reason for this was because of the low level of light or because of the peculiar quality of light there, and he asked you to design an experiment that would resolve this question. Describe such an experiment, its alternate possible outcomes, and which alternative hypothesis would be eliminated by each outcome.

Ans.: Place a red filter over the seeds. This would both lower the quantity of light and change the dominant color of light from mostly far-red to mostly red. If low light intensity was the main reason the seeds did not germinate, then placing the filter over the seeds would not change the result. If light quality was the main reason the seeds did not germinate, then changing the color to red would induce them to germinate.

(4 pts) 5. (a) What is the function of an SCF complex, and what does it have to do with auxin action?

Ans.: The SCF complex serves as an E3 ligase promoting the ubiquitination and, thus, proteolytic destruction of target proteins. One auxin receptor, TIR 4, is an F-box protein, so, upon activation it functions in the SCF complex targeting repressors of auxin-regulated genes.

(4 pts) (b) What is an ARF and what kind of protein normally binds to it and regulates its function?

Ans.: An ARF is a transcription factor that serves as a positive regulator of auxin-induced gene expression. Aux/IAA proteins can bind to ARFs in the absence of auxin and block their ability to turn on promoters for auxin-induced genes.

(c) Why do scientists think there may be other kinds of auxin receptors different from the TIR proteins?

Ans.: Because physiological evidence indicates some auxin receptors are probably on the plasma membrane, and TIR proteins are localized inside the cell.

5. (a) What is ETR1, where is it located, and what 2 binding sites are critical for its function?

Ans.: ETR1 is an ethylene receptor localized on ER membranes. It has a binding site for ethylene and a binding site for CTR1, a negative regulator of ethylene responses.

(4 pts) (b) What is the function of EBF proteins, and if the gene encoding it is knocked out, how would the ethylene response of the knockout mutant differ from that of wild-type plants, and why?

Ans.: EBF proteins are F-box proteins, whose expression is up-regulated by ethylene. They target EIN transcription factors for ubiquitination and proteolytic destruction. Because EIN proteins are positive regulators of ethylene-induced gene expression, mutants KOed in EBF would have a slightly greater sensitivity (hypersensitivity) to ethylene, since EIN would be more stable.

(c) What is the functional relationship of CTR1 to signaling steps downstream of it ?

Ans.: As long as CTR1 is bound to ethylene receptors it represses (negatively regulates) downstream signaling steps in the ethylene transduction pathway. The binding of ethylene to its receptors dissociates CTR1 from the receptor and allows downstream signaling steps to be de-repressed.

6. (a) In cytokinin signaling, distinguish the function of B-type ARR from that of A-type ARR.

Ans.: B-type ARRs are positive regulators of genes that promote cytokinin responses; some A-type ARRs are negative regulators of cytokinin responses.

(b) In cytokinin signaling describe the various transfers of a phosphoryl group from its initial site on the activated receptor to AHP. Include which amino acid side chains it moves to and from.

Ans.: Phosphoryl group is transferred from a histidine to an aspartic acid on the activated receptor, and then from that aspartic acid to a histidine on the AHP.

(c) How does phosphorylation of AHP allow it to affect cytokinin-induced transcription changes?

Ans.: After its phosphorylation AHP moves from the cytoplasm to the nucleus where it transfers a phosphoryl group to B-type ARRs, thus activating them.

(4 pts.) 7. (a) What are two major physiological responses controlled by ABA and how does ABA affect them?

Ans.: ABA suppresses seed germination and promotes stomatal closure.

(b) Early experiments had suggested that extracellular perception is critical for ABA to achieve its functions. Why is it currently difficult to understand how ABA does this?

Ans.: Currently there is no clear candidate for a plasma-membrane localized receptor that could be activated by extracellular ABA.

8. (a) In wound signaling describe one step upstream and two steps downstream of systemin that could lead to the production of protease inhibitors, and for one of these steps describe why that step would be necessary for the next step in the transduction chain.

Ans.: A step upstream of systemin would be ATP release at wound site and activation of wound signaling pathways, including superoxide production. Three steps downstream would include lipase activation producing linolenic acid, which would induce JA production. Lipase activation would be needed to produce the free fatty acid linolenic acid from membrane phospholipids.

(b) Describe an experiment that proves one of the steps in the wound signaling pathway is needed to provide the plant some benefit in its defense against insect feeding.

Ans.: KO of gene either for prosystemin or lipoxygenase (which catalyzes the production of JA) prevents protease inhibitor production, and these plants are more susceptible to damage from insect feeding.

(c) Proteases are needed by plants for their normal metabolism, so why aren't the protease inhibitors induced in plants by wounding harmful to plants?

Ans.: Because plants package the inhibitors in vacuoles, where it cannot damage cytoplasmic proteins

9. (a) What is the hypersensitive response, what hormone induces it, and what is the survival benefit of this response?

Ans.: The hypersensitive response, which is induced by salicylic acid, is programmed cell death that is induced in plant tissue by pathogen infection. The survival benefit is that it severs the connection between the infected cell and adjoining uninfected cell and thus inhibits the spread of the pathogen in the plant.

(b) Define SAR and describe an experiment that suggests that the hormone that promotes SAR cannot be the agent that moves from the original site of infection to distant uninfected sites and signals those sites to begin SAR.

Ans.: SAR is systemic acquired resistance, a response that is induced in uninfected parts of a plant after the plant is infected by pathogens. Salicylic acid (SA) is needed for the SAR response. An experiment that suggested SA cannot be the mobile agent to induce SAR showed that infected leaves that were expressing

the *NahG* gene and thus had reduced SA levels, could be grafted on to uninfected plants and induce SAR in them.

10. (a) When G-protein-linked receptors are activated, this often leads to an increase in $[Ca^{2+}]_{cyt}$. How does this happen?

Ans.: Activated receptors activate $G\alpha$, which activate PLC, which generates IP3 from PIP2, and this IP3 can induce the opening of intracellular calcium channels, releasing Ca^{2+} from internal stores, thus increase $[Ca^{2+}]_{cyt}$

(b) The earliest report of phytochrome-induced change in calcium transport in 1980 revealed that red light induced an efflux of calcium from cells. Later in 1992 another paper reported that phytochrome induced a rapid increase in $[Ca^{2+}]_{cyt}$. Explain why the results of these two papers are not contradictory.

Ans.: A phytochrome-induced increase in $[Ca^{2+}]_{cyt}$ would activate calmodulin, which would bind to and activate calmodulin-dependent calcium pumps on the plasma membrane, which would pump calcium out of the cell.

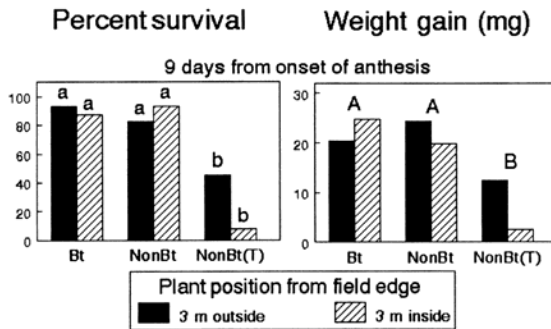
(c) In measuring the effects of a stimulus on $[Ca^{2+}]_{cyt}$, why would it be useful to record how often the $[Ca^{2+}]_{cyt}$ changed during the first minutes after the stimulus was given?

Ans.: The frequency of calcium spikes (peaks followed by troughs) induced by different stimuli can be different and these differences can lead to different downstream signaling steps, thus helping to confer more specificity to calcium signaling.

(4 pts.) 11. (a) What result led scientists to think Bt corn might represent a threat to Monarch butterflies?

Ans.: In a laboratory experiment, scientists fed larvae of Monarch butterflies a very high dose of pollen from Bt plants, and many of them died.

(4 pts.) (b) How do the data below relate to the concern raised in Question 11a?



Survival and weight gain of monarch larvae feeding on milkweeds placed at 3 m inside and outside the edge of plots consisting of Bt11, non-Bt (untreated), and non-Bt (cyhalothrin-treated) sweet corn.

Ans.: This experiment shows that in a real-life field test, larvae of Monarch butterflies survived just as well on bt corn plants as on non-bt corn plants whether they were monitored 3 meters inside or 3 m outside the corn field, and they survived a lot better than they did on corn plants that were treated with insecticide.