Question 1

Biological systems are frequently subjected to a range of dynamic stimuli, creating perturbations that challenge its ability to perform its intended function with accuracy (Hiroaki Kitano & Fumitoshi Matsuno, 2008). Organisms have evolved to withstand these fluctuations in the system through an array of robust traits and homeostatic-feedback mechanisms. This essay will define biological robustness and fragility with examples, then discuss why biological robustness is an integral part of survival and the consequences of fragility and how to avoid them.

Biological robustness can be defined as a characteristic or trait of a system that sustains its functionality despite internal or external perturbations (Hiroaki Kitano & Fumitoshi Matsuno, 2008). These traits are observed pervasively through all levels of biological organisations, from systemic to the cellular and to molecular levels of organisms. Some examples of such traits could include pattern segmentation during embryogenesis, correct protein folding, transcriptional regulatory networks within gene expression pathways, cell cycle regulations, etc. Often, disruptions to said systems arise in the forms of genetic mutations, physical and chemical environmental variations, like that of physical trauma or a shift in the metabolic or chemical equilibrium.

The chemotaxis adaptation mechanism of E. coli is a classic example of biological robustness. E. coli bacteria use chemotaxis to move towards nutrient rich environmental zones and away from areas sequestered with toxins and other competing bacterial strains. E. coli bacteria can fine tune their chemotaxis response by modifying the sensitivity of their chemoreceptors through dimerization of receptors (Ames et al., 2002) via methylation and demethylation processes through the regulation of CheR and CheB enzymes (Hansen et al., 2008). A complex network of signalling pathways relay information from these chemoreceptors to the flagella motors through phosphorylation between receptors and regulatory proteins CheA and CheY , hence, regulating favourable flagellar movement. Such traits are pervasive in its ability to adapt and maintain robustness in environmental conditions that are in a constant state of flux.

The presence of robust phenotypic characteristics is integral to the fitness of an organism. The simple explanation to survivability is achieved through homeostatic regulations of pathways within a biological system in response to environmental or internal stressors, as seen in the E. coli example above. However, robustness is not limited to stability (Waddington, 1953) but in fact needs to be adaptive in order to withstand environmental variability to ensure the viability of reproductive organisms (Hiroaki Kitano & Fumitoshi Matsuno, 2008). Adaptive robustness also allows for a varied level of functionality to support the fitness of an organism. As seen in tardigrades, that can transition into extremophiles and remain viable under extreme temperatures and pressures (Seki & Toyoshima, 1998). In essence robustness traits ensures that organisms can adapt to ever-changing environmental conditions and maintain functionality and viability.

Despite such robust traits in place, biological systems are still susceptible to damage and malfunction. Biological fragility can be defined as the inability of a system to maintain functionality in the presence of perturbations in the system (Rihn et al., 2013). They can be caused by various factors, including genetic mutations, environmental stressors, aging and disease epidemics to name a few. An example of biological fragility can be observed through the perturbations of the heat-shock protein (Hsp90).

A study found that Hsp90 mitigates cryptic genetic variation, which produces morphological variation in Drosophila developmental pathways (Rutherford & Lindquist, 1998). When Hsp90 is compromised, the previously silent genes are perpetually expressed regardless of Hsp90 functional restoration (Rutherford & Lindquist, 1998). This depicts the fragility in the system where a single molecular mechanism is the gateway for disruption in Drosophila pattern segmentations resulting in a multitude of morphological variation some of which may be detrimental to survival.

Consequentially fragility can cause dysfunction within biological networks, altering an organism's survival and reproductive success (Pasqualetti et al., 2020). Often accumulation of genetic mutations within the genome can result in the fragility of gene expression systems. Sometimes even extremely robust traits become detrimental for an organism's ability to adapt to changing environments. However biological fragility can be overcome where organisms can influence their environment to mitigate exposure to harmful perturbations or to seek out suitable environments for survivability (Whitacre, 2012). This is observed within various species where the immediate environment is carefully designed and structured in order to achieve the optimal viability. For example, behavioural and cultural traits are taught and passed down from one generation to the next. Construction of safe and long-standing habitats (Laland & Sterelny, 2006) also allow for survivability of an individual and their offspring.

In conclusion, biological robustness and fragility is the ability or the inability, respectively, of an organism or system to sustain its functionality against perturbations. Robustness is required for a organism to sustain fitness, through various robust traits. However, not all robust traits can protect a system from fragility and damage. Biological systems are able to maintain resilient mechanisms through adaptation, and diversification and constantly need to fine tune between the system and the environment to ensure survival (Whitacre, 2012).

References

Ames, P. et al. (2002) “Collaborative signaling by mixed chemoreceptor teams in escherichia coli” Proceedings of the National Academy of Sciences, 99(10), pp. 7060–7065. Available at: https://doi.org/10.1073/pnas.092071899.

Hansen, C.H., Endres, R.G. and Wingreen, N.S. (2008) “Chemotaxis in escherichia coli: A molecular model for robust precise adaptation” PLoS Computational Biology, 4(1). Available at: https://doi.org/10.1371/journal.pcbi.0040001.

Hiroaki Kitano and Fumitoshi Matsuno (2008) “Biological robustness,” 2008 SICE Annual Conference [Preprint]. Available at: https://doi.org/10.1109/sice.2008.4654600.

Laland, K.N. and Sterelny, K. (2006) “Perspective: Seven reasons (not) to neglect niche construction,” Evolution, 60(9), p. 1751. Available at: https://doi.org/10.1554/05-570.1.

Pasqualetti, F. et al. (2020) “Fragility limits performance in complex networks,” Scientific Reports, 10(1). Available at: https://doi.org/10.1038/s41598-020-58440-6.

Rihn, S. et al. (2013) “Extreme genetic fragility of the HIV-1 capsid,” Retrovirology, 10(S1). Available at: https://doi.org/10.1186/1742-4690-10-s1-p73.

Rutherford, S.L. and Lindquist, S. (1998) “Hsp90 as a capacitor for morphological evolution,” Nature, 396(6709), pp. 336–342. Available at: https://doi.org/10.1038/24550.

Seki, K. and Toyoshima, M. (1998) “Preserving tardigrades under pressure,” Nature, 395(6705), pp. 853–854. Available at: https://doi.org/10.1038/27576.

Waddington, C.H. (1953) “Genetic assimilation of an acquired character” Evolution, 7(2), p. 118. Available at: https://doi.org/10.2307/2405747.

Whitacre, J.M. (2012) “Biological robustness: Paradigms, mechanisms, and systems principles,” Frontiers in Genetics, 3. Available at: https://doi.org/10.3389/fgene.2012.00067.