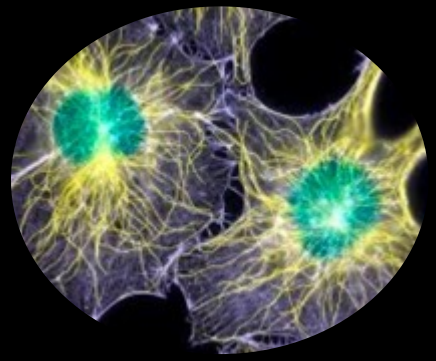

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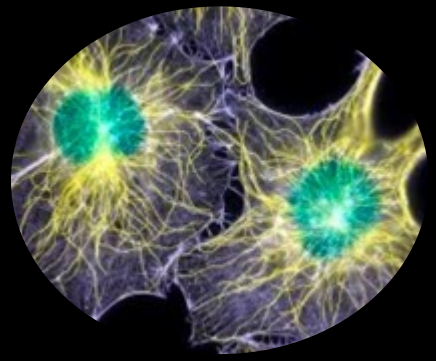
Mestrado em Biologia Molecular e Genética

2º Semestre

6 ECTS



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THE CONCEPT OF LIVING MATTER IN PHSYIOLOGY

Mestrado em Biologia Molecular e Genética

2º Semestre

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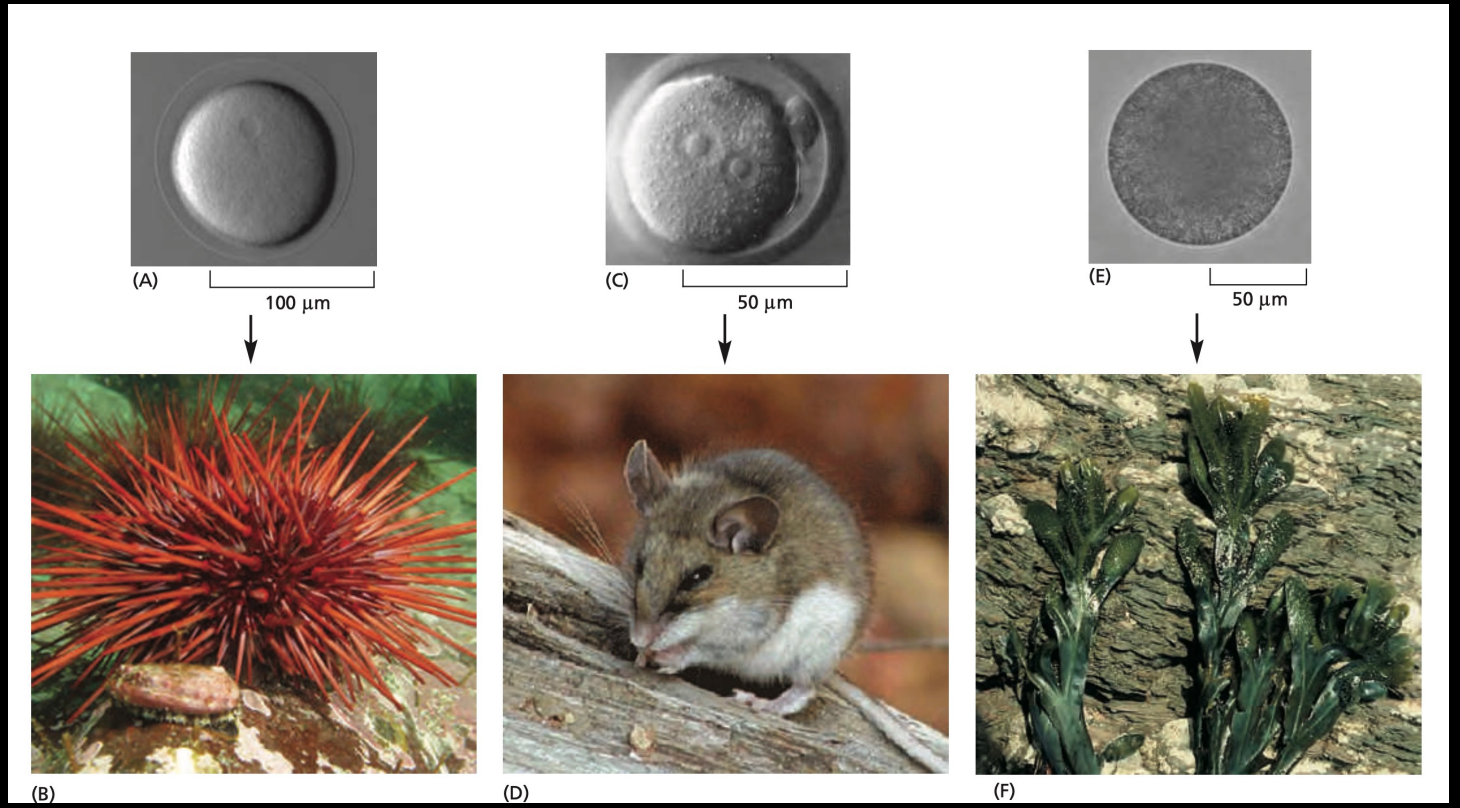
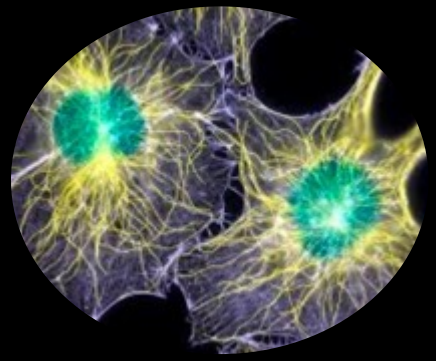
Homeostasy

- astonishing variety in individual particulars
- astonishing constancy in fundamental mechanisms





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Homeostasy

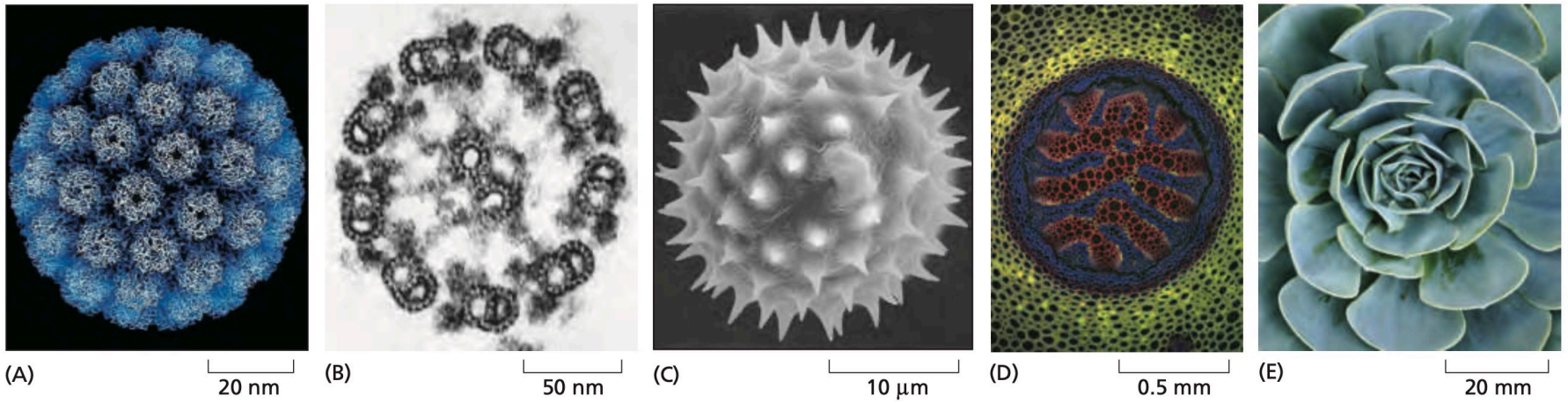

Figure 2–12 Biological structures are highly ordered. Well-defined, ornate, and beautiful spatial patterns can be found at every level of organization in living organisms. In order of increasing size: (A) protein molecules in the coat of a virus (a parasite that, although not technically alive, contains the same types of molecules as those found in living cells); (B) the regular array of microtubules seen in a cross section of a sperm tail; (C) surface contours of a pollen grain (a single cell); (D) cross section of a fern stem, showing the patterned arrangement of cells; and (E) a spiral arrangement of leaves in a succulent plant. (A, courtesy of Robert Grant, Stéphane Crainic, and James M. Hogle; B, courtesy of Lewis Tilney; C, courtesy of Colin MacFarlane and Chris Jeffree; D, courtesy of Jim Haseloff.)

The second law of thermodynamics

https://www.biologyonline.com/dictionary/homeostasis

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Homeostasy

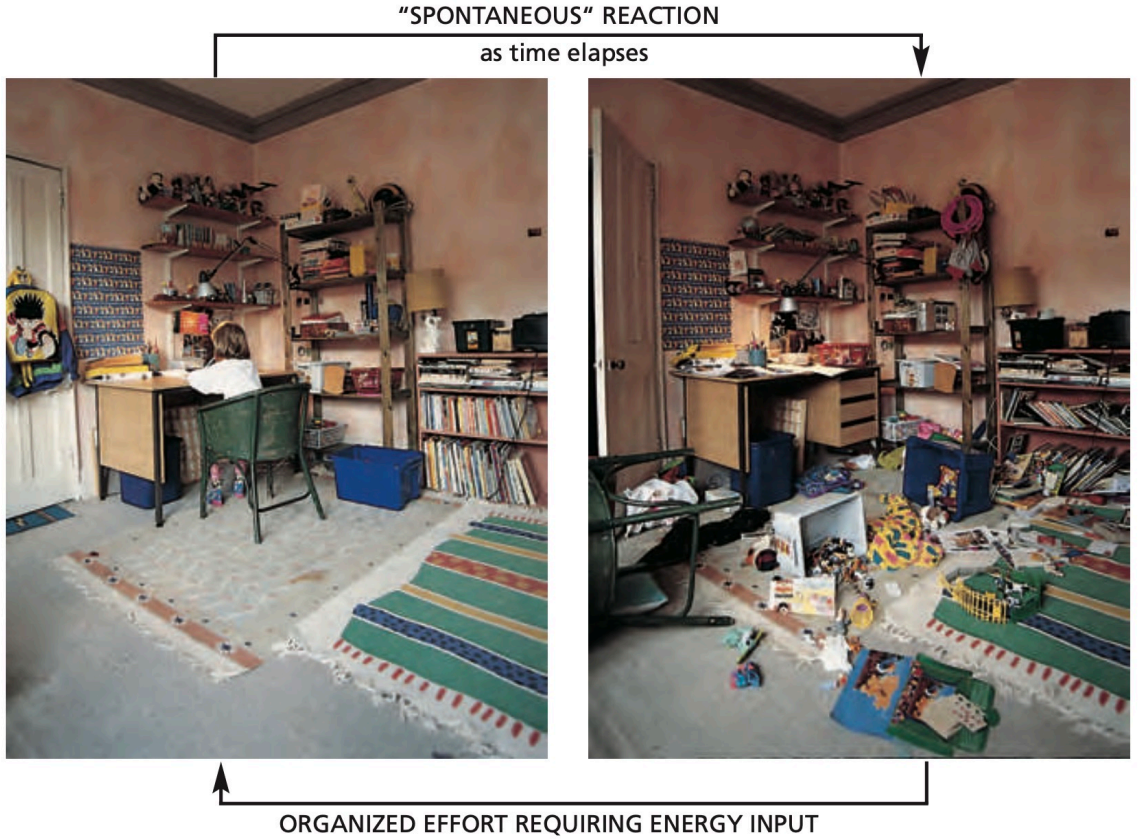
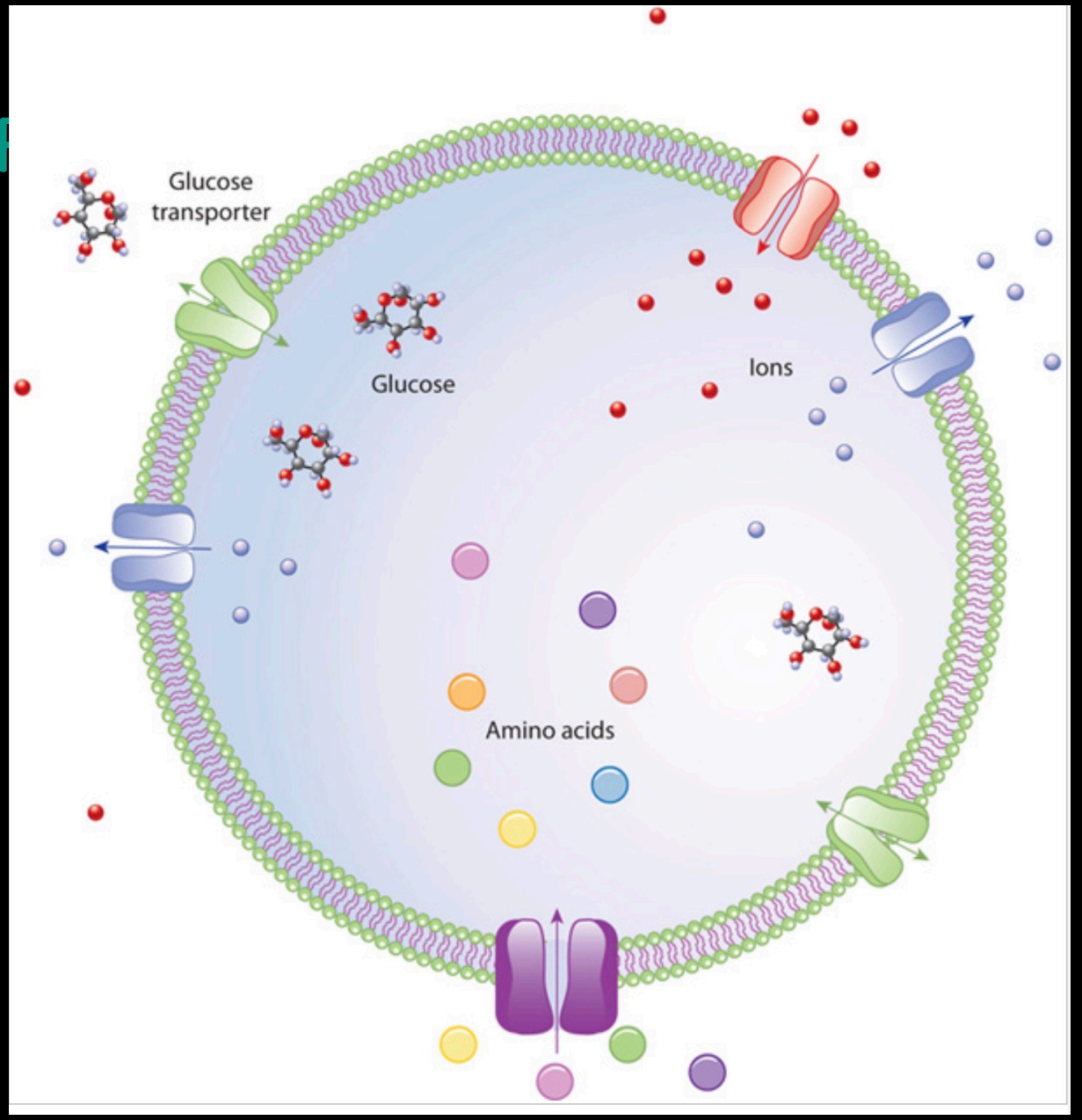
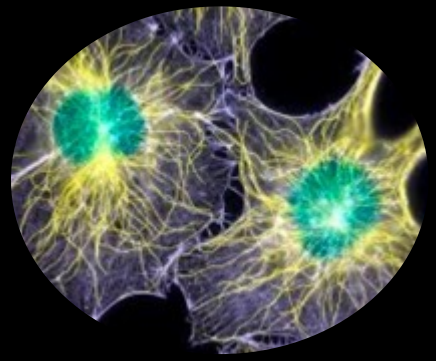


Figure 2–15 An everyday illustration of the spontaneous drive toward disorder. Reversing this tendency toward disorder requires an intentional effort and an input of energy: it is not spontaneous. In fact, from the second law of thermodynamics, we can be certain that the human intervention required will release enough heat to the environment to more than compensate for the reordering of the items in this room.

The membrane





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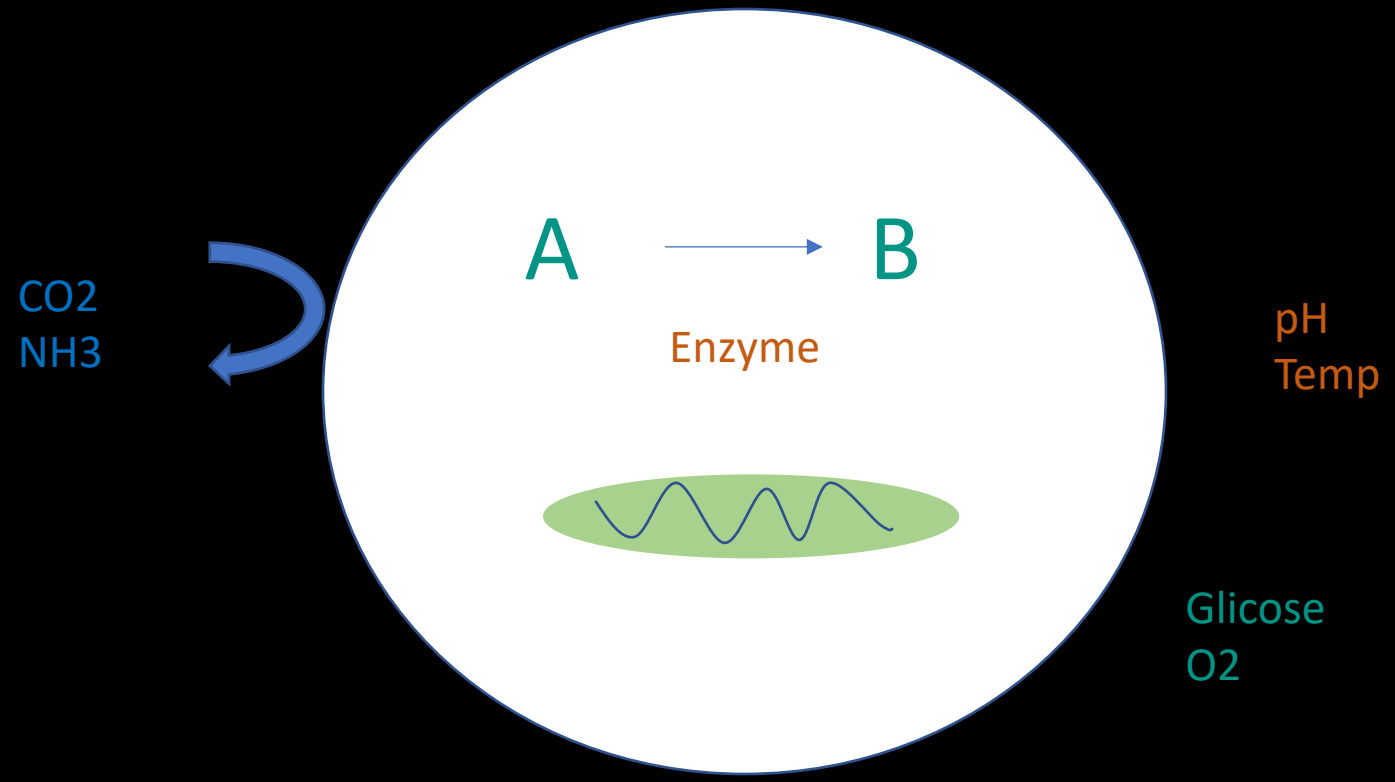

Davies 2016

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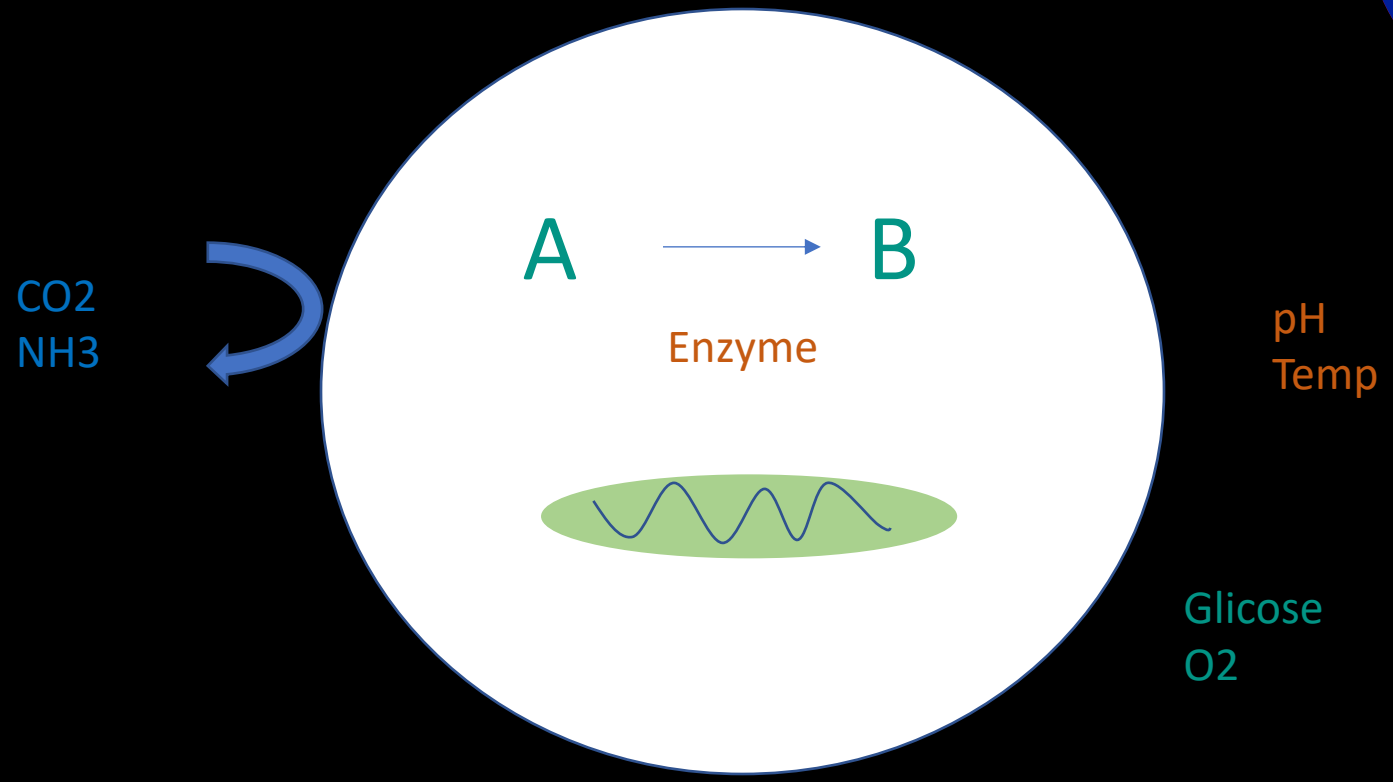
Homeostasis

life:)



Davies 2016

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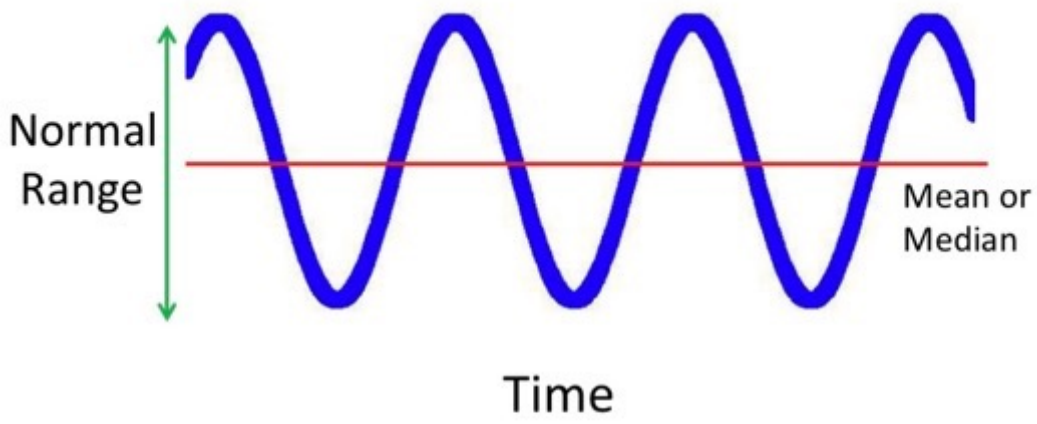
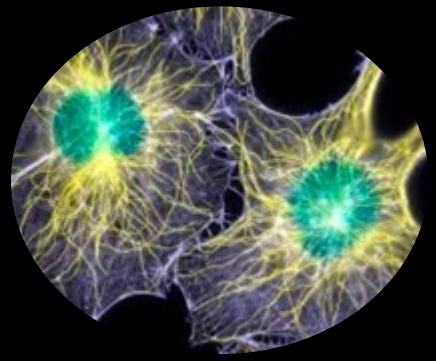


Fig. 3. A graphic depiction of the principle of homeostasis. According to Arthur C. Guyton's *Textbook of Medical Physiology* (Guyton, 1991), "The term homeostasis is used by physiologists to mean, maintenance of nearly constant conditions in the internal environment." Any biological function or measurement, therefore, will oscillate around a mean or median, within a range that is considered a 'normal' or physiological.

Davies 2016



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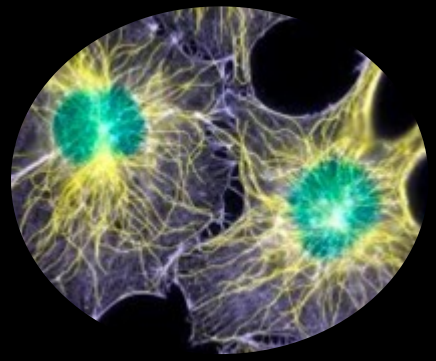
Homeostasis

1 - Constancy in an open system, such as our bodies represent, requires mechanisms that act to maintain this constancy.

2 - Steady-state conditions require that any tendency toward change automatically meets with factors that resist change.

3 - The regulating system that determines the homeostatic state consists of a number of cooperating mechanisms acting simultaneously or successively.

4 – Homeostasis does not occur by chance, but is the result of organized self-government



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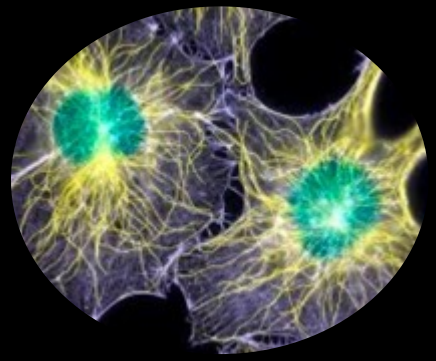
Homeostasis

Dynamic equilibrium

- Disruptors
- Detectors
- Control Systems
- Effectors



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Homeostasis

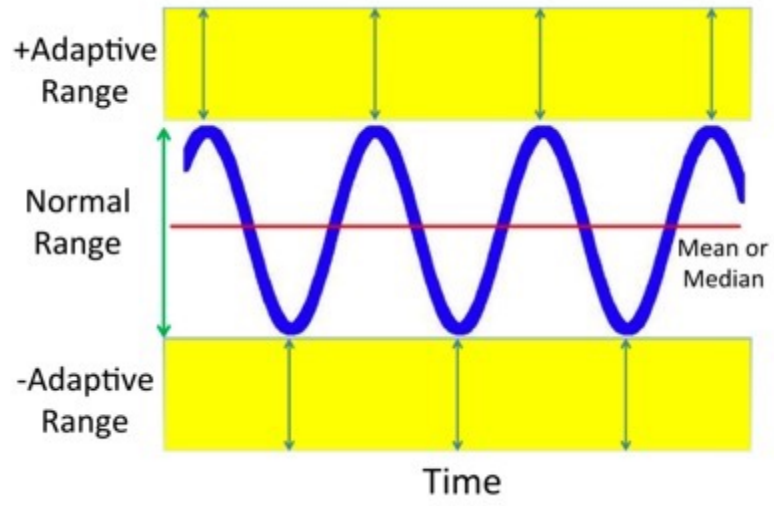

Fig. 4. A graphic representation of adaptive homeostasis. Here, in addition to the normal or physiological range, are added both positive and negative adaptive ranges that can be transiently induced via signal transduction pathways in response to sub-toxic, non-damaging, stimuli. Thus, for example, a signal given by nanomolar levels of H_2O_2 can act via the Keep1–Nrf2 system to increase synthesis of protective levels of proteasome, immunoproteasome, and Pa28 (or 11S) proteasome regulator for a period of several hours (Pickering et al., 2010, 2012, 2013): an example of positive Adaptive Homeostasis. Loss of the H_2O_2 stimulus then returns the system to the basal homeostatic range. Similarly, when organisms are exposed to a diet rich in amino acids, they turn off production of amino acid synthetases, thus decreasing capacity to synthesize amino acids: an example of Negative Homeostasis. Restoration of a 'normal' diet would then reverse the transient decrease in capacity back within the normal homeostatic range.

Davies 2016

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Homeostasy

Molecular Aspects of Medicine 49 (2016) 1–7

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journal homepage: www.elsevier.com/locate/mam

Review

Adaptive homeostasis

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Homeostasis

Review: How do SnRK1 protein kinases truly work?

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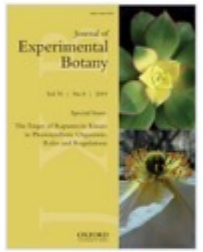
**Corresponding author: pcoello@unam.mx*

Highlights

- *SnRK1 specific complexes might form in response to a particular stress.*

Journal of Experimental Botany

SOCIETY FOR EXPERIMENTAL BIOLOGY



Volume 70, Issue 8
1 April 2019

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SnRK1 and TOR: modulating growth–defense trade-offs in plant stress responses

Leonor Margalha ✉, Ana Confraria ✉, Elena Baena-González ✉

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Abstract

The evolutionarily conserved protein kinase complexes SnRK1 and TOR are central metabolic regulators essential for plant growth, development, and stress responses. They are activated by opposite signals, and the outcome of their activation is, in global terms, antagonistic. Similarly to their yeast and animal counterparts, SnRK1 is activated by the energy deficit often associated with stress to restore homeostasis, while TOR is activated in nutrient-rich conditions to promote growth. Recent evidence suggests that SnRK1 represses TOR in plants, revealing evolutionary conservation also in their crosstalk. Given their importance for integrating environmental information into growth and developmental programs, these signaling pathways hold great promise for reducing the growth penalties caused by stress. Here we review the literature

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TABLE 3–3 Some Molecules Covalently Attached to Proteins Regulate Protein Function

Modifying group	Some prominent functions
Phosphate on Ser, Thr, or Tyr	Drives the assembly of a protein into larger complexes (see Figure 15–11)
Methyl on Lys	Helps to create distinct regions in chromatin through forming either mono-, di-, or trimethyl lysine in histones (see Figure 4–36)
Acetyl on Lys	Helps to activate genes in chromatin by mocifying histones (see Figure 4–33)
Palmityl group on Cys	This fatty acid addition drives protein association with membranes (see Figure 10–18)
N-acetylglucosamine on Ser or Thr	Controls enzyme activity and gene expression in glucose homeostasis
Ubiquitin on Lys	Monoubiquitin addition regulates the transport of membrane proteins in vesicles (see Figure 13–50)
	A polyubiquitin chain targets a protein for degradation (see Figure 3–70)

Ubiquitin is a 76-amino-acid polypeptide; there are at least 10 other ubiquitin-related proteins in mammalian cells.

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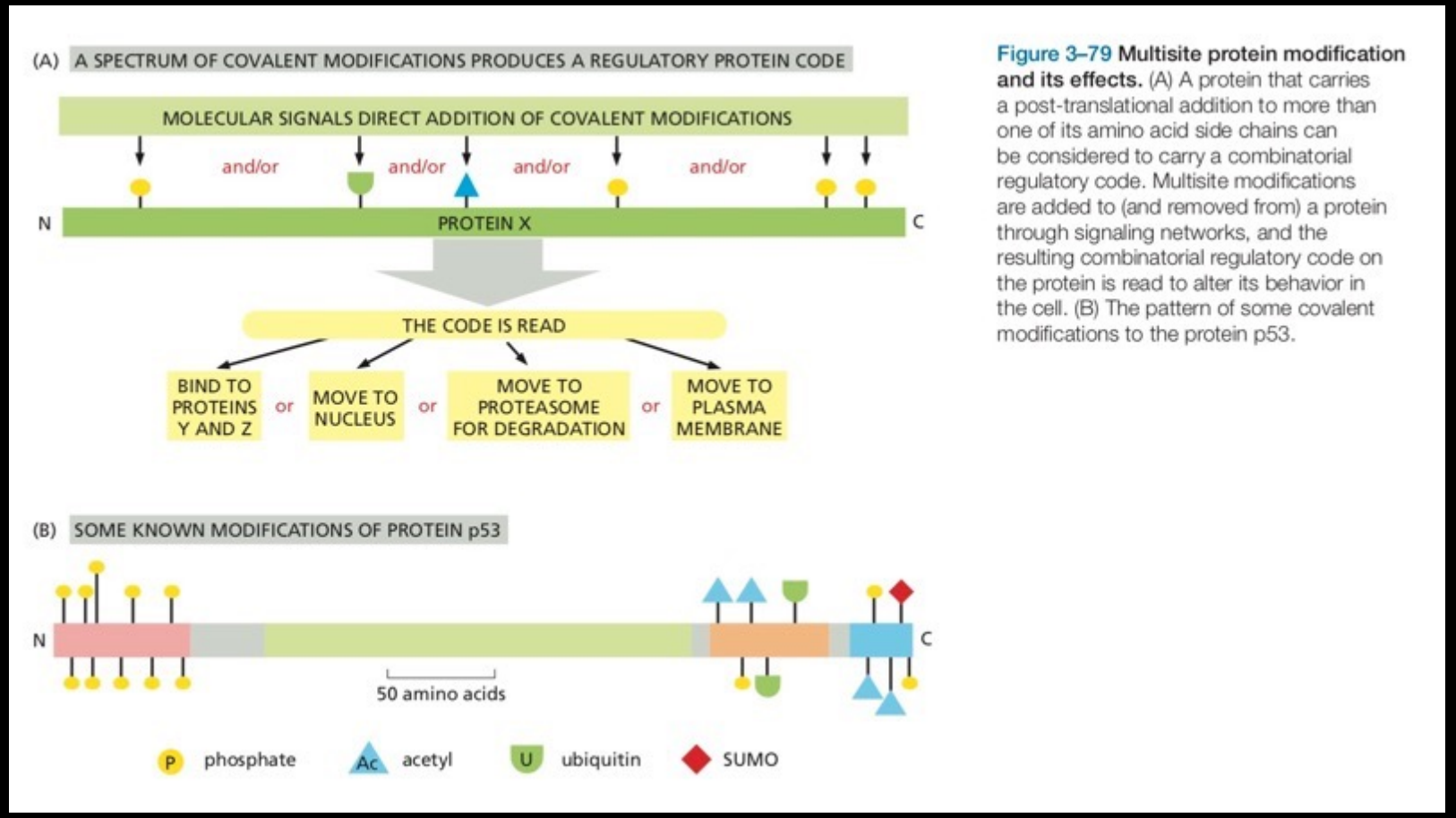
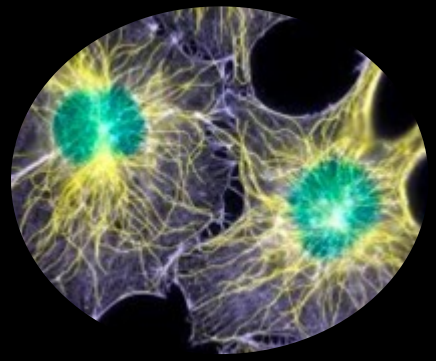


Figure 3–79 Multisite protein modification and its effects. (A) A protein that carries a post-translational addition to more than one of its amino acid side chains can be considered to carry a combinatorial regulatory code. Multisite modifications are added to (and removed from) a protein through signaling networks, and the resulting combinatorial regulatory code on the protein is read to alter its behavior in the cell. (B) The pattern of some covalent modifications to the protein p53.



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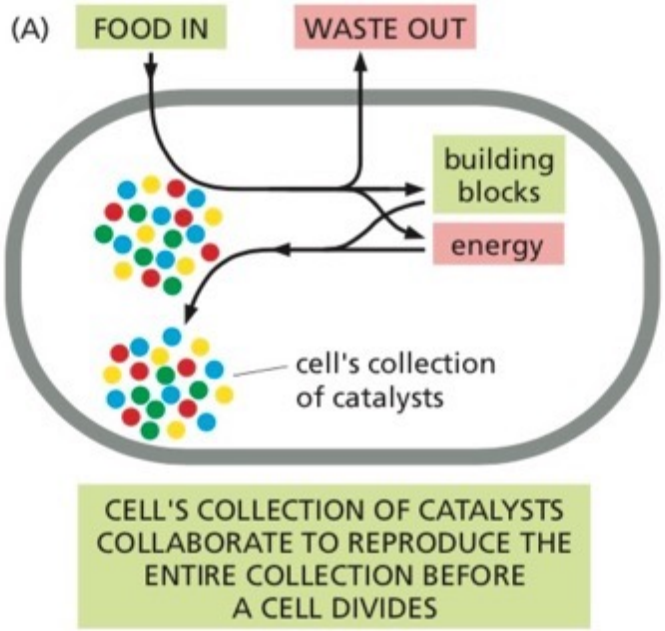

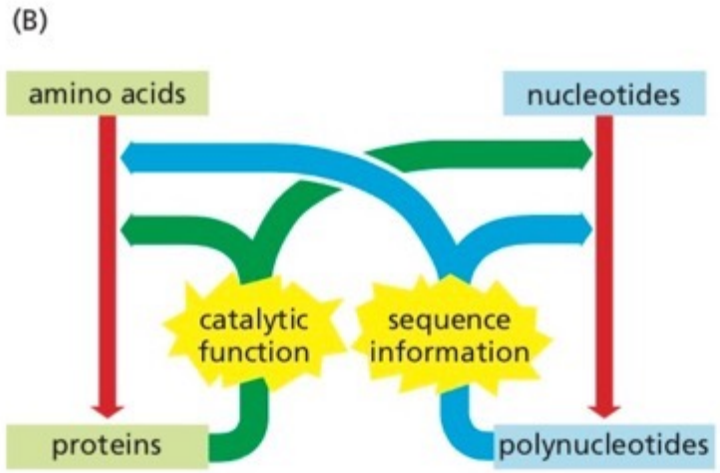

Figure 1–8 Life as an autocatalytic process. (A) The cell as a self-replicating collection of catalysts. (B) Polynucleotides (the nucleic acids DNA and RNA, which are nucleotide polymers) provide the sequence information, while proteins (amino acid polymers) provide most of the catalytic functions that serve—through a complex set of chemical reactions—to bring about the synthesis of more polynucleotides and proteins of the same types.

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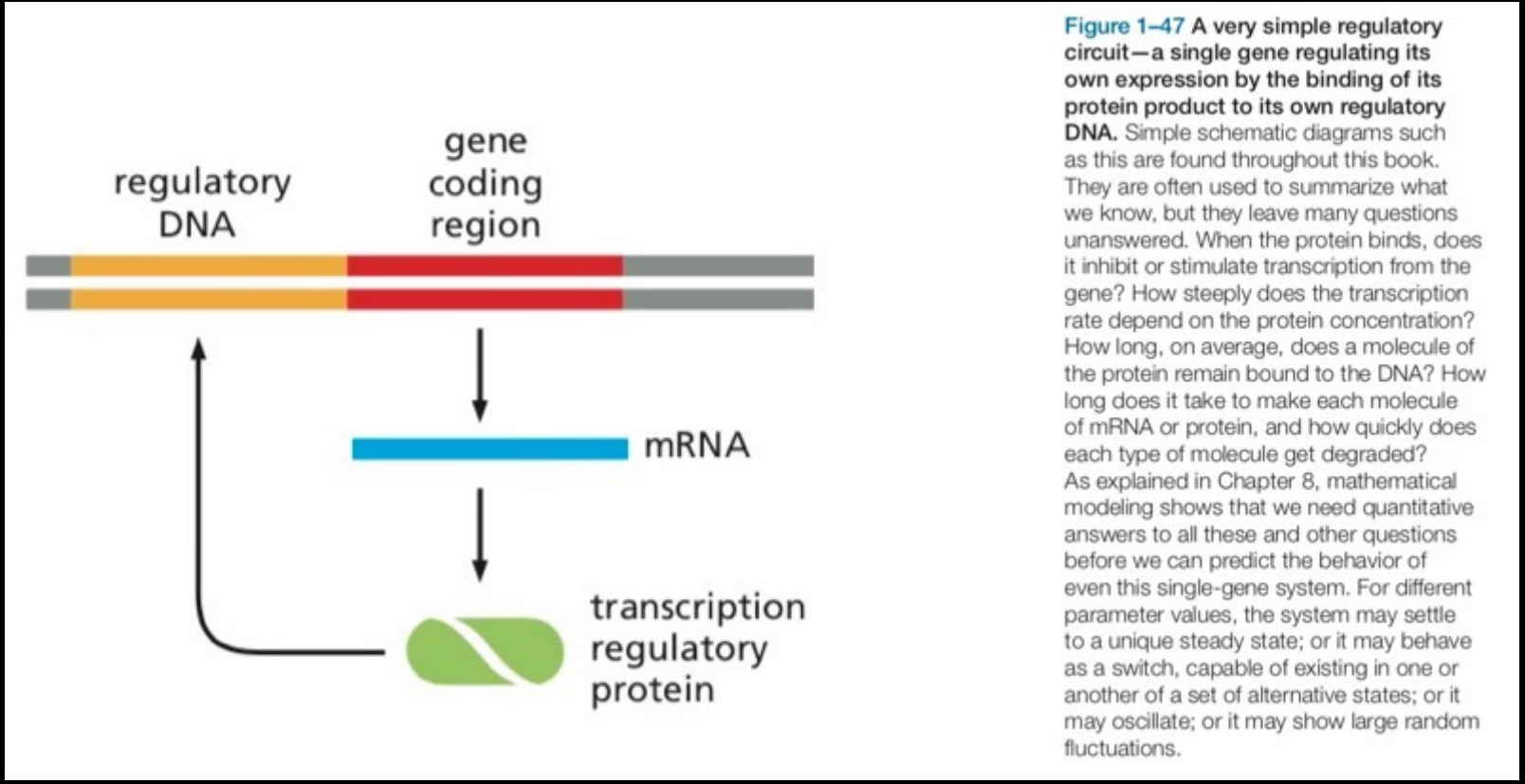

Figure 1–47 A very simple regulatory circuit—a single gene regulating its own expression by the binding of its protein product to its own regulatory DNA. Simple schematic diagrams such as this are found throughout this book. They are often used to summarize what we know, but they leave many questions unanswered. When the protein binds, does it inhibit or stimulate transcription from the gene? How steeply does the transcription rate depend on the protein concentration? How long, on average, does a molecule of the protein remain bound to the DNA? How long does it take to make each molecule of mRNA or protein, and how quickly does each type of molecule get degraded? As explained in Chapter 8, mathematical modeling shows that we need quantitative answers to all these and other questions before we can predict the behavior of even this single-gene system. For different parameter values, the system may settle to a unique steady state; or it may behave as a switch, capable of existing in one or another of a set of alternative states; or it may oscillate; or it may show large random fluctuations.

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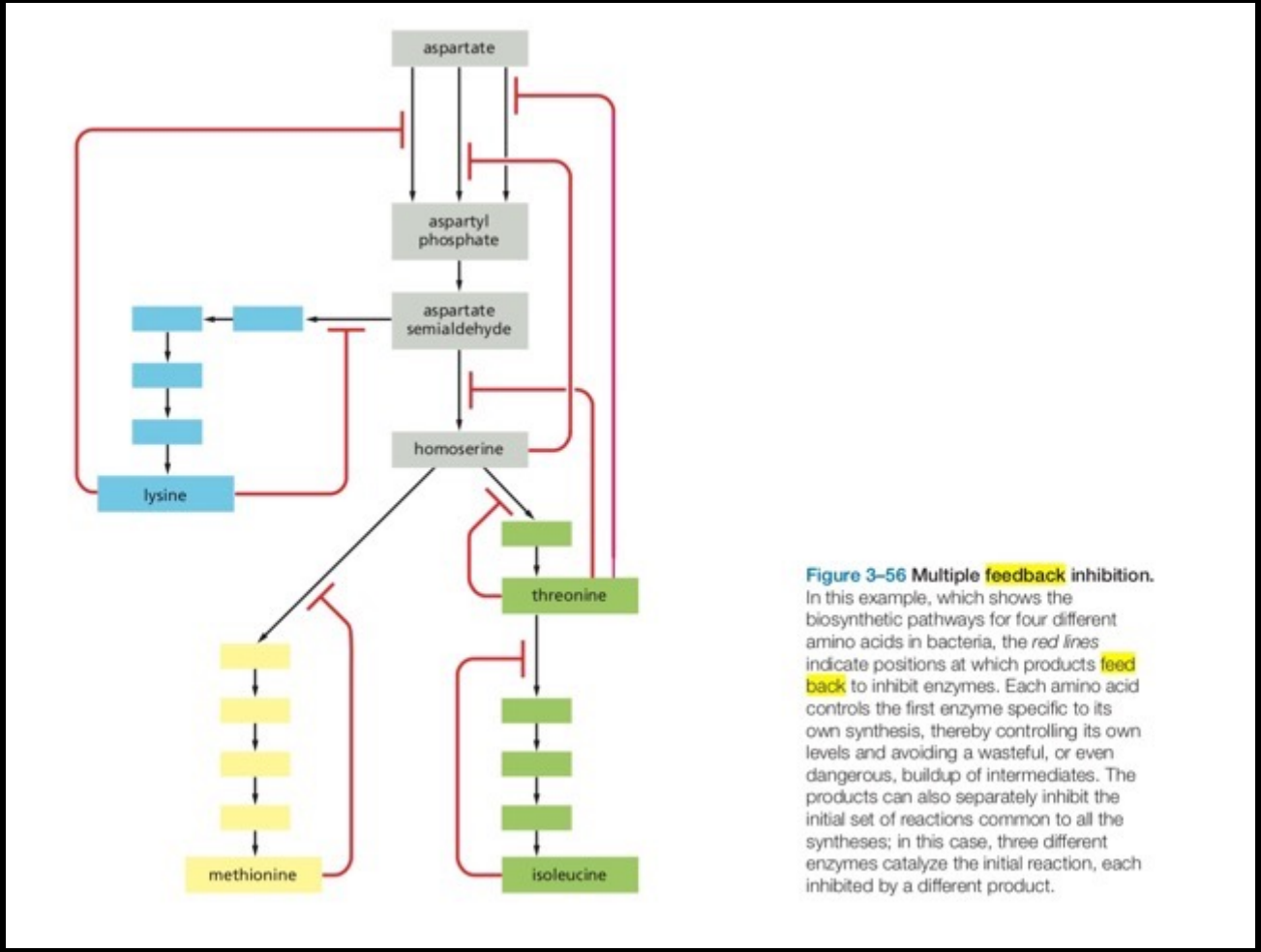

Figure 3–56 Multiple feedback inhibition. In this example, which shows the biosynthetic pathways for four different amino acids in bacteria, the *red lines* indicate positions at which products feed back to inhibit enzymes. Each amino acid controls the first enzyme specific to its own synthesis, thereby controlling its own levels and avoiding a wasteful, or even dangerous, buildup of intermediates. The products can also separately inhibit the initial set of reactions common to all the syntheses; in this case, three different enzymes catalyze the initial reaction, each inhibited by a different product.

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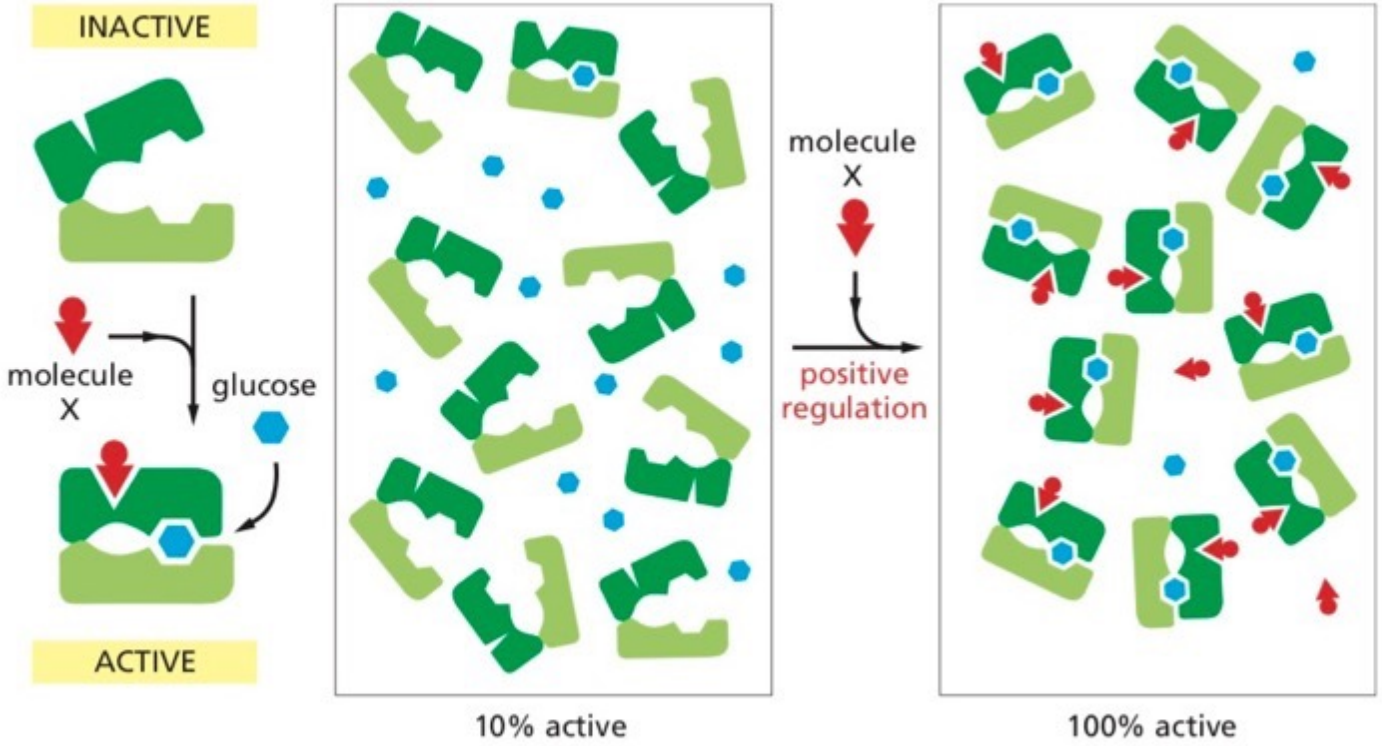
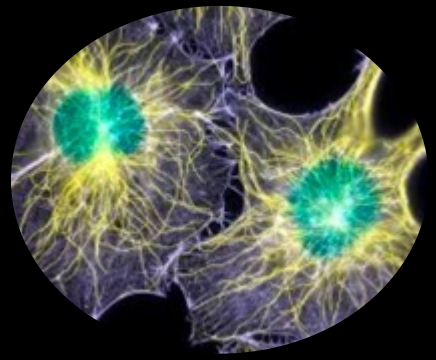


Figure 3–57 Positive regulation caused by conformational coupling between two separate binding sites. In this example, both glucose and molecule X bind best to the *closed* conformation of a protein with two domains. Because both glucose and molecule X drive the protein toward its closed conformation, each ligand helps the other to bind. Glucose and molecule X are therefore said to bind *cooperatively* to the protein.



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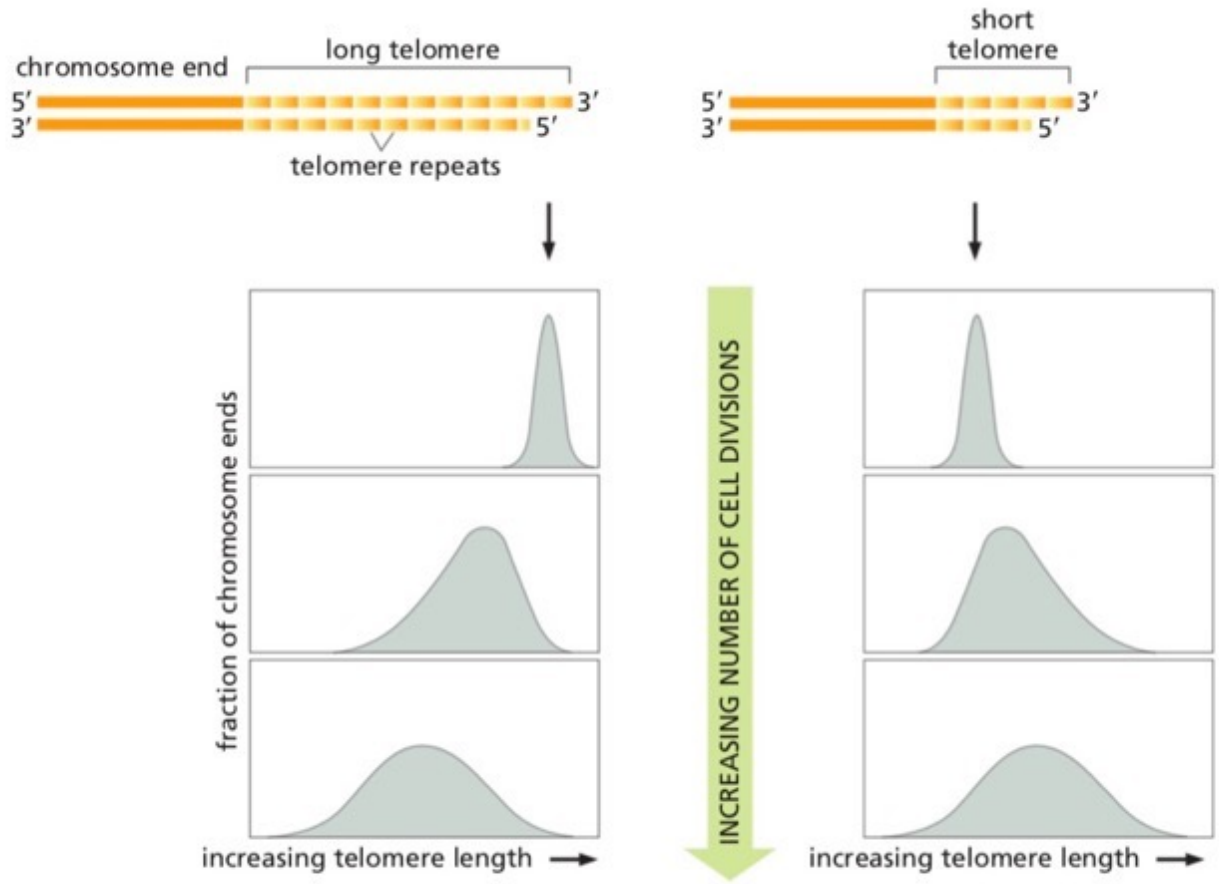

Figure 5–36 A demonstration that yeast cells control the length of their telomeres. In this experiment, the telomere at one end of a particular chromosome is artificially made either longer *(left)* or shorter *(right)* than average. After many cell divisions, the chromosome recovers, showing an average telomere length and a length distribution that is typical of the other chromosomes in the yeast cell. A similar feedback mechanism for controlling telomere length has been proposed for the germ-line cells of animals.

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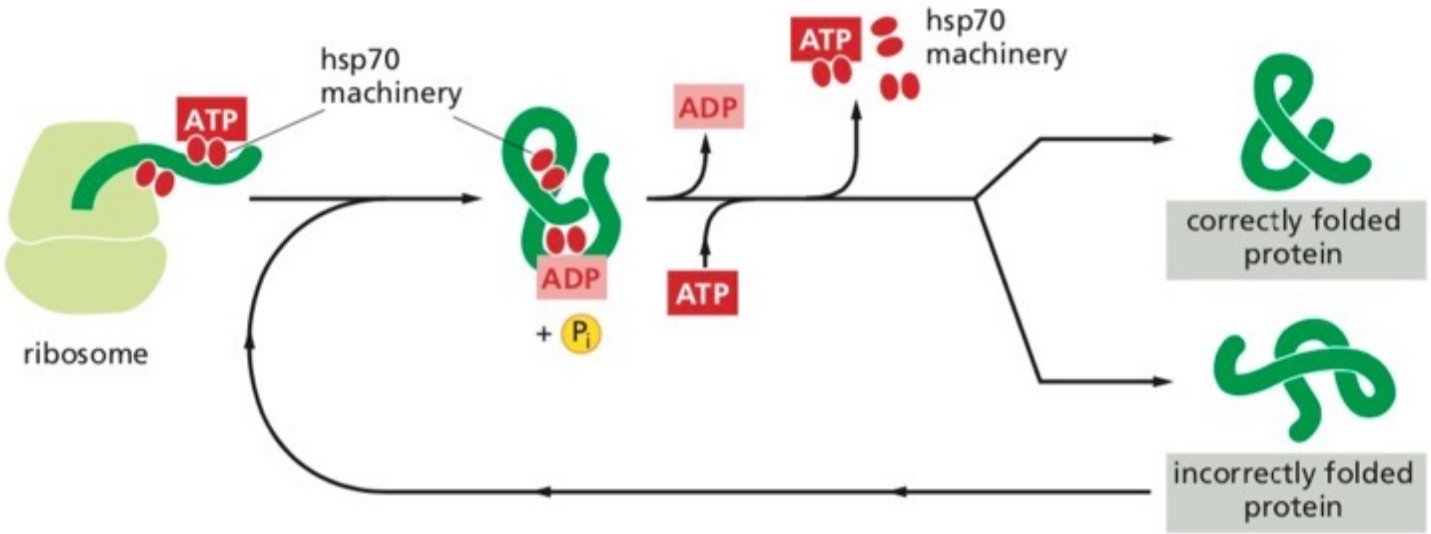

Figure 6–80 The hsp70 family of molecular chaperones. These proteins act early, recognizing a small stretch of hydrophobic amino acids on a protein's surface. Aided by a set of smaller hsp40 proteins (not shown), ATP-bound hsp70 molecules grasp their target protein and then hydrolyze ATP to ADP, undergoing conformational changes that cause the hsp70 molecules to associate even more tightly with the target. After the hsp40 dissociates, the rapid rebinding of ATP induces the dissociation of the hsp70 protein after ADP release. Repeated cycles of hsp binding and release help the target protein to refold.

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Homeostasis

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Current Opinion in Plant Biology

SnRK1 activation, signaling, and networking for energy homeostasis

Nathalie Crepin and Filip Rolland

The SnRK1 kinases are key regulators of the plant energy balance, but how their activity is regulated by metabolic status is still unclear. While the heterotrimeric kinase complex is well conserved among plants, fungi, and animals, plants appear to have modified its regulation to better fit their unique physiology and lifestyle. The SnRK1 kinases control metabolism, growth, and development, and stress tolerance by direct phosphorylation of metabolic enzymes and regulatory proteins and by extensive transcriptional regulation. Diverse types of transcription factors have already been implicated, with a well-studied role for the heterodimerizing group C and group S1 bZIPs. SnRK1 is also part of a more elaborate metabolic and stress signaling network, which includes the TOR kinase and the ABA-signaling SnRK2 kinases.

coordinate source, and sink activities, and undergo different developmental transitions that are associated with important changes in metabolic activities and requirements. Consistently, SnRK1 also controls many different aspects of plant development, from embryogenesis and germination to flowering and senescence [3,4]. Still, many questions remain about how SnRK1 controls and coordinates such diverse processes, and how exactly its activity is regulated in response to metabolic status. Here, we highlight some of the latest insight in both the upstream regulatory and downstream signaling mechanisms and discuss how SnRK1 functions in a more intricate network with Target of Rapamycin (TOR) and abscisic acid (ABA) signaling.

Bacterial cells can take up the amino acid tryptophan from their surroundings, or, if the external supply is insufficient, they can synthesize tryptophan from small molecules in the cell. The tryptophan repressor inhibits transcription of the genes in the tryptophan operon, which encodes the tryptophan biosynthetic enzymes. Upon binding tryptophan, the tryptophan repressor binds to a site in the promoter of the operon.

1 Why is tryptophan-dependent binding to the operon a useful property for the tryptophan repressor?

2 What would you expect to happen to the regulation of the tryptophan biosynthetic enzymes in cells that express a mutant form of the trypto- phan repressor that (i) cannot bind to DNA or (ii) binds to DNA even when no tryptophan is bound to it?

3 What would happen in scenarios (i) and (ii) if the cell produced normal tryptophan repressor from a second, unmutated copy of the gene?

Alberts, Bruce, author.
Molecular biology of the cell / Bruce Alberts, Alexander Johnson, Julian Lewis, David Morgan, Martin Raff, Keith Roberts, Peter Walter ; with problems by John Wilson, Tim Hunt. -- Sixth edition.

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Assunto: FCM2023-Nome do aluno

Homeostasis/Feedback Theoretical exercise 1