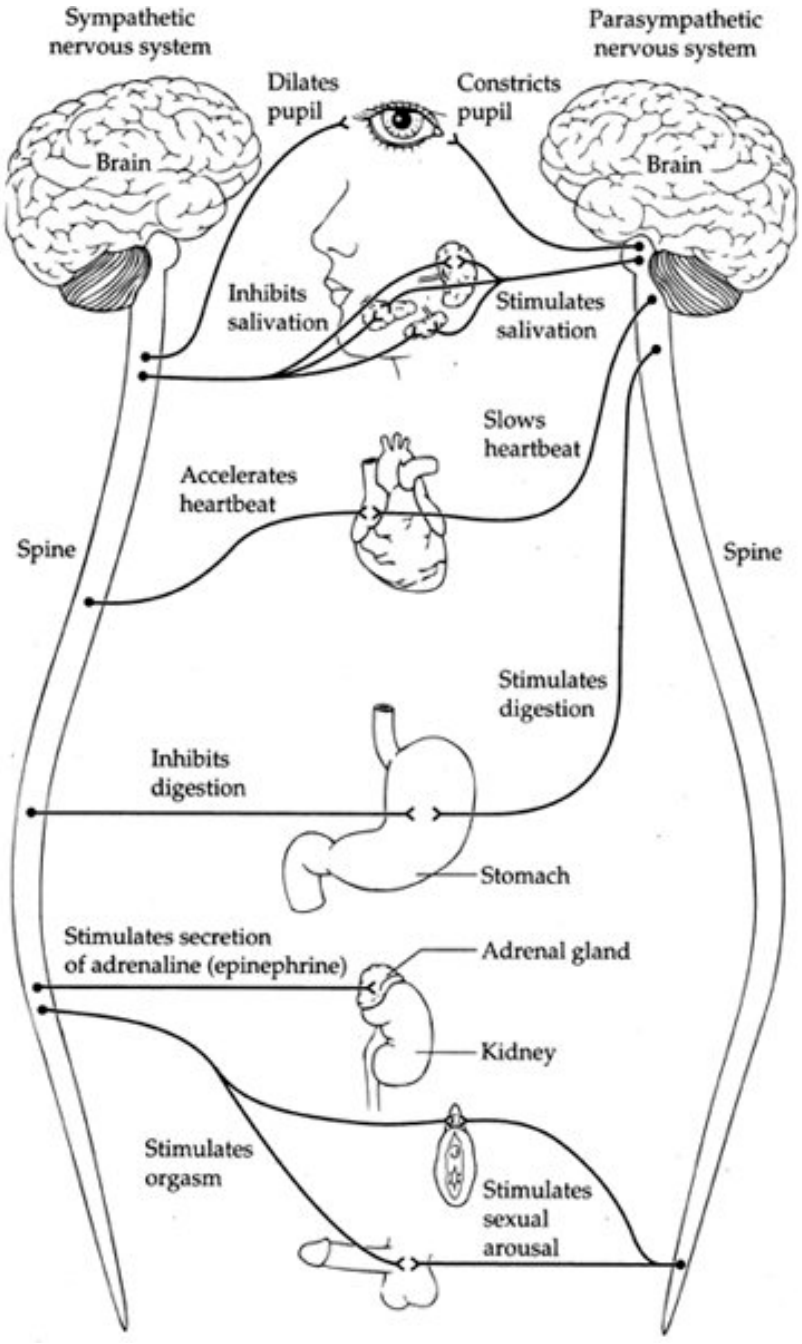
Why Zebras Don't Get Ulcers

Third Edition

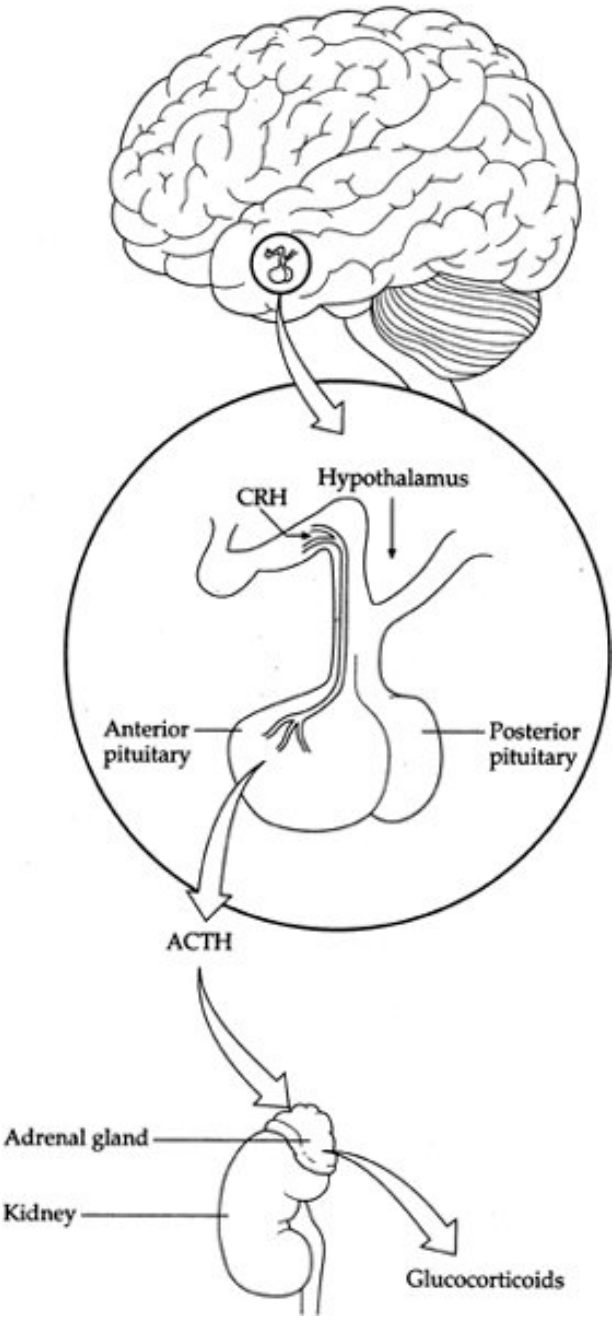
By Robert M. Sapolsky

Figure 1



Outline of some of the effects of the sympathetic and parasympathetic nervous systems on various organs and glands.

Figure 2



Outline of the control of glucocorticoid secretion. A stressor is sensed or anticipated in the brain, triggering the release of CRH (and related hormones) by the hypothalamus. These hormones enter the private circulatory system linking the hypothalamus and the anterior pituitary, causing the release of ACTH by the anterior pituitary. ACTH enters the general circulation and triggers the release of glucocorticoids by the adrenal gland.

Table 1

What you stick in your mouth	How it winds up in your bloodstream	How it gets stored if you have a surplus	How it gets mobilized in stressful emergency
Proteins →	Amino acids →	Protein →	Amino acids
Starch, sugars, carbohydrates →	Glucose →	Glycogen →	Glucose
Fat →	Fatty acids and glycerol →	Triglycerides →	Fatty acids, glycerol, ketone bodies

Figure 3

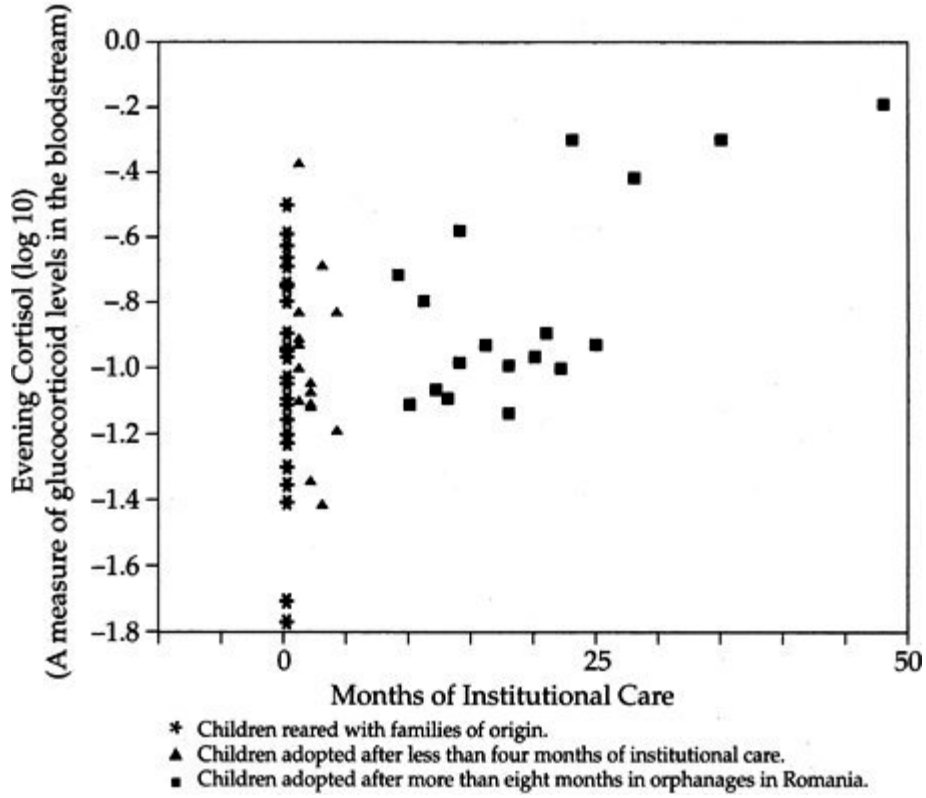
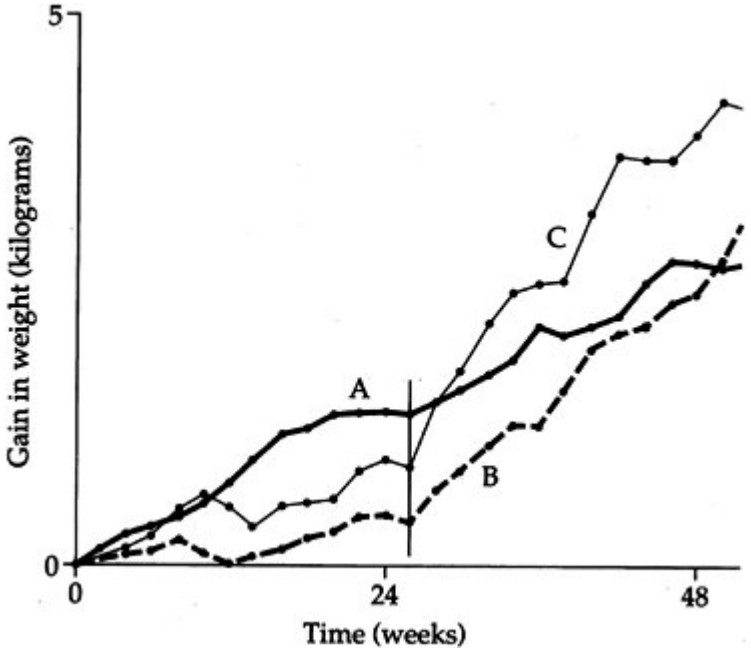
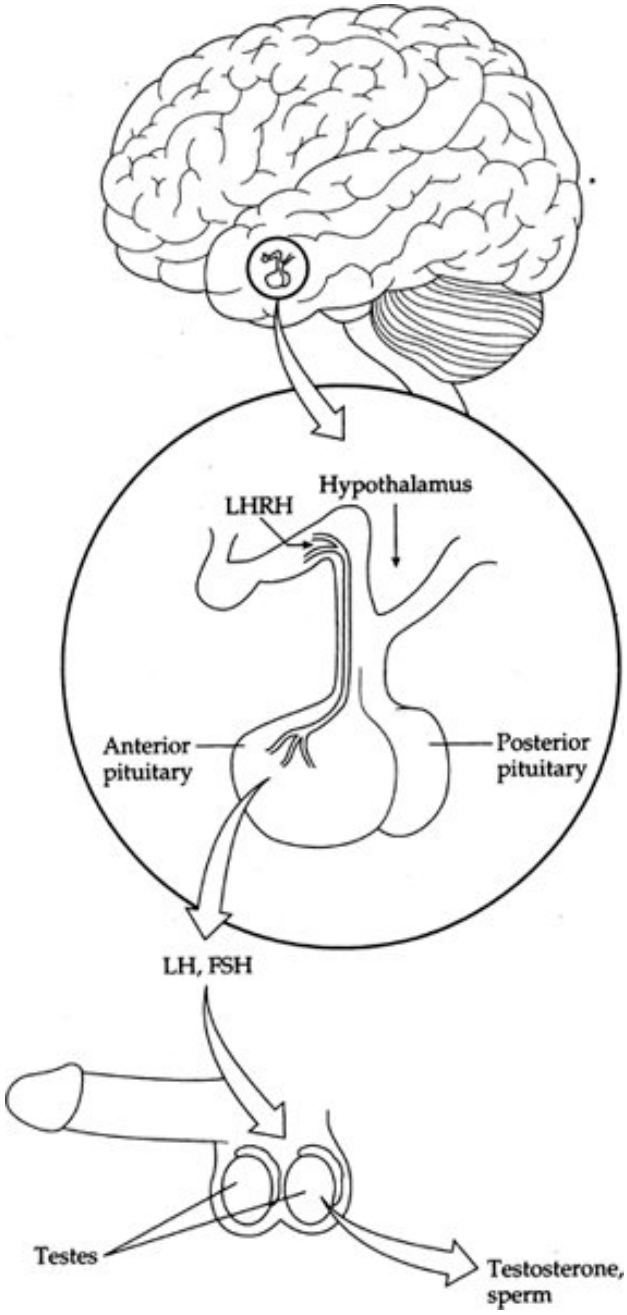

Evening Cortisol (log 10)
(A measure of glucocorticoid levels in the bloodstream)
0.0
-.2
-.4
-.6
-.8
-1.0
-1.2
-1.4
-1.6
-1.8
0
25
50
Months of Institutional Care
* Children reared with families of origin.
▲ Children adopted after less than four months of institutional care.
■ Children adopted after more than eight months in orphanages in Romania.

Figure 4



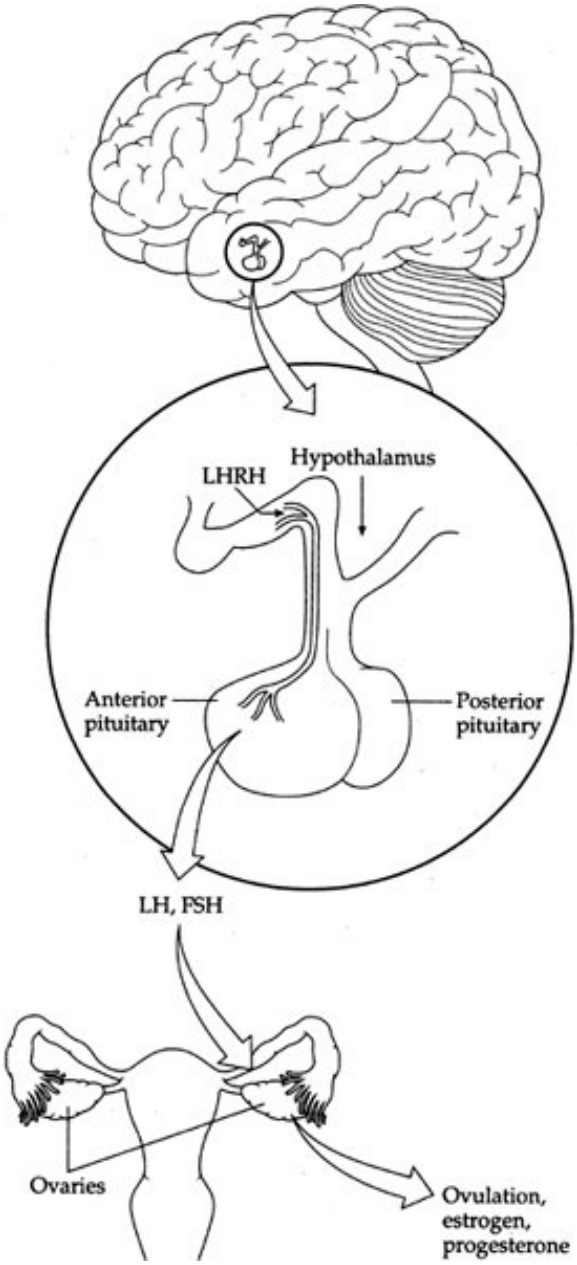
Growth rates in the two German orphanages. During the first 26 weeks of the study, growth rates in Orphanage A, under the administration of Fräulein Grun, were much greater than those in Orphanage B, with the stern Fräulein Schwarz. At 26 weeks (vertical line), Fräulein Grun left Orphanage A and was replaced by Fräulein Schwarz. The rate of growth in that orphanage promptly slowed; growth in Orphanage B, now minus the stern Fräulein Schwarz, accelerated and soon surpassed that of Orphanage A. A fascinating elaboration emerges from the fact that Schwarz was not completely heartless, but had a subset of children who were her favorites (Curve C), whom she had transferred with her.

Figure 5



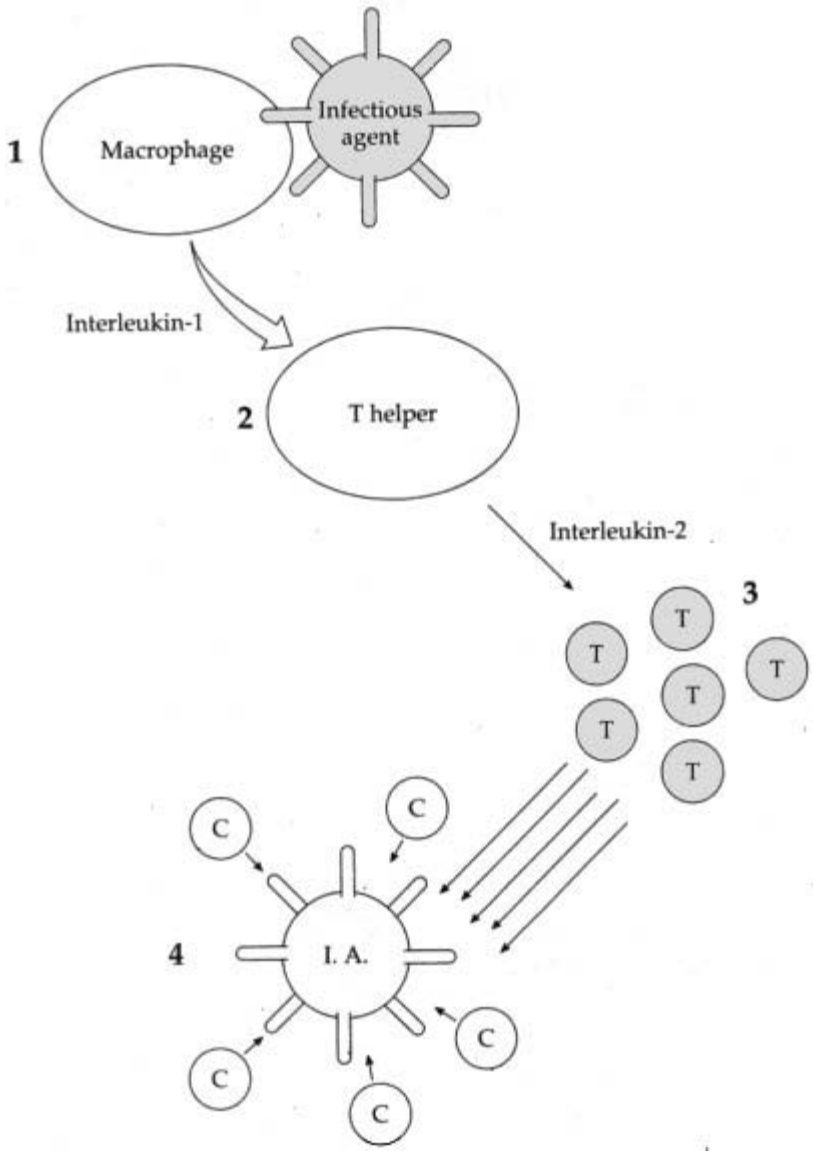
A simplified version of male reproductive endocrinology. The hypothalamus releases LHRH into the private circulatory system that it shares with the anterior pituitary. LHRH triggers the release by the pituitary of LH and FSH, which work at the testes to cause testosterone secretion and sperm production.

Figure 6



A simplified version of female reproductive endocrinology. The hypothalamus releases LHRH into the private circulatory system that it shares with the anterior pituitary. LHRH triggers the release by the pituitary of LH and FSH, which in turn bring about ovulation and hormone release from the ovaries.

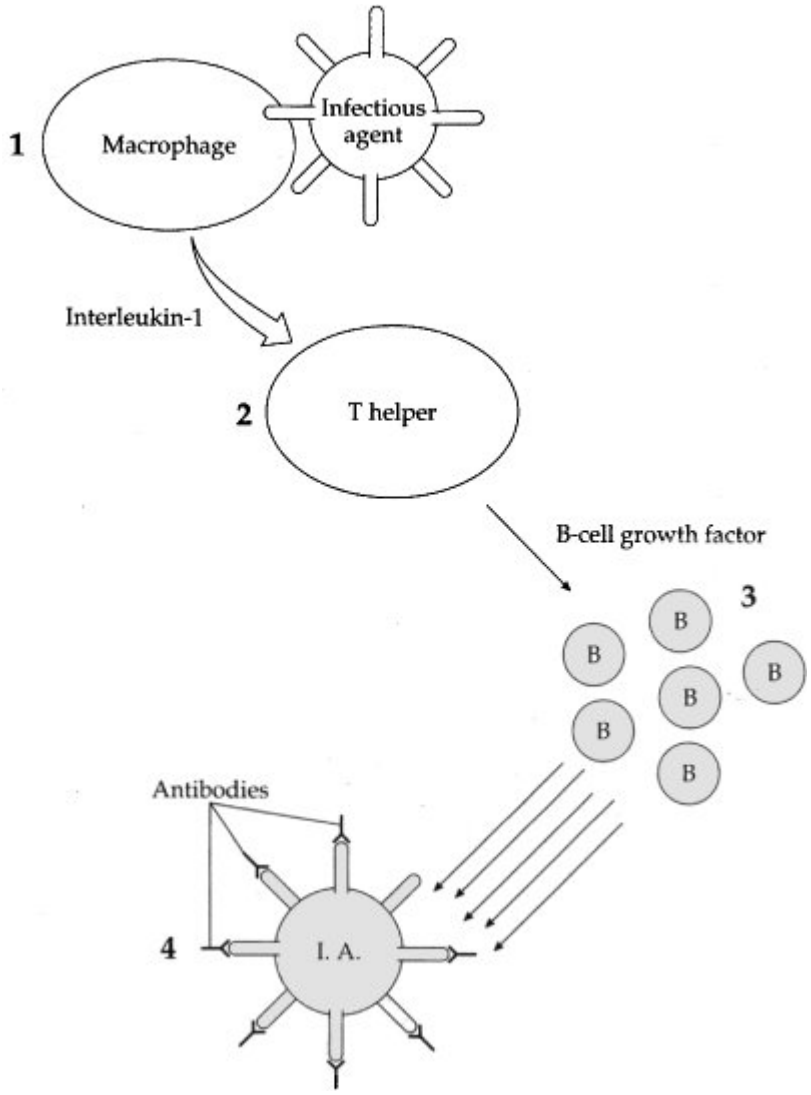
Figure 7



The cascade of cell-mediated immunity.

(1) An infectious agent is encountered by a type of monocyte called a macrophage.
(2) This stimulates the macrophage to present the infectious agent to a T helper cell (a type of white blood cell) and to release interleukin-1 (IL-1), which stimulates T helper cell activity.
(3) The T helper cell, as a result, releases interleukin-2 (IL-2), which triggers T-cell proliferation.
(4) This eventually causes another type of white blood cell, cytotoxic killer cells, to proliferate and destroy the infectious agent.

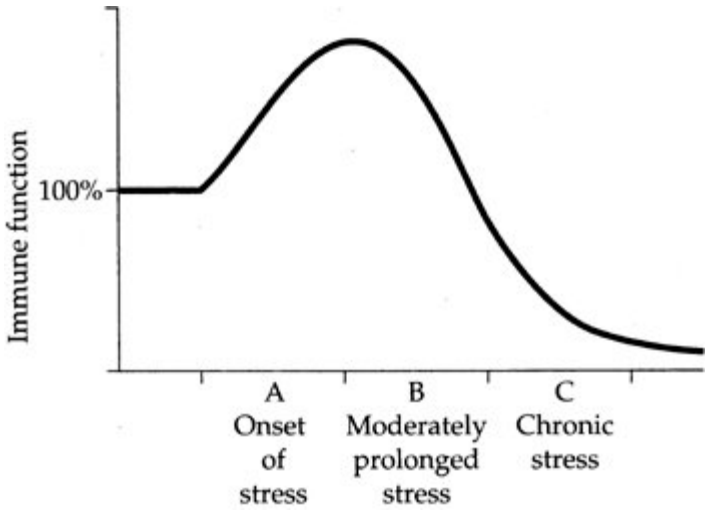
Figure 8



The cascade of antibody-mediated immunity.

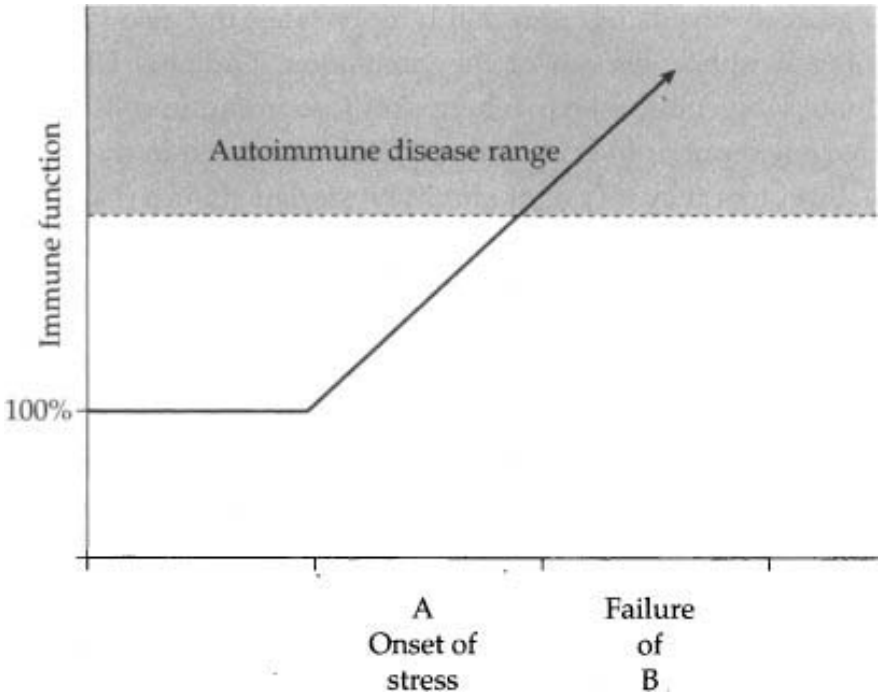
(1) An infectious agent is encountered by a macrophage.
(2) This encounter stimulates it to present the infectious agent to a T helper cell and to release interleukin-1 (IL-1), which stimulates T helper cell activity.
(3) The T helper cell then secretes B-cell growth factor, triggering differentiation and proliferation of another white blood cell, B cells.
(4) The B cells make and release specific antibodies that bind to surface proteins on the infectious agent, targeting it for destruction by a large group of circulating proteins known as complement.

Figure 9



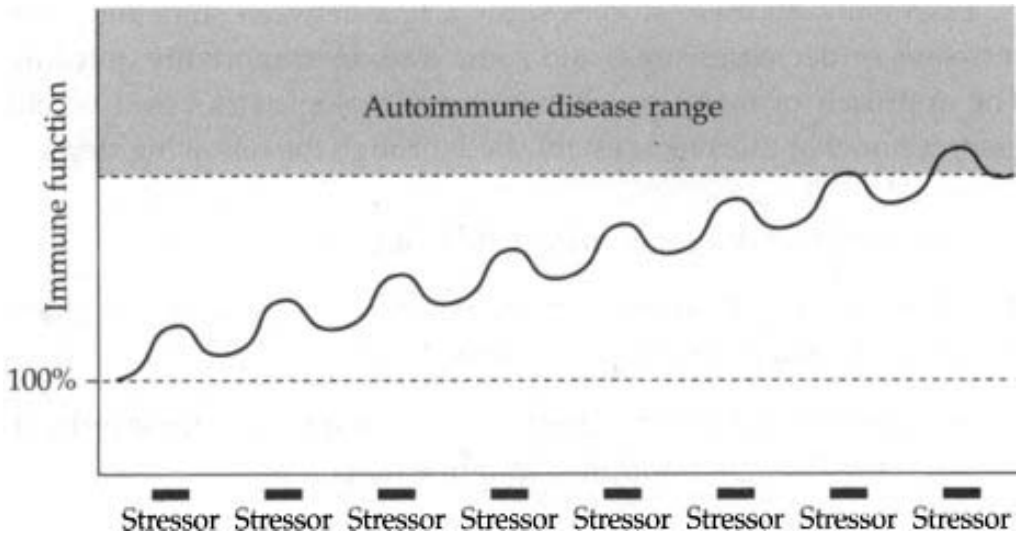
Stress turns out to transiently stimulate the immune system.

Figure 10



A schematic representation of how a failure to inhibit immune function during stress can bias you toward autoimmune disease.

Figure 11



A schematic representation of how repeated stress increases the risk of autoimmune disease.

Figure 12

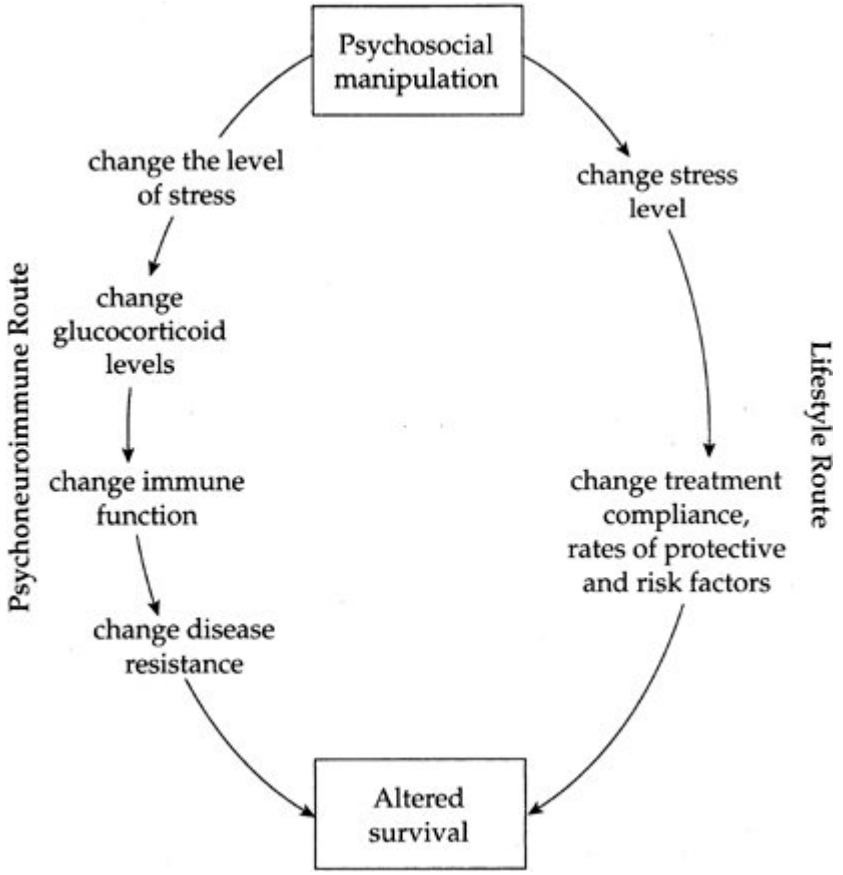
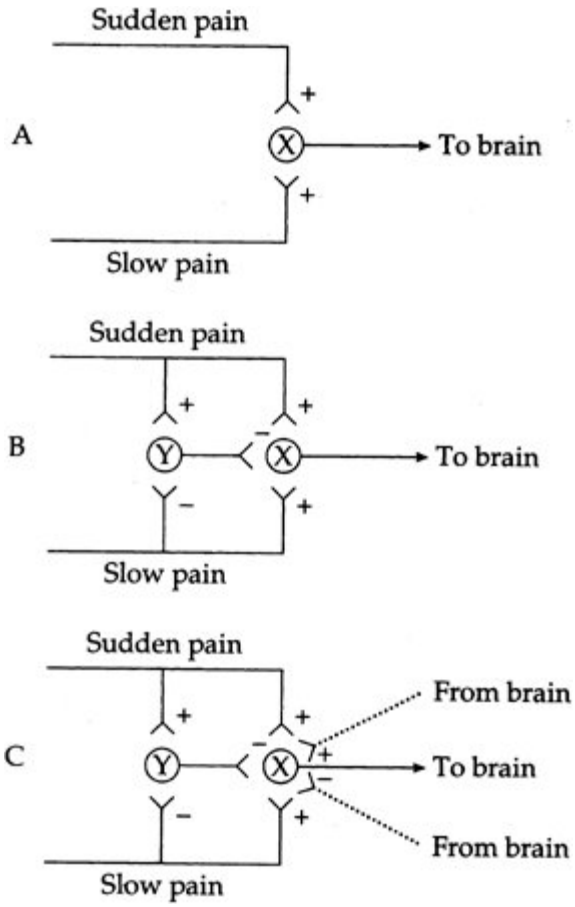

Psychosocial manipulation
change the level of stress
change stress level
change glucocorticoid levels
change immune function
change disease resistance
change treatment compliance, rates of protective and risk factors
Altered survival
Psychoneuroimmune Route
Lifestyle Route

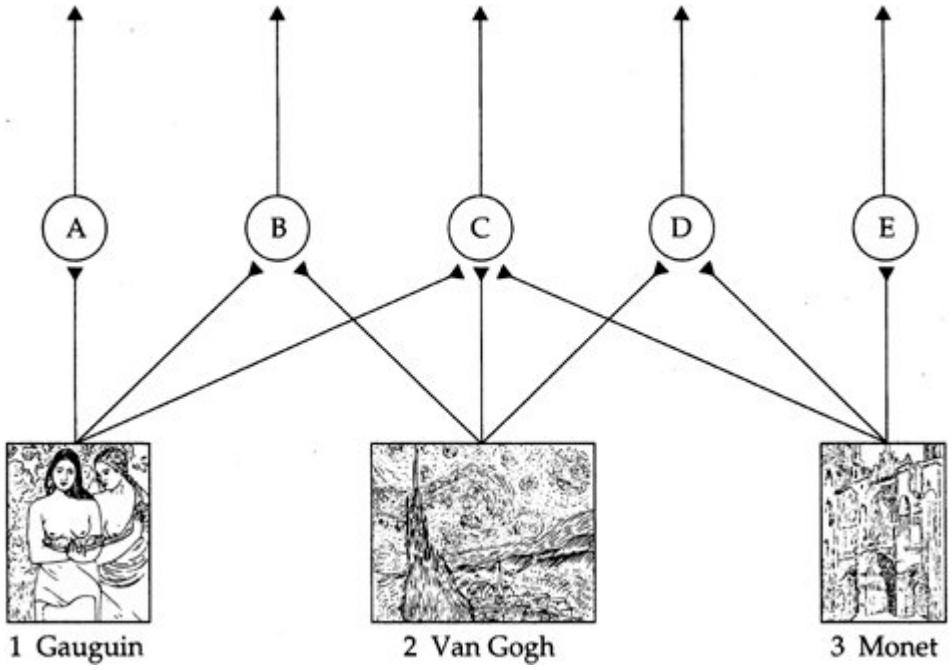
Figure 13



The Wall-Melzack model of how pain information is passed to the brain, and how it can be modulated by the brain.

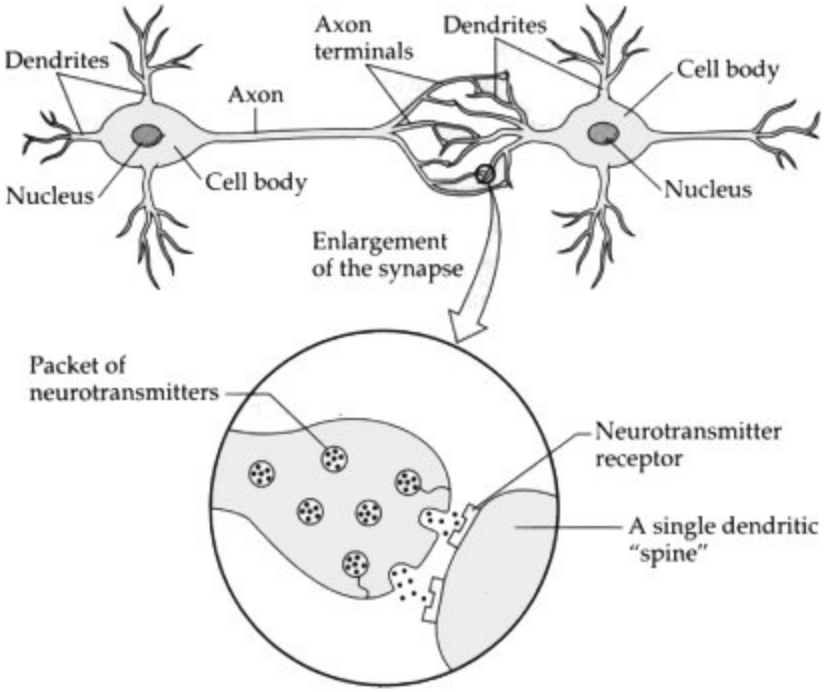
(A) A neuron (X) in the spinal cord sends a signal to the brain that something painful has happened, once it is stimulated by a pain fiber. Such pain fibers can carry information about sudden pain or slow, diffuse pain.
(B) A more realistic version of how the system actually works, showing why sudden and slow pain information is differentiated. In the case of sudden pain, the sudden pain fiber stimulates neuron X, causing a pain signal to be relayed to the brain. The sudden pain fiber also stimulates an interneuron (Y) that inhibits neuron X, after a brief delay. Thus, neuron X sends a pain signal to the brain for only a short time. In contrast, the slow pain fiber stimulates neuron X and inhibits interneuron Y. Thus, Y does not inhibit X, and X continues to send a pain signal to the brain, producing a slow, diffuse pain.
(C) Both stimulatory and inhibitory fibers come from the brain and send information to neuron X, modulating its sensitivity to incoming pain information. Thus, the brain can sensitize neuron X to a painful signal, or blunt its sensitivity.

Figure 14



A highly hypothetical neural network involving a neuron that "knows" about Impressionist paintings.

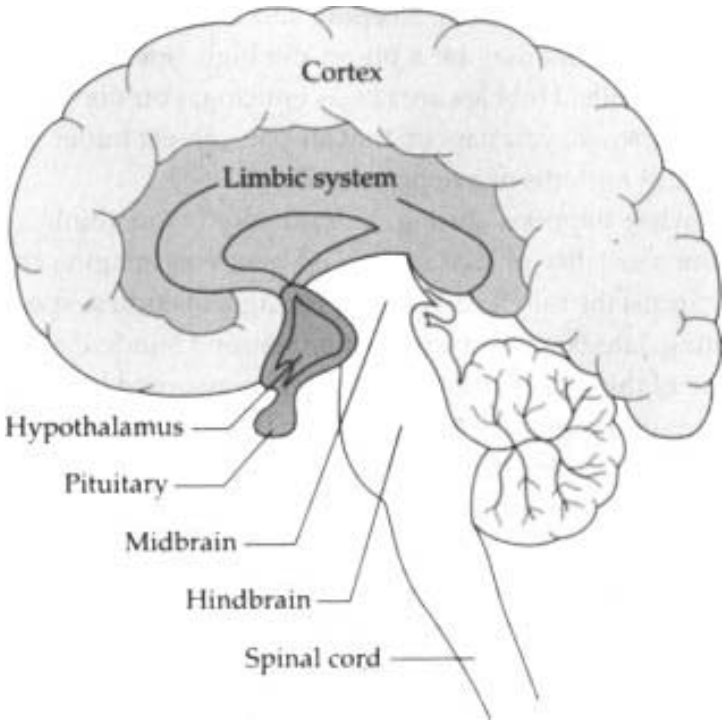
Figure 15



A single axon terminal.

A neuron that has been excited conveys information to other neurons by means of chemical signals at synapses, the contact points between neurons. When the impulse reaches the axon terminal of the signaling neuron, it induces the release of neurotransmitter molecules. Transmitters diffuse across a narrow cleft and bind to receptors in the adjacent neuron's dendritic spine.

Figure 16



The triune brain.

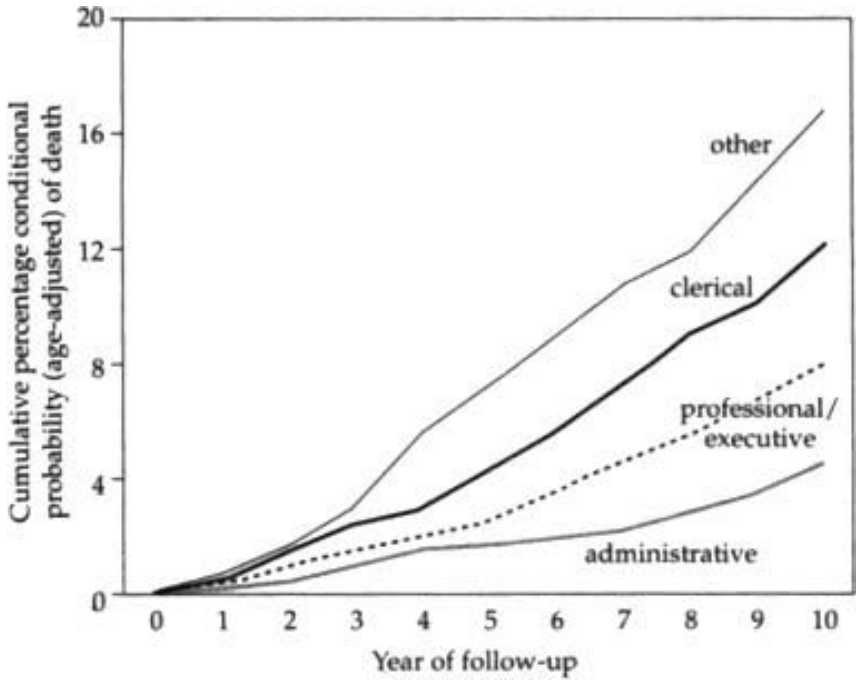
Figure 17

"Gary."

Figure 18

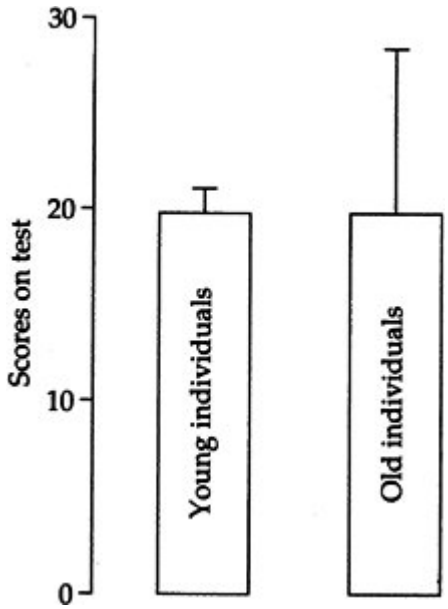
"Kenneth" (with infant).

Figure 19



The Whitehall Study, Mortality by Professional Level of Follow-up.

Figure 20



Schematic presentation of the fact that a group of young and old individuals may receive the same average score on a given test, yet the variability in the scores is typically greater among the older populations.