

Environmental Pathology And Nutritional Disease



Modes of Environmental Exposure



It's drug addiction.



Labor Day

From International Labor Organization :

Every 15 seconds:

A worker dies from work related accident or disease

160 workers have a work-related accident

6300 deaths per day from occupational accident or work-related disease

2.3 million deaths/year

317 million accidents on the job per year

Environmental Pathology

Effects of Environmental Agents:

Inflammation: acute and chronic
granulomatous

Hypersensitivity: immune-mediated

Necrosis

Neoplasia: initiators and promoters

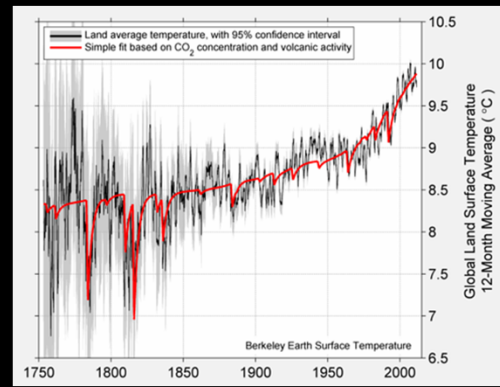
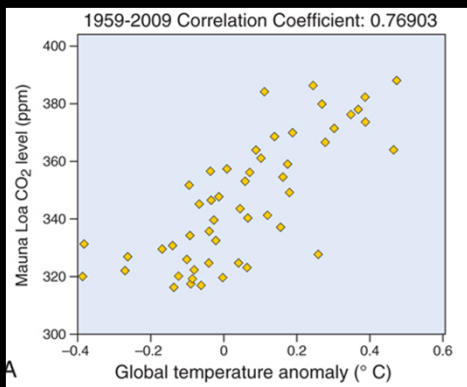
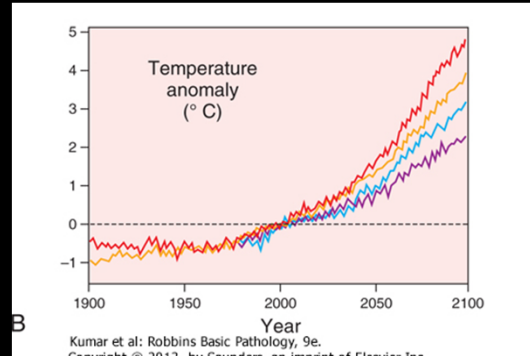
Environmental and Nutritional Diseases

Part 1:

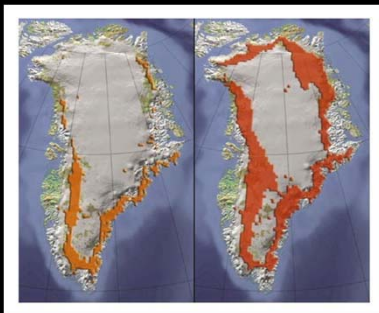
Climate Change

Physical injury

Nutritional Disease

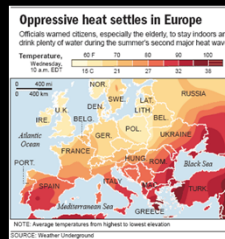


Increase from 1992 (Left) to 2002 (Right) in the Amount of the Greenland Ice Sheet Melted in the Summer.

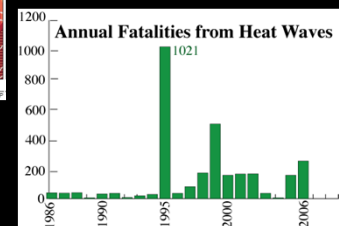


Epstein PR. N Engl J Med 2005;353:1433-1436.

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Heat Waves



Climate Change and Infectious Disease



Climate Change and Diarrheal Illness

Haiti 2010



Numbers of Disability-Adjusted Life-Years Due to Causes That Are Attributable to Climate Change, as of 2000.

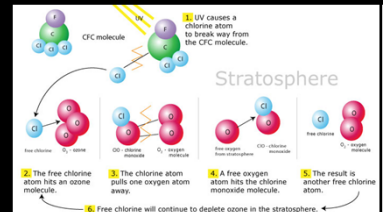
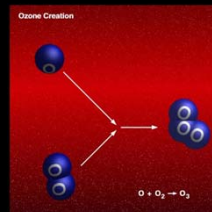
Region	Total DALYs	DALYs/Million Population
Africa	1,894,000	3071.5
Eastern Mediterranean	768,000	1586.5
Latin America and Caribbean	121,000	188.5
Southeast Asia	2,572,000	1703.5
Western Pacific	169,000	111.4
Developed countries	8,000	8.9

* Disability-adjusted life-years (DALYs) are life-years that are lost owing to disability or premature death. Causes that may be attributable to climate change include diarrheal disease, vectorborne disease, malnutrition, and injury from natural disasters. Data are from the World Health Organization.*

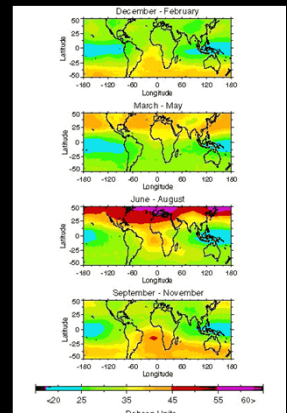
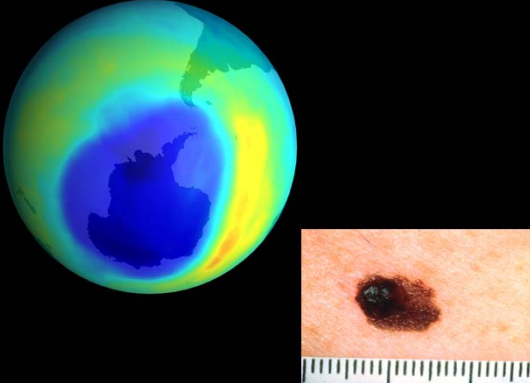
Shuman EK. N Engl J Med 2010;362:1061-1063.

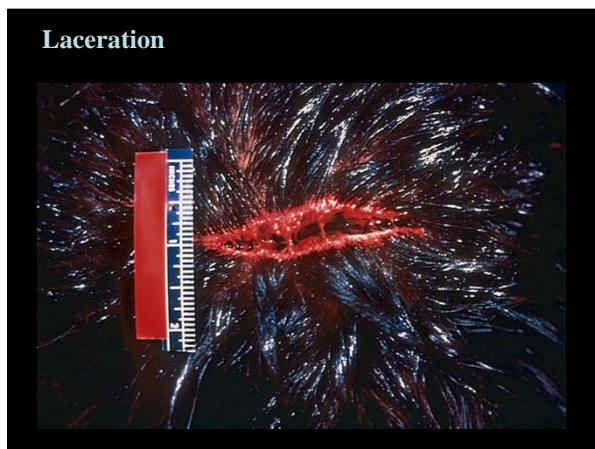
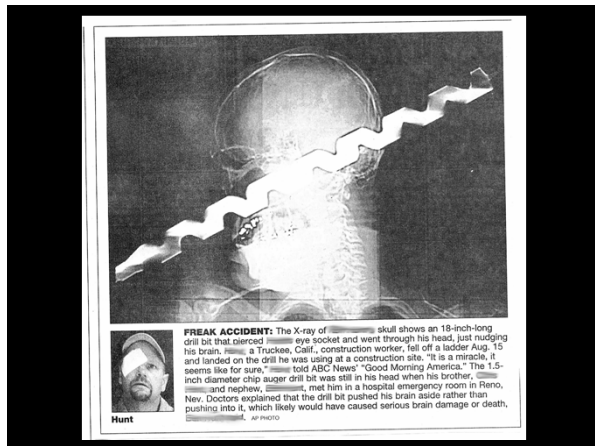
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Ozone



Ozone • September 6, 2000 • Total Ozone Mapping Spectrometer (TOMS)

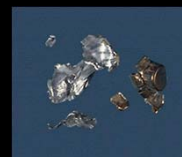
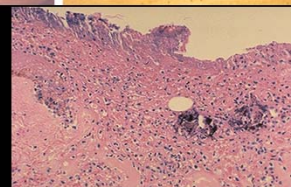
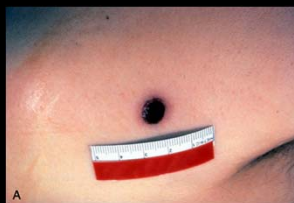




Stab and Incised Wounds

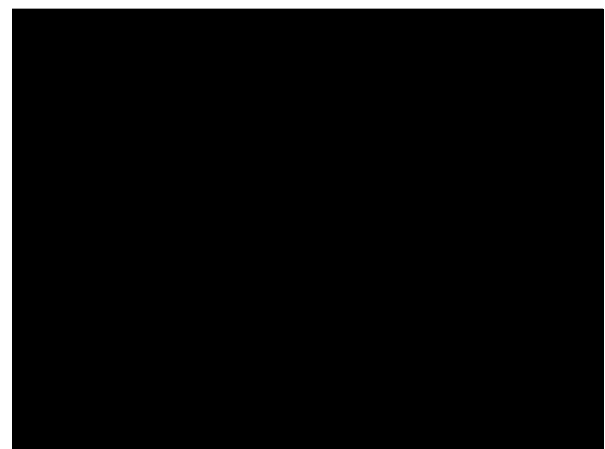
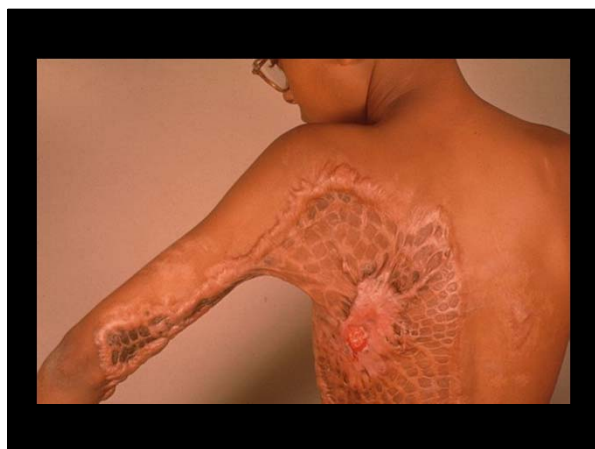
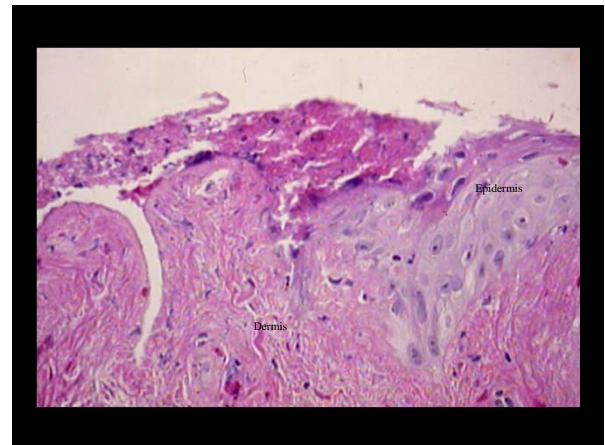
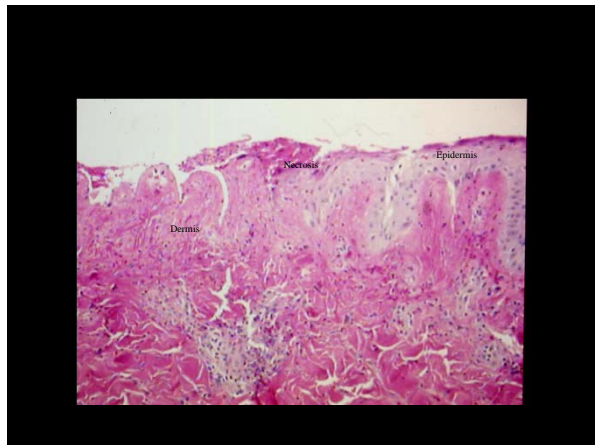
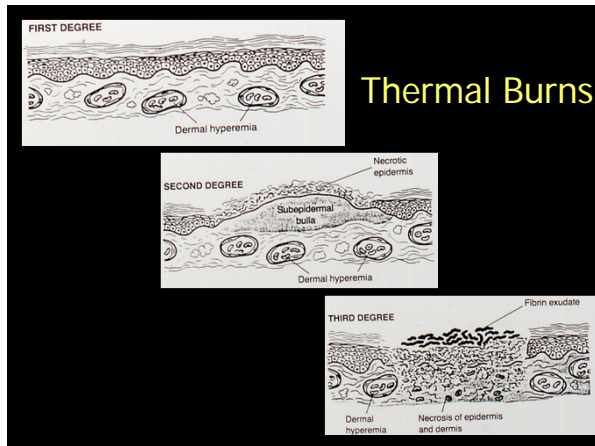


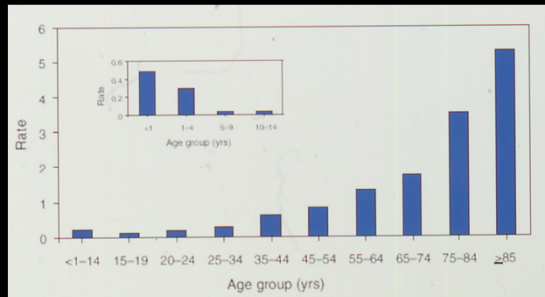
Gunshot Wounds: Entrance



Gunshot Wound: Contact Range

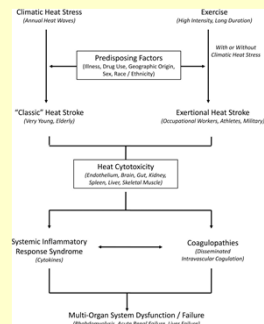






Mortality from heat related illness due to excessive natural heat by age US 1979-1999
Rate per million population

Summary of environmental and predisposing factors that interact to cause "classic" (passive) and exertional heat stroke.

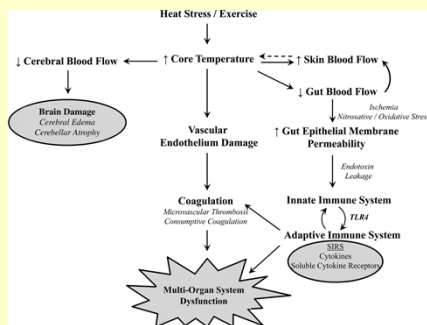


Leon L R, Helwig B G J Appl Physiol 2010;109:1980-1988

Journal of Applied Physiology

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Summary of heat stroke pathophysiological changes that culminate in multi-organ system dysfunction and death.



Leon L R, Helwig B G J Appl Physiol 2010;109:1980-1988

Journal of Applied Physiology

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Hypothermia



Risk Factors for Decreased Thermostability.

Decreased heat production	Increased heat loss
Endocrinologic failure Hypothyroidism Hypoparathyroidism Hypoadrenalism Insufficient fuel Hypoglycemia Malnutrition Extreme physical exertion Neuromuscular physical exertion Age extremes Impaired shivering Inactivity Lack of adaptation	Induced vasodilation Pharmacologic Toxin-induced Dermatologic causes Burns Dermatitis Iatrogenic causes Exposure Cold infusions Emergency deliveries Environmental causes Immersion Nonimmersion
Impaired thermoregulation	Miscellaneous associated clinical states
Peripheral failure Neuropathies Acute spinal cord transection Diabetes Central nervous system failure or neurologic abnormalities Pharmacologic causes Metabolic causes Toxins Cerebrovascular accident Central nervous system trauma Neoplasm Degenerative disease	Multi-system trauma Shock Cardiopulmonary disease Systemic acidosis Infections — bacterial, viral, parasitic Cardiomyopathy Vascular insufficiency Pneumonia Uremia Recurrent or episodic hypothermia

Danzel DF, Pozos RS. N Engl J Med 1994;331:1756-1760.

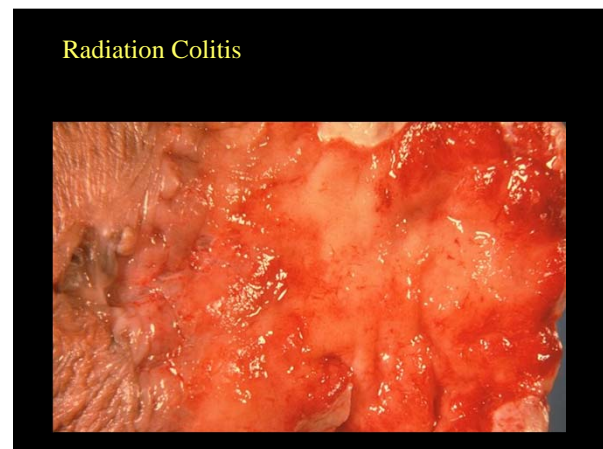
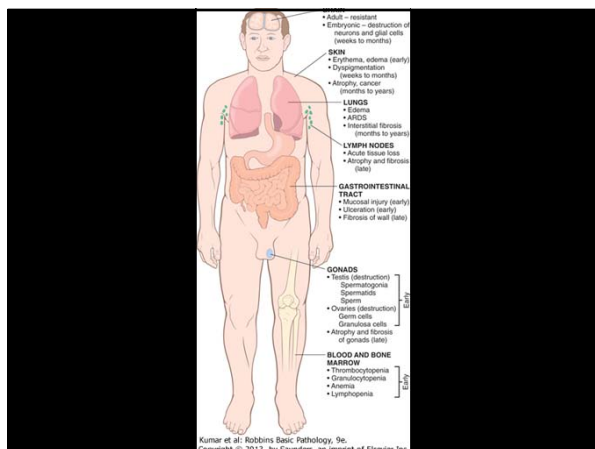
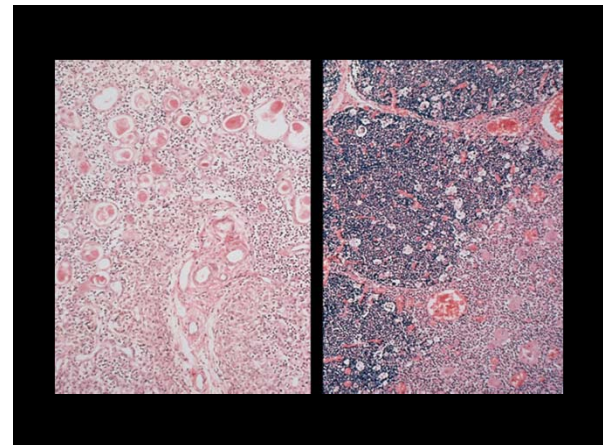
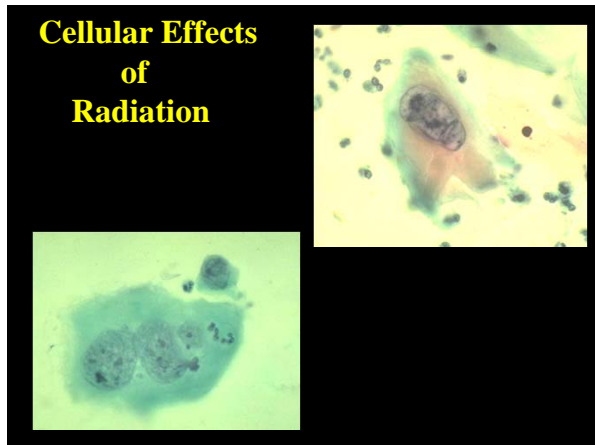
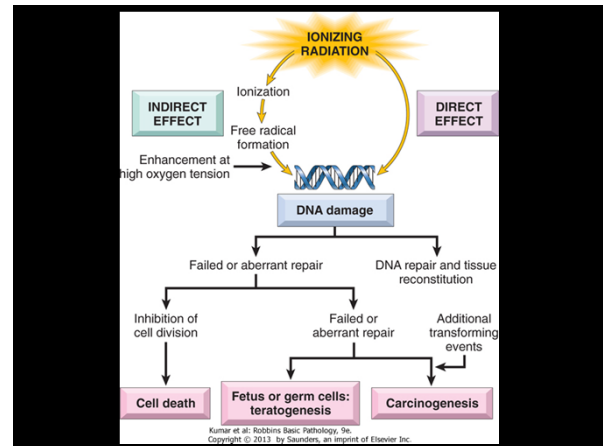
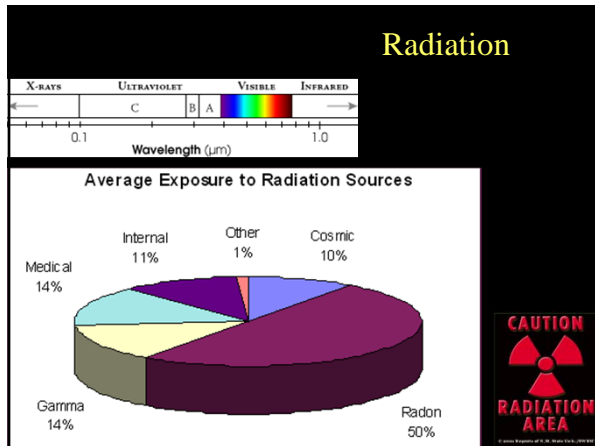
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Physiologic Changes Associated with Hypothermia.

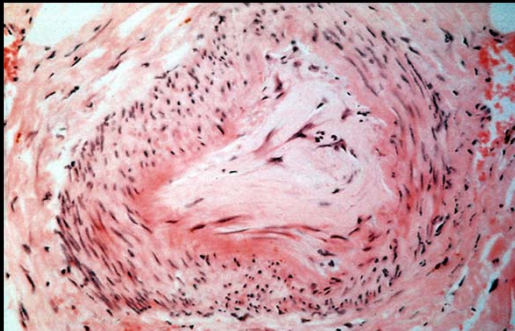
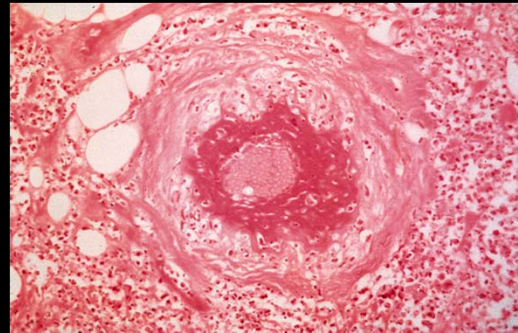
SEVERITY OF HYPOTHERMIA	BODY TEMPERATURE	CENTRAL NERVOUS SYSTEM	CARDIOVASCULAR	RESPIRATORY	RENAL AND ENDOCRINE	NEUROMUSCULAR
Mild	35°C (95°F) to 32.2°C (90°F)	Linear depression of cerebral metabolism; anesthetic quality; dysarthria; impaired judgment; maladaptive behavior	Tachycardia, then progressive bradycardia; cardiac-cycle prolongation; vasoconstriction; increase in cardiac output and blood pressure	Tachypnea, then progressive decrease in respiratory minute volume; declining oxygen consumption; bronchospasm	Cold diuresis; increase in catecholamines; adrenal steroids; mineralocorticoids; and thyroxine; increase in metabolism with shivering	Increased shivering; muscle tone; then fatiguing shivering; induced thermogenesis; ataxia
Moderate	<32.2°C (90°F) to 28°C (82.4°F)	Electroencephalographic abnormalities; progressive depression of level of consciousness; pupillary dilatation; paradoxical undressing; hallucinations	Progressive decrease in pulse and cardiac output; increased atrial and ventricular arrhythmias; nonspecific and suggestive (J wave) electrocardiographic changes; prolonged systole	Hypoventilation; 50% decrease in carbon dioxide production per 8°C drop in temperature; absence of protective airway reflexes; 50% decrease in oxygen consumption	50% increase in renal blood flow; renal autoregulation intact; no insulin activity	Hyporeflexia; diminishing shivering; induced thermogenesis; rigidity
Severe	<28°C (82.4°F)	Loss of cerebrovascular autoregulation; decline in central blood flow; coma; loss of ocular reflexes; progressive decrease in electroencephalographic activity	Progressive decreases in blood pressure, heart rate, and cardiac output; moribund dysrhythmias; decreased ventricular arrhythmia threshold; asystole	Pulmonary congestion and edema; 75% decrease in oxygen consumption; apnea	Decrease in renal blood flow parallels decrease in cardiac output; extreme oliguria; prothrombosis; 80% decrease in basal metabolism	No motion; decreased nerve-conduction velocity; peripheral anesthesia

Danzel DF, Pozos RS. N Engl J Med 1994;331:1756-1760.

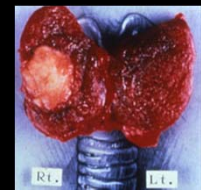
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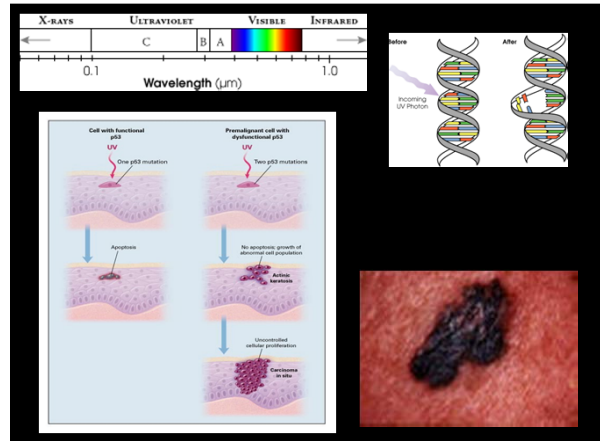
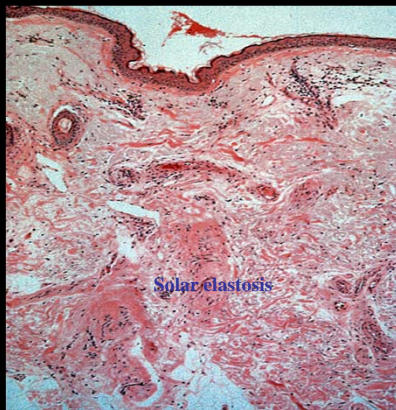
Radiation Dermatitis

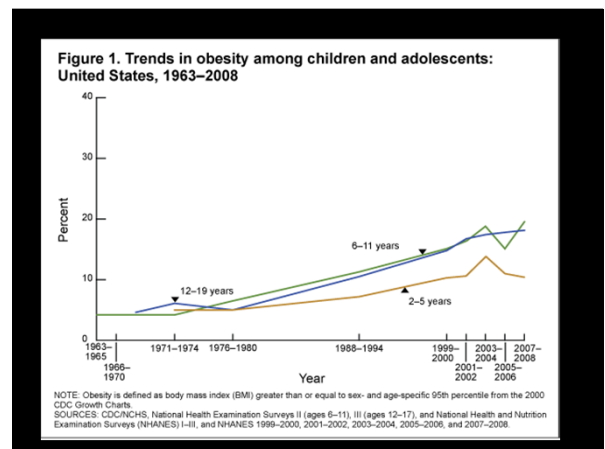
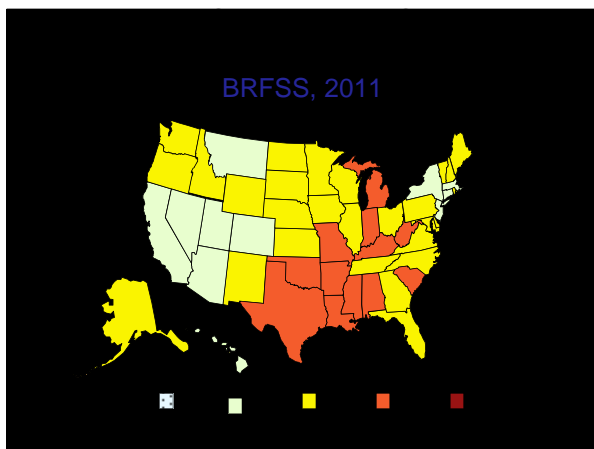
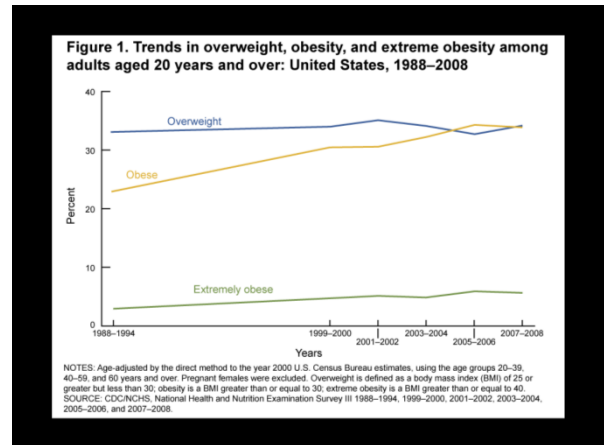
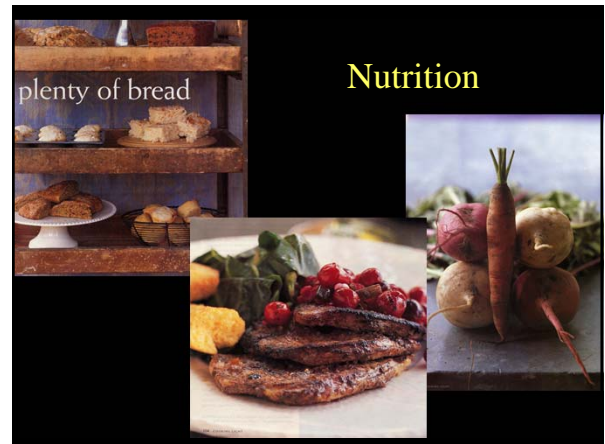
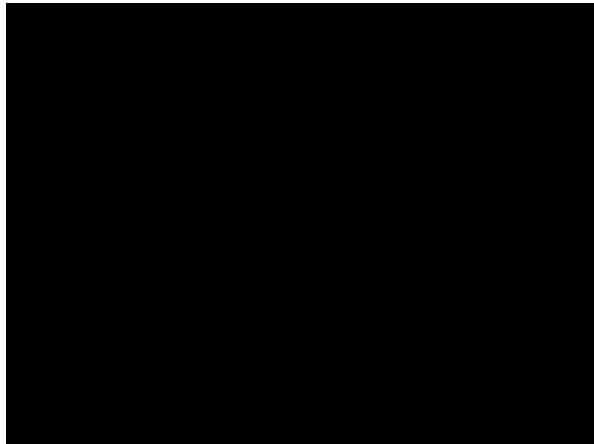


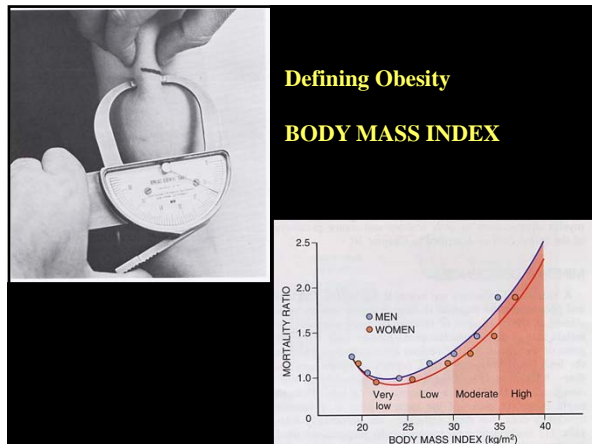
Neoplastic Complications of Radiation



Effects of UV radiation







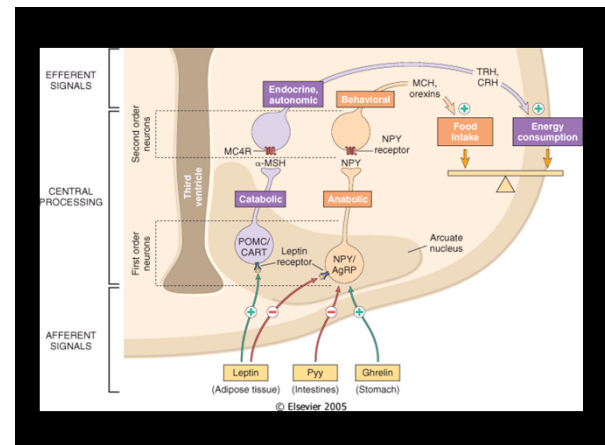
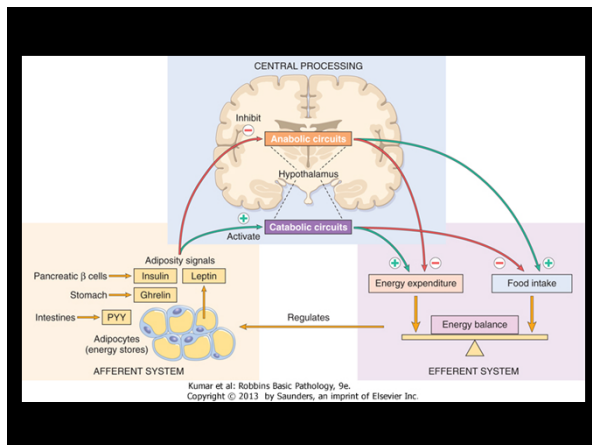
TYPES OF OBESITY - Are you an Apple or a Pear ??

Apple/Android

- Excess Fat on the Abdomen
- Common in Men
- Significant correlation with Metabolic Syndrome.

Pear/Gynoid

- Excess Fat on the thighs and buttocks
- Common in Women
- Non significant correlation with Metabolic Syndrome.



Study: Hormone that curbs appetite could treat obesity

Associated Press

Giving overweight people an extra dose of a hormone that tells the brain when to stop eating appears to curb their appetites by nearly a third, pointing to a potential new treatment for obesity.

The researchers at Imperial College London had previously shown that the hormone could suppress the appetites of lean people, but they worried it would not work in obese people because studies of another appetite-suppressing hormone, leptin, had proved disappointing.

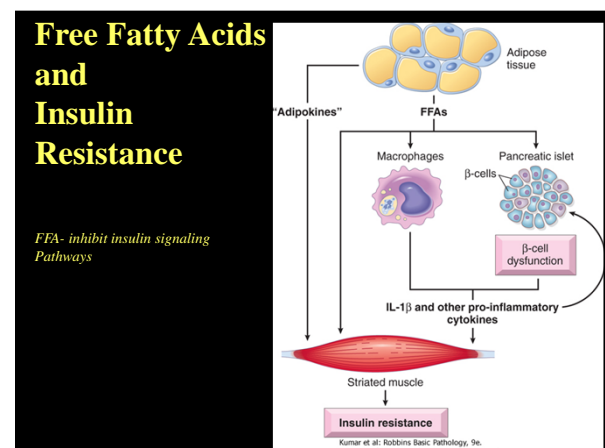
"We had to do a second trial to see if obese people were as sensitive to the hormone," said researcher Dr. Stephen R. Bloom. "The answer: 'Yes, they are. They're just as sensitive as thin people.'"

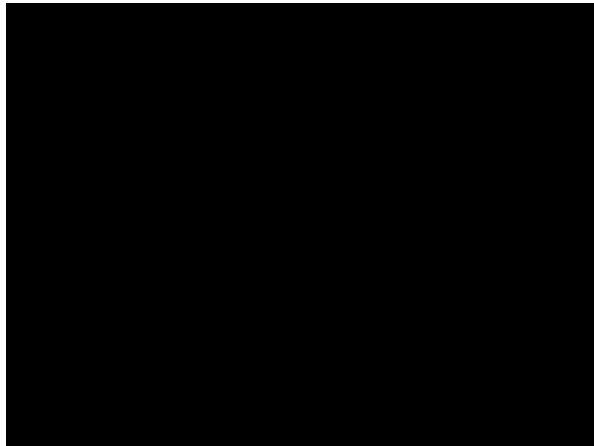
In the small study, both obese and lean people ate about 30 percent less from a buffet lunch after they were given a dose of the hormone, PYY-36, to trick the brain into thinking they had already eaten. The research also showed lower natural levels of PYY in the obese, which may explain why they are hungrier and overeat.

Bloom said long-term use of the hormone would have to be studied before it could be developed into a treatment for obesity that would consist of injections given before meals.

"We haven't yet shown you get actual weight reduction. We've only shown you eat less," Bloom said.

Herald Times
Sept 3, 2003





Protein-Calorie
Malnutrition:

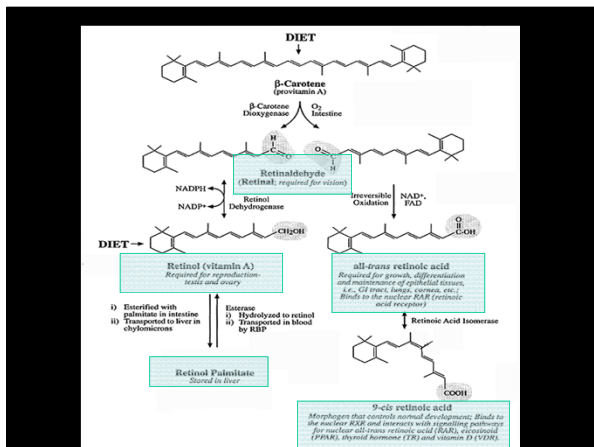
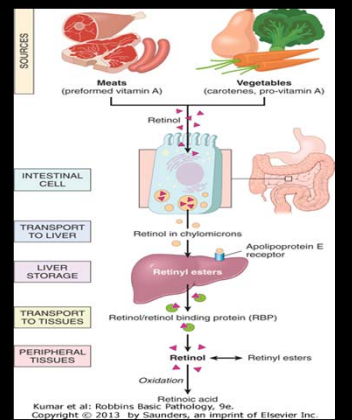
Marasmus



Protein Calorie Malnutrition:
Kwashiorkor

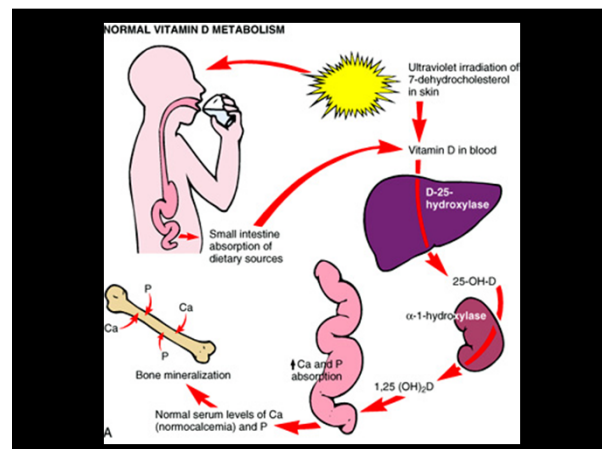
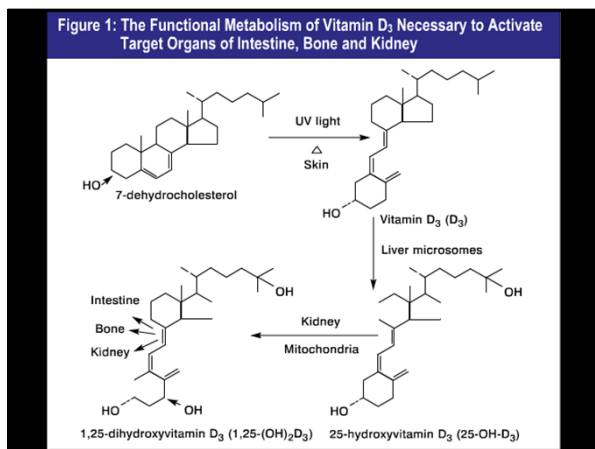
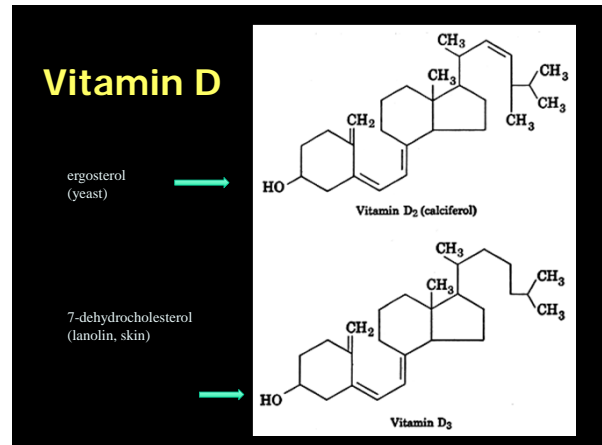
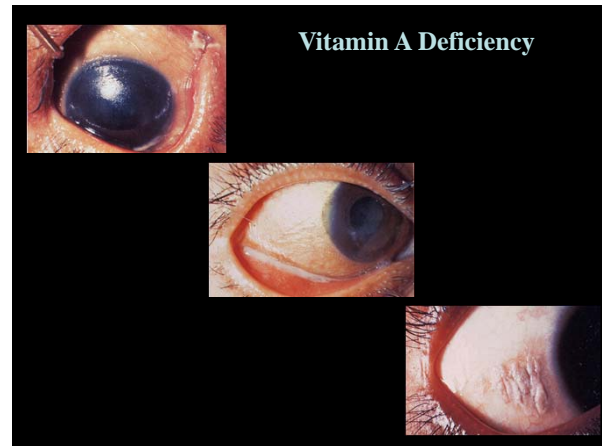
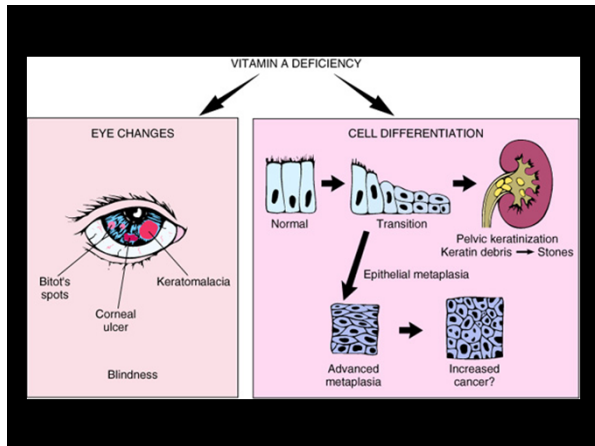


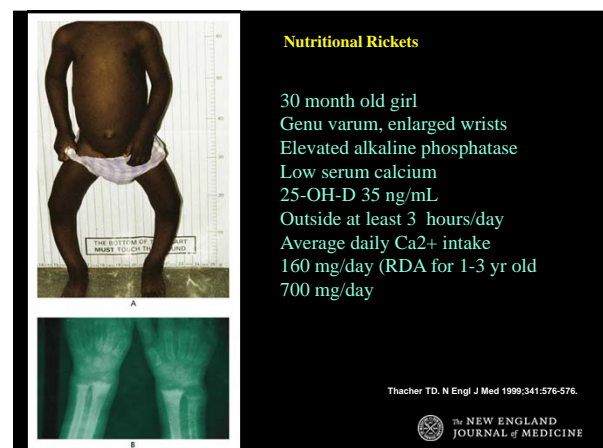
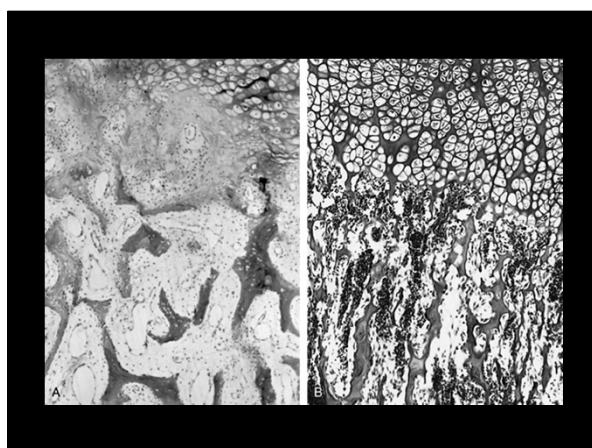
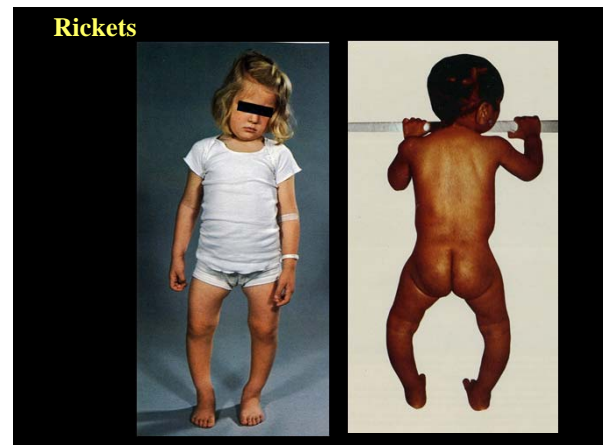
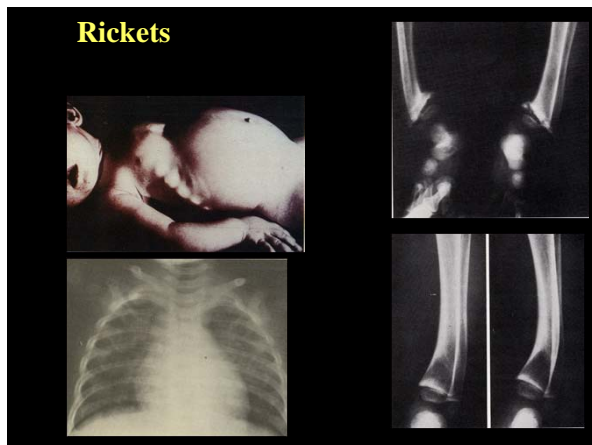
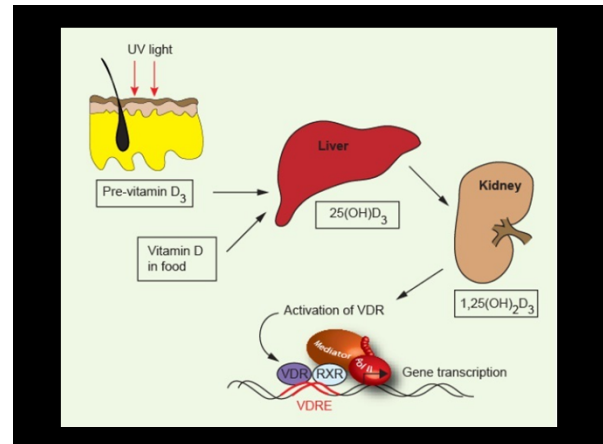
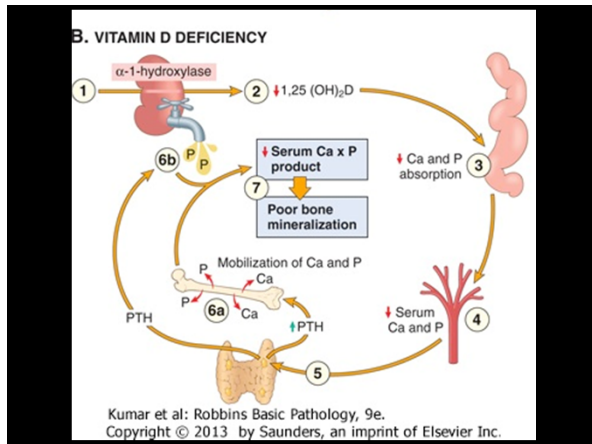
Vitamin A

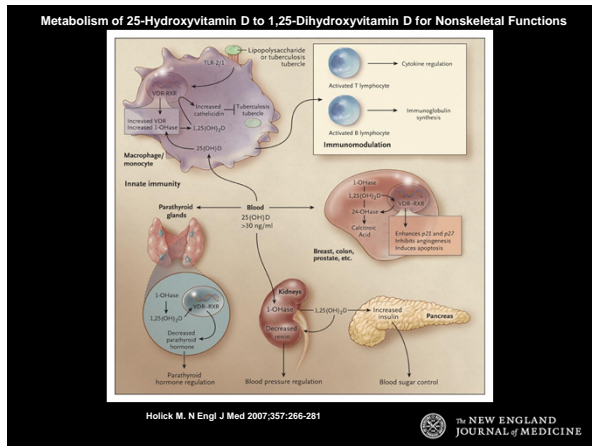


Vitamin A
Deficiency







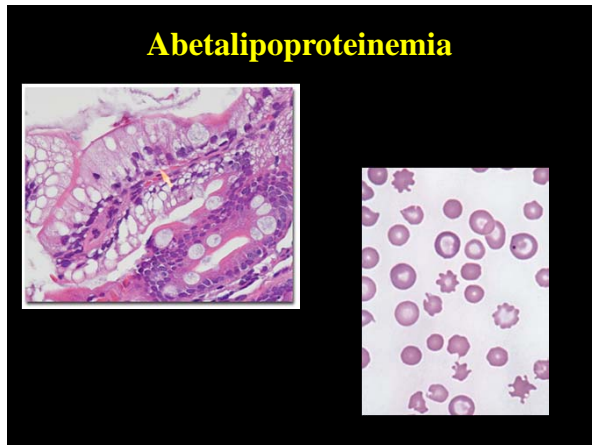


Vitamin E

The diagram shows the chemical structures of Vitamin E, including tocopherols and tocotrienols. The structures are categorized by their side chains:

- alpha-tocopherol:** R¹ = CH₃, R² = CH₃
- beta-tocopherol:** R¹ = CH₃, R² = H
- gamma-tocopherol:** R¹ = H, R² = CH₃
- delta-tocopherol:** R¹ = H, R² = H

The diagram also shows the chemical structures of tocotrienols, which have unsaturated side chains. A bottle of Natural Health Supply Vitamin E is shown on the right.



Vitamin K

The diagram shows the chemical structures of the three forms of Vitamin K:

- K₁ (Phylloquinone):** A naphthoquinone ring with a phytyl side chain.
- K₂ (Menatetrenone/MK4):** A naphthoquinone ring with a menaquinone side chain.
- K₃ (Menadiolone):** A naphthoquinone ring with a menadiolone side chain.

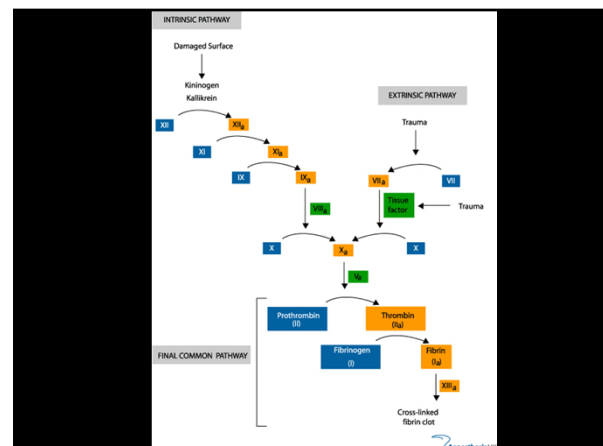
Figure 1. The three forms of vitamin K.

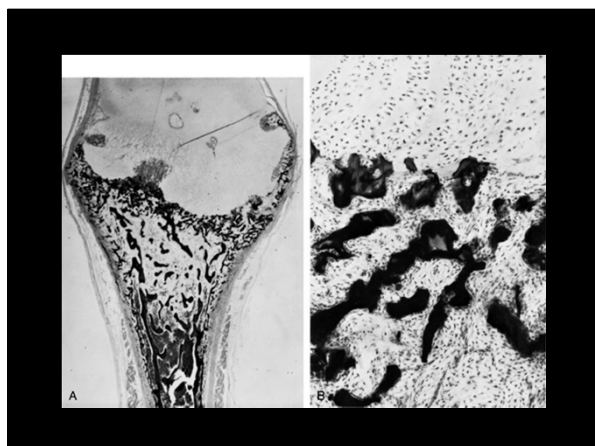
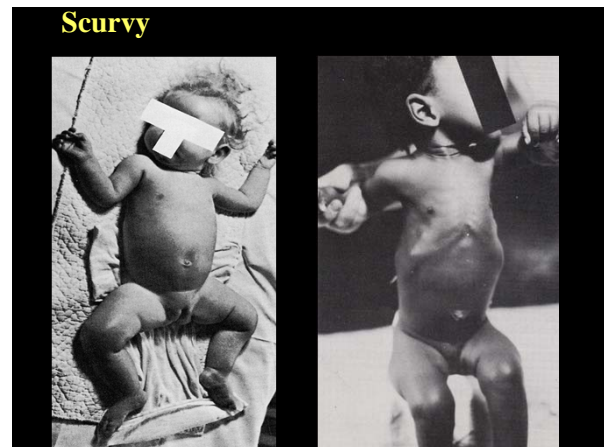
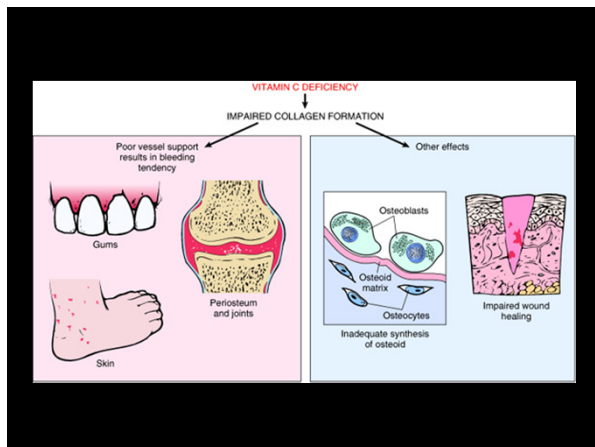
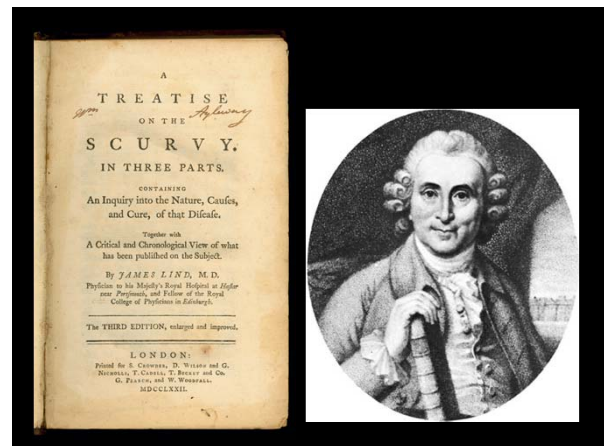
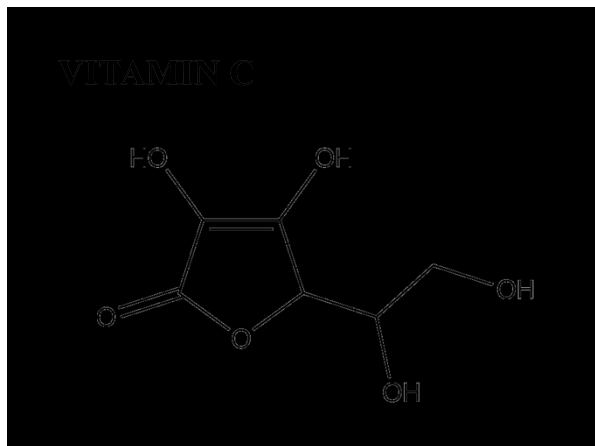
Vitamin K Cycle:

Warfarin inhibits Vitamin K epoxide reductase

The diagram illustrates the Vitamin K cycle. It shows the conversion of prothrombin to active vitamin K (KH₂) and the inhibition of this cycle by warfarin. The cycle involves the following steps:

- Prothrombin is converted to active vitamin K (KH₂) by Vitamin K epoxide reductase (VKOR).
- Active vitamin K (KH₂) is used in the synthesis of prothrombin.
- Warfarin inhibits VKOR, preventing the conversion of prothrombin to active vitamin K.
- The inhibition of VKOR leads to a decrease in active vitamin K, which in turn leads to a decrease in prothrombin, resulting in a decrease in blood clotting.



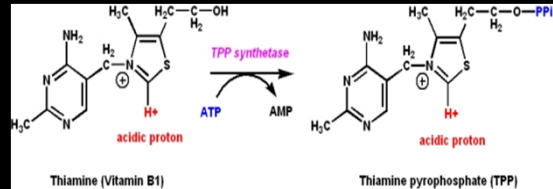


The B Vitamins

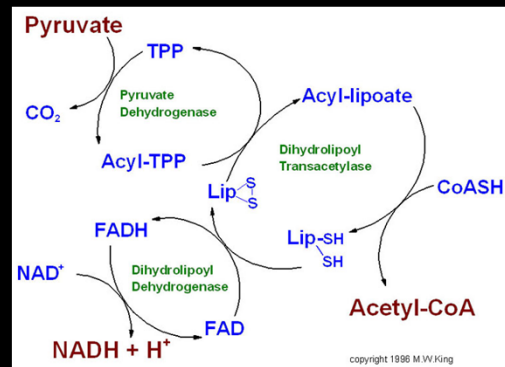
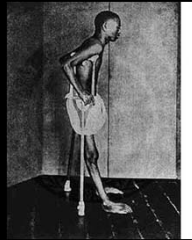
- B1 thiamin (thiamine)
- B2 riboflavin
- B3 niacin
- B4 adenine *
- B5 pantothenic acid
- B6 piridoxine
- B7 biotin
- B8 myoinositol *
- B9 folic acid
- B10 para-aminobenzoic acid (PABA) *
- B12 cyanocobalamin

* Kicked out of the club

Thiamin (e) Vitamin B1

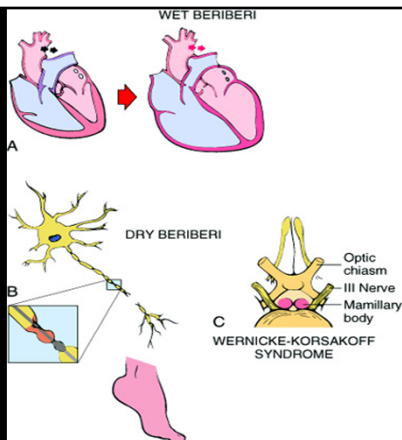


Christiaan Eijkman
and
polished rice

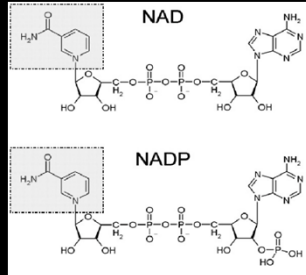
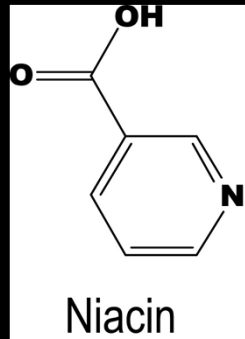


Thiamine Deficiency:

Beri Beri



Niacin (Vitamin B3)



Dr. Joseph Goldberger and Pellagra



Pellagra

