Pathology - Topic 12 - Environmental and nutritional diseases

Environmental Causes of Disease

- An environmental exposure causes disease when the absorbed dose and tissue response exceed the body's capacity to adapt, repair or eliminate the harmful agent.

How Exposure Produces Injury

- The route, duration, timing and combination of exposures influence the outcome.
- The same exposure can produce different effects in people with different ages, nutritional states, genetic susceptibilities and underlying diseases.
    - A careful occupational, household and personal exposure history is therefore part of pathological interpretation.

Air Pollution

- Air pollution exposes the respiratory system to harmful particles and gases that can injure the lungs and affect other organs.

Particles, Reactive Gases and Tissue Effects

- Fine airborne particles activate inflammatory and oxidative responses in the respiratory tract.
    - The particle's size and chemical composition influence its deposition and biological effects.
- Local inflammation can worsen respiratory disease, while systemic inflammatory and vascular effects contribute to cardiovascular risk.
    - Long-term exposure is also associated with increased risk of lung cancer and other adverse outcomes.
- Ozone, nitrogen dioxide and sulphur dioxide irritate or injure the respiratory system through different chemical mechanisms.
    - These pollutants can aggravate airway reactivity and impair respiratory defence in susceptible people.

Particle Size and Cellular Injury

- PM10 and PM2.5 describe particle fractions with aerodynamic diameters up to approximately 10 and 2.5 micrometres respectively.
- Smaller particles penetrate more deeply into the respiratory tract, while chemical composition influences oxidant injury and inflammatory activation.
- Macrophage activation, endothelial dysfunction and altered autonomic responses help connect inhaled pollution with cardiovascular effects.

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Indoor and Occupational Exposure

Household Smoke and Ventilation

- Smoke from inefficient combustion of household fuels can create substantial indoor exposure to particles and toxic gases.
- Poor ventilation increases the concentration inhaled over time.

Occupational Dusts and Exposure History

- Some workplace dusts cause specific fibrosing or neoplastic diseases in addition to general irritation.
- The particular material and exposure history are necessary to distinguish these disorders from ordinary urban air pollution.

Silica and Asbestos Disease

- Crystalline silica activates macrophages and can lead to nodular pulmonary fibrosis and increased susceptibility to tuberculosis.
- Asbestos fibres can produce interstitial fibrosis and pleural disease and increase the risks of lung carcinoma and mesothelioma.
- These examples illustrate why a specific dust history must accompany the general label of occupational pollution.

Carbon Monoxide

Impaired Oxygen Delivery and Cellular Effects

- Carbon monoxide binds haemoglobin strongly and reduces effective oxygen transport and delivery.
- It can also interfere with cellular oxygen use, producing important effects in the brain and heart.
- Severe exposure may cause neurological injury, arrhythmias or death.

Clinical Recognition and Direct Measurement

- A normal-looking skin colour or a routine oxygen-saturation reading cannot reliably exclude carbon-monoxide poisoning.
- Carboxyhaemoglobin reduces the amount of haemoglobin available to bind oxygen and shifts the remaining oxygen–haemoglobin relationship toward tighter binding.
- Standard pulse oximetry cannot reliably distinguish oxyhaemoglobin from carboxyhaemoglobin, so co-oximetry is used for direct assessment.

Delayed Neurological Injury

- Delayed neurological injury can follow substantial exposure even after the initial hypoxic event has improved.

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Tobacco-Related Disease

- Tobacco-related disease results from toxic and carcinogenic exposures that damage the respiratory, cardiovascular and other systems.

Airway and Lung Changes

- Tobacco smoke contains irritants, oxidants and carcinogens that act through several pathways.
    - Nicotine promotes dependence and sympathetic effects, while many combustion products cause additional tissue injury.
- Repeated airway exposure impairs mucociliary clearance and promotes mucus production and chronic inflammation.
    - Squamous metaplasia may replace normal ciliated epithelium and further reduce airway clearance.
- Protease activity and oxidative injury contribute to destruction of alveolar walls in emphysema.
    - Loss of elastic recoil and small-airway abnormalities impair expiration.

Structural Basis of Obstructive Disease

- Goblet-cell hyperplasia and enlargement of mucus glands increase airway secretions in chronic bronchitic injury.
- Small-airway inflammation and fibrosis narrow the lumen, while emphysematous destruction reduces the surface area for gas exchange.
- Oxidant injury can impair antiprotease activity and favour protease-mediated destruction of alveolar walls.

Cancer and Vascular Effects

- Carcinogens in smoke can damage DNA and favour expansion of altered cells.
    - Tobacco is associated with cancers of the lung and several other organs, not only tissues directly touched by smoke.
- Smoking promotes endothelial dysfunction, platelet activation and atherosclerotic disease.
    - Carbon monoxide adds to impaired oxygen delivery, while sympathetic stimulation can increase cardiac demand.
- Smokeless tobacco also causes important disease and can increase oral cancer risk in relevant exposure settings.
    - The absence of combustion does not make a tobacco product harmless.

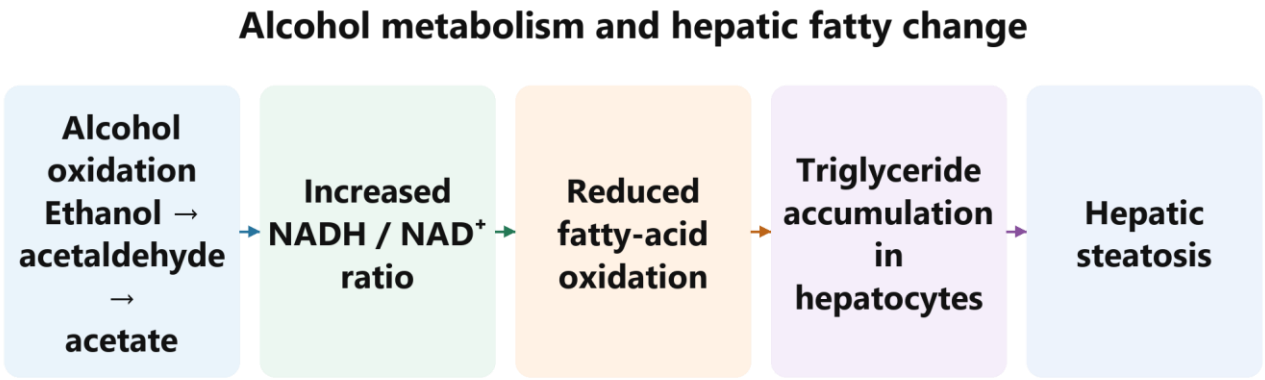
Alcohol-Related Disease

- Alcohol-related disease includes tissue injury caused by ethanol, its metabolites and associated metabolic disturbances.

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Metabolism and Cellular Effects

Alcohol Metabolism and Redox Balance



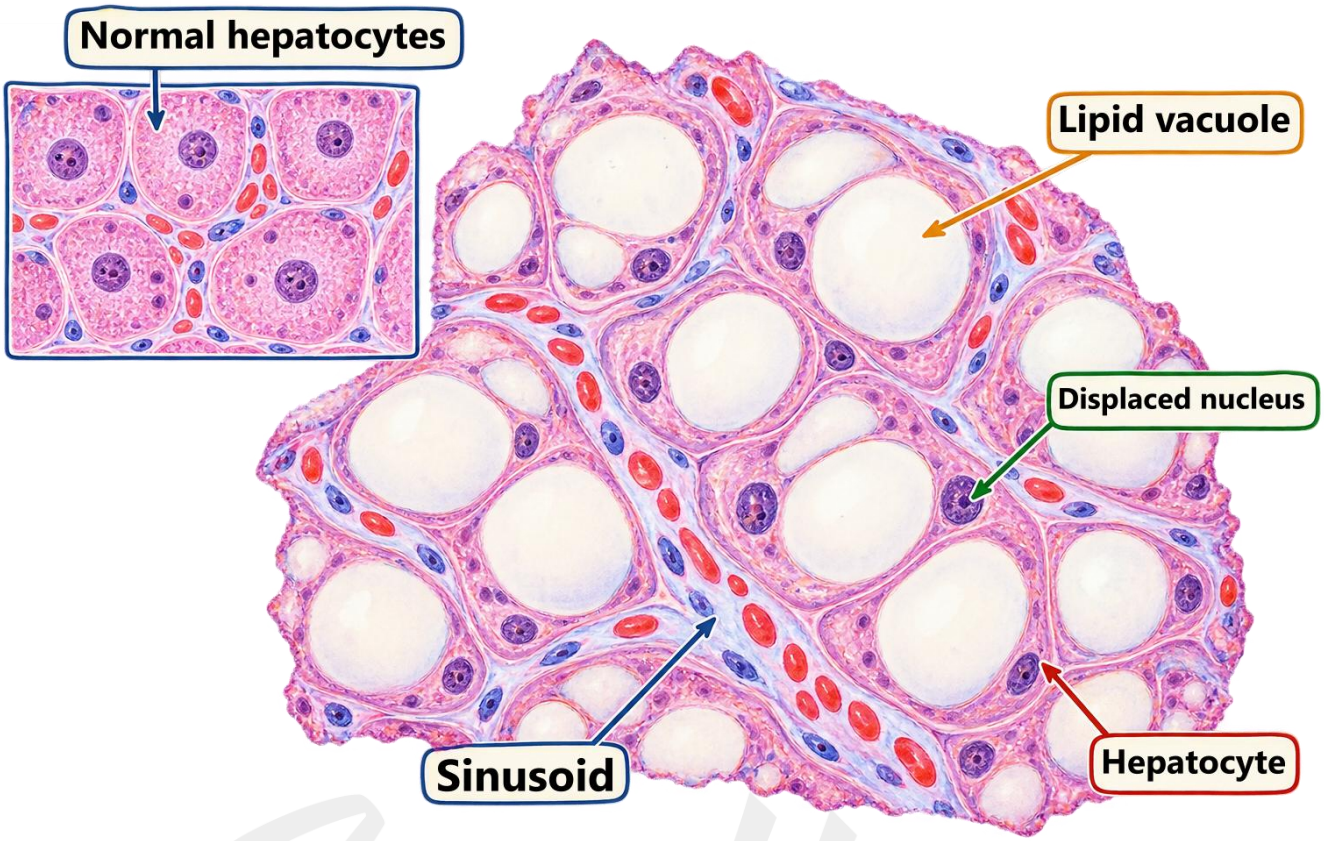
- Alcohol dehydrogenase converts ethanol into **acetaldehyde**, which is then further metabolised mainly to acetate.
- Acetaldehyde can bind cellular molecules and contribute to toxicity and carcinogenesis.
- Alcohol oxidation increases the NADH-to-NAD ratio and alters lipid and carbohydrate metabolism.
- Reduced fatty-acid oxidation and other metabolic effects favour triglyceride accumulation in hepatocytes.

Mechanisms of Hepatic Injury

- Induction of CYP2E1 and other processes can increase oxidative stress.
- Mitochondrial injury, inflammatory signalling and changes in intestinal permeability add to hepatic damage.
- The increased NADH concentration shifts pyruvate toward lactate and can impair gluconeogenesis, particularly when glycogen stores are depleted.
- Acetaldehyde–protein adducts and oxidative stress can activate inflammatory and fibrogenic pathways.
- Increased intestinal permeability allows microbial products to reach the liver and stimulate resident macrophages, further amplifying injury.

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The Liver Spectrum



Macrovesicular fatty change displaces the hepatocyte nucleus and may accompany nutritional or toxic metabolic injury

1. Steatosis

- **Steatosis** is accumulation of fat within hepatocytes and may make the liver enlarged, yellow and greasy.
- Large lipid vacuoles can displace the nucleus to the edge of a hepatocyte.

2. Steatohepatitis

- Alcohol-associated steatohepatitis can show ballooned hepatocytes, lobular inflammation and variable neutrophils.
- Mallory–Denk bodies are cytoplasmic aggregates of damaged intermediate-filament proteins that may be present.

3. Fibrosis and Cirrhosis

- Pericellular and perivenular fibrosis can progress to bridging fibrosis and cirrhosis.
- Not every person progresses through the same sequence or at the same rate.
- These histological features are not uniquely caused by alcohol and must be interpreted with the clinical history.

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