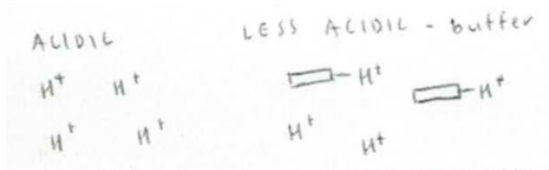


BUFFERS

Can bind or release H^+ to minimise changes in the concentration of free H^+ .



If H^+ are added to a solution, the buffer will bind most of them preventing a sudden drop in pH.

If H^+ are removed from a solution, the buffer will release H^+ thus preventing a sudden rise in pH.

BLOOD BUFFERING

The most important buffers in blood are bicarbonate and Hb.

Hb buffering

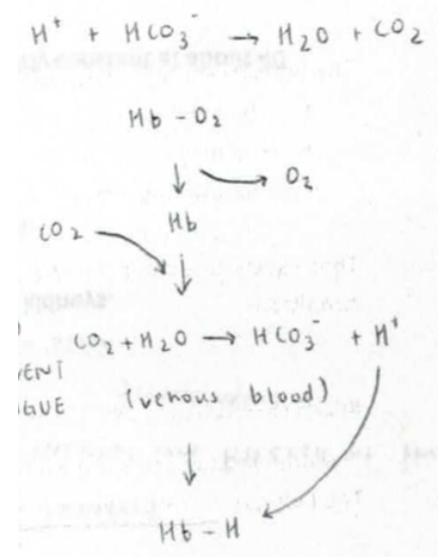
In the tissues CO_2 is added to the blood, lowering the pH.

Once Hb has release O_2 , it can bind H^+ .

Plasma proteins and phosphates make minor contributions.

NB: Level of contribution (1) HCO_3^- (2) Hb (3) Phosphates (4) Proteins.

- Acidosis \rightarrow fatigue in exercise
- $HCO_3^- \rightarrow$ prevents acidosis \rightarrow prevents fatigue in exercise



INTRACELLULAR BUFFERING

The most important intracellular buffers are proteins and phosphates.

- Creatine-P + ADP \rightarrow creatine (acts as a buffer) + ATP

URINARY BUFFERS

H^+ is secreted in proximal and distal tubules. If urinary pH falls below 4.5, H^+ excretion will cease to prevent damage.

The main urinary buffers are phosphates and ammonia.

- Collect free H^+ to protect the urinary system by preventing urine from becoming too acidic, which can damage the renal system.

Proton excretion in the kidneys:

- Is achieved by active transport
- Is linked to Na^+ reabsorption
- Is stimulated by aldosterone

H^+ and K^+ compete for Na^+ exchange. Increased H^+ pumping and decreased K^+ pumping occur in acidosis. Therefore acidosis may result in hyperkalaemia.

Normally, all HCO_3^- which is filtered by the glomerulus is reabsorbed in the kidneys. When $[HCO_3^-]$ is high (>28 mmol/l), the renal threshold is exceeded and bicarbonate will appear in the urine.

In summary:

If we have excess H^+ , swap Na^+ for H^+ BUT retain K^+ . ACIDOSIS \rightarrow HYPERKALAEMIA (excess K^+)

If we have excess K^+ , swap Na^+ for K^+ BUT retain H^+ . HYPERKALAEMIA \rightarrow ACIDOSIS (excess H^+)

NB: pH of urine often reflects pH of the blood.

ACID BASE ABNORMALITIES

Classified by aetiology and type.

1. Aetiology – underlying cause (which system is broken)

- Metabolic – primary abnormality is in bicarbonate levels or non-volatile acid production or excretion
- Respiratory – primary abnormality is in CO_2 control – determines H^+ /pH of blood
- Mixed – only seen in severely ill patients

2. Type

- Acidosis
- Alkalosis

METABOLIC ABNORMALITIES

Due to primary changes in:

- $[HCO_3^-]$
- [Non-volatile acid]

Bicarbonate

- The primary loss of HCO_3^- causes metabolic acidosis e.g. diarrhoea
 - o HCO_3^- deficiency \rightarrow cannot neutralise stomach acid \rightarrow acidosis
- The direct gain of HCO_3^- causes metabolic alkalosis e.g. via ingestion

stomach \rightarrow intestine
acidic \rightarrow neutral (HCO_3^-)