بسم الله الرحمن الرحيم



# BioChemistry | Lecture #5 **pH and Buffers** pt.3

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#### Recap related to the previous lecture:

• Buffers are really important to maintain the PH value that being changed drastically, they are in solutions ( aqueous solutions) such as our blood.

- bisphosphoglycerate exists in high concentrations in RBCs which is an intermediate for glycolysis.
- glycolysis is the only process within your body that occurs in all the body cells.
- Any protein in your body can work as a buffer by resisting and maintaining PH changes

# Q/ What does this system depends on? from where do we get this buffer and what are its properties?

Ans/ it depends on CO2 because it's present on high concentrations within the body. (This high concentration is due to Krebs cycle of the aerobic respiration.) It also depends on H2O, which is the main component of the body.

\* CO2 is calcified as an acid, so when it attaches itself to water, it gives H2CO3 which is called "carbonic acid", this acid gives HCO3- "bicarbonate acid". So basically each acid led to another acid.. Which also means each one is a conjugated base to the other.



**Bicarbonate buffer** 



(within minutes)

Excretion via kidneys (hours to days)

### Titration curve of bicarbonate buffer

- \*You should know that the ideal PH of the blood is 7.4 (the normal range is 7.35 – 7.45 which is not considered as pathological)
- You can see that the pka of the ideal system
   =6.1
- which kind of doesn't make sense, because for this buffer to work effectively, the buffering reign should be (5.1–7.1), while PH of the blood = 7.4 which means it is outside of the range, so why this buffer was chosen to be the buffering system of the blood when it's outside of its buffering capacity?
- There are many reasons



### Why is this buffer effective?

Relatively high concentration in the ECF (24mmol/L)

The higher concentration the lower of the difference of Pka value to PH value.

- Components are effectively under physiological control
  - CO2 by lungs
  - Bicarbonate by kidneys
- It is an open system (continuously interacts with its environment. "Can control the amount that is released "

\*as we know the buffer in the blood resist changes in PH and keep it in the normal range but it's not always successful despite existing in high concentrations.

Q/ Why does the buffer fail?

Ans/ Because of the constant supplying of the base or acid which results in two conditions: "Acidosis" PH is below 7.35 "Alkolalosis" PH is higher than 7.45

#### Acidosis and alkalosis

- Can be either metabolic or respiratory
- Acidosis (pH< 7.35)</li>
  - Metabolic: production of ketone bodies (starvation)
  - Respiratory: pulmonary (asthma; emphysema)
- Alkalosis (pH > 7.45)
  - Metabolic: administration of salts
  - Respiratory: hyperventilation (anxiety)

The sources of these two conditions are the two open systems (metabolic and respiratory), the lungs control the respiratory as it controls the amount of CO2.

Remember that CO2 is an acid, so any condition that increases the amount of it results an acidosis, and any respiratory problem that causes decreasing the CO2 amount results alkalosis.

 $\ensuremath{^*\text{We}}\xspace$  differentiate between the two conditions by the PH value .

#### Respiratory conditions



**Respiratory Alkalosis** 

**CO2** decreases

 $H^+ + HCO_3^- \leftrightarrow H_2CO_3 \leftrightarrow \infty_2 + H_2O$ 

#### Metabolic conditions

**Metabolic Acidosis** 

## $H^+ + HCO^- \leftrightarrow HCO \leftrightarrow CO + HO$

**Metabolic Alkalosis** 

 $H^{+} + HCO_{3} \leftrightarrow H_{2}CO_{3} \leftrightarrow CO_{2} + H_{2}O$ 

Any breathing issues that affect the CO2 amount

#### Causes of respiratory acid-base disorders



#### Causes of metabolic acid-base disorders



metabolic acidosis

Vomiting leads the cells to secrete more H+ towards the space to make up for the loss of acids but this causes H+ concentration deficiency in the blood Loss of H+ in vomit

#### **Alkali ingestion**

#### Potassium deficiency

Because it is a charged molecule and this deficiency leads to increase H+ excretion by the kidney

### $H^{+}(aq) + HCO_{3}^{-}(aq) \longrightarrow H_{2}CO_{3}(aq) \longrightarrow H_{2}O_{1} + CO_{2}(g)$

This equation is at equilibrium, but what if there's an issue with the lungs? It means either CO2 concentration is increased or decreased, which will disturb the equilibrium and in this case the body will compensate it, for example: if the CO2 increases the protons concentration will increase, the compensation will be done by the kidneys which will control HCO3- by stopping excretion it this will result in raising the concentration of HCO3which will bind to the increased H+ (the protons) to go back to the equilibrium.

So in this case "respiratory acidosis" the \*primary change is: increased CO2 concentration and the \* compensatory mechanism is: increasing HCO3- concentration.

## Compensation<sup>H+ (aq) + HCO<sub>3</sub><sup>-</sup> (aq) $\rightarrow$ H<sub>2</sub>CO<sub>3</sub>(aq) $\rightarrow$ H<sub>2</sub>O<sub>(1)</sub> + CO<sub>2</sub>(g)</sup>

- If metabolic: hyperventilation or hypoventilation
- If respiratory: renal mechanisms
- May be complete or partial

PCO2/ partial pressure of PCO

You need to

**ABG** values

memorize the

ABG refers to arterial blood gasses **Normal values** 

e of		ABG	VBG	CBG
	рН	7.35-7.45	7.25-7.35	7.35 - 7.45
	PCO2 (mmHg)	35-45	41-51	35 - 48
	PO2 (mmHg)	80-100	35-40	80-100
	HCO3 (mmol/L)	22-26	22-26	22 - 27

Acid-Base DisorderPrimaRespiratory acidosispCORespiratory alkalosispCOMetabolic acidosisHCOMetabolic alkalosisHCO

Primary Change  $pCO_2 up$   $pCO_2 down$   $HCO_3^- down$  $HCO_3^- up$ 

**CO2** 

Compensatory Change HCO<sub>3</sub> $\mu$ p HCO<sub>3</sub>downPCO<sub>2</sub> down PCO<sub>2</sub> up

### FULLY COMPENSATED

Maintained PH value

$$H^+(aq) + HCO_3^-(aq) \longrightarrow H_2CO_3(aq) \longrightarrow H_2O_{(1)} + CO_2(g)$$

	рН	pCO <sub>2</sub>	HCO <sub>3</sub>
Resp. acidosis	Normal but<7.40		
Resp. alkalosis	Normal but>7.40		
Met. Acidosis	Normal but<7.40		
Met. alkalosis	Normal but>7.40		

#### Partially compensated it try to get the PH back to normal value but not completely reach it

	рН	pCO <sub>2</sub>	
Res.Acidosis	down	up	up
Res.Alkalosis	up	Down	Down
Met. Acidosis	down J	Down	Down J
Met.Alkalosis	up	up 🔒	up

	Disorder	Characteristics	Selected situations
	Respiratory acidosis with metabolic acidosis	↓in pH ↓ in HCO₃ ↑ in PaCO₂	<ul><li>Cardiac arrest</li><li>Intoxications</li><li>Multi-organ failure</li></ul>
Not required just to know	Respiratory alkalosis with metabolic alkalosis	↑in pH ↑ in HCO₃- ↓ in PaCO₂	<ul> <li>Cirrhosis with diuretics</li> <li>Pregnancy with vomiting</li> <li>Over ventilation of COPD</li> </ul>
met	Respiratory acidosis with metabolic alkalosis	pH in normal range ↑ in PaCO <sub>2</sub> , ↑ in HCO <sub>3</sub> -	<ul> <li>COPD with diuretics, vomiting, NG suction</li> <li>Severe hypokalemia</li> </ul>
	Respiratory alkalosis with metabolic acidosis	pH in normal range ↓ in PaCO <sub>2</sub> ↓ in HCO <sub>3</sub>	<ul> <li>Sepsis</li> <li>Salicylate toxicity</li> <li>Renal failure with CHF or pneumonia</li> <li>Advanced liver disease</li> </ul>
	Metabolic acidosis with metabolic alkalosis	pH in normal range HCO <sub>3</sub> - normal	<ul> <li>Uremia or ketoacidosis with vomiting, NG suction, diuretics, etc.</li> </ul>



#### For any feedback, scan the code or click on it

#### Corrections from previous versions:

Versions	Slide # and Place of Error	Before Correction	After Correction
V0 → V1			
V1 → V2			

رسالة من الفريق العلمي:

