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CBB - B10 - 211 - Cell Biology and Biochemistry

Assignment - 2

1.

A. Palmitic acid made of 16C

C_{2n} $\quad 2n = 16$

$\boxed{n = 8}$

Complete oxidation → 2355 kcal

Chem. energy available in High energy phosphate bond = 7.3 kcal

ATP Molecules maximally generated = $\frac{2355}{7.3}$ = 322.6 ATP molecules

B. As n = 8 so 8 Acetyl CoA is formed

(n-1) = no. of $NADH$ & $FADH_2$ = 7

7 NADH = 7 × 3 = 21

7 $FADH_2$ = 7 × 2 = 14

1 NADH = 3 ATP

1 $FADH_2$ = 2 ATP

1 Acetyl CoA forms = 12 ATP

8 forms ⟶ 12 × 8 = 96

So total = 96 + 21 + 14 = 131

Since 2 ATP used to activate fatty acid = 131 - 2 = 129 ATP

because in presence of CoA-SH & acyl CoA synthetase fatty acids activated before oxidation. Beta oxidation of fatty acid occur in within mitochondria. So name beta-oxidation is broken b/w the second carbon/beta carbon & 3rd carbon/gamma carbon. Fatty acid oxidation rate high during fasting ~~animal~~, higher concentration of unesterified fatty acid in blood stream of fasting animal relative to concentration in fed animal one cause of ketosis. main metabolic pathway by which energy is released is β-oxidation.

Fatty Acid Activation oxidation of beta TCA cycle-

$$\text{Efficiency} = \frac{129}{322.6} = 0.3998$$

$$= 39.98\% \approx 40\%$$

(c.)

Remaining is 60% i.e = 2356×0.6

$$= 1413 \text{ kcal}$$

we know

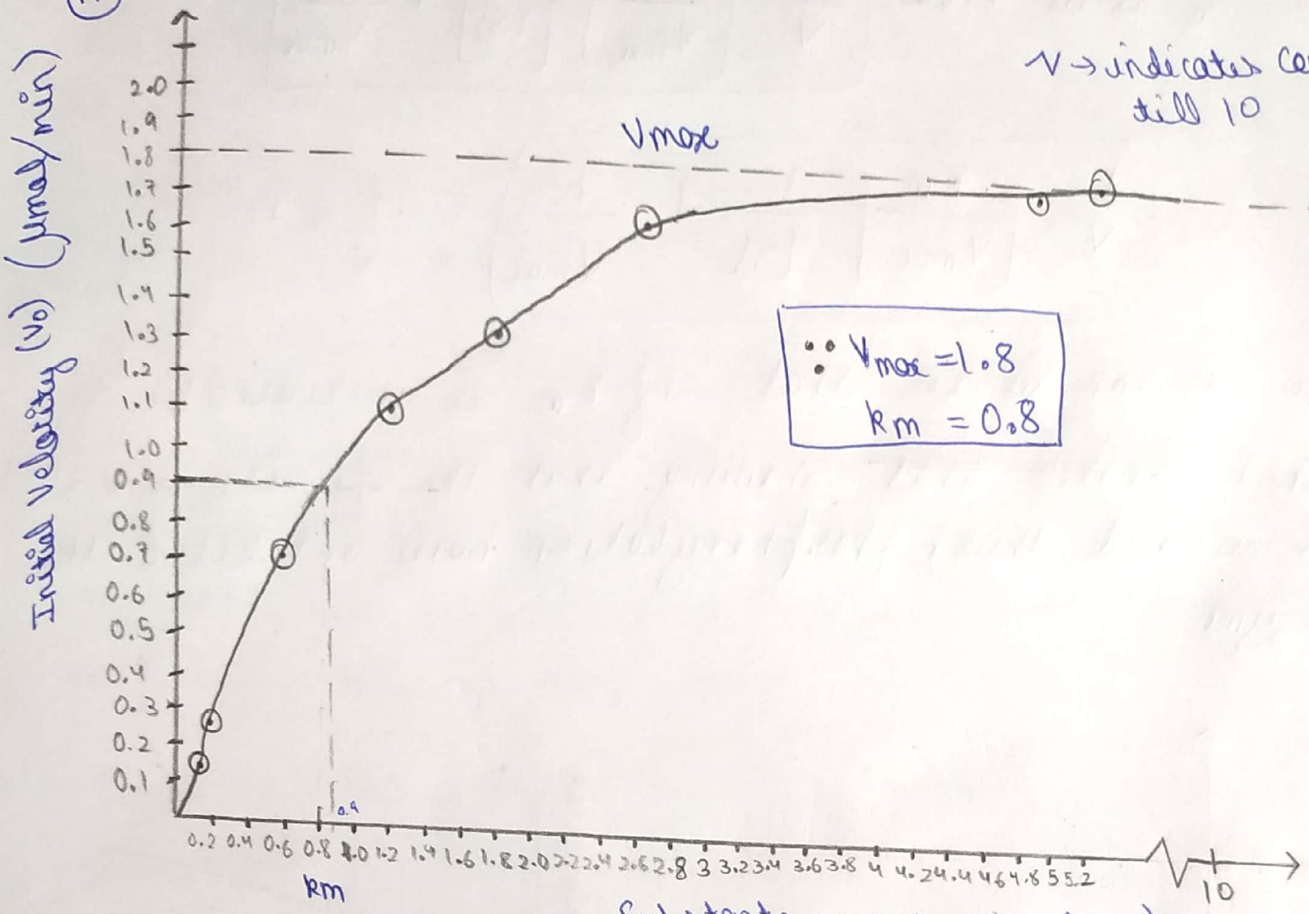
$$\boxed{Q = mc\Delta T}$$

$$\Delta T = \frac{Q}{mc} = \frac{1413}{75} = 18.84^{\circ}C$$

So 18.84°C is the temperature raised by 1 mole of Palmitic acid. oxidation

(2.) (A.)

Michaelis Menten



From graph we observe Km to be 0.9 μM

$V_{max} = 1.8$ μmol/min

(B.) (Lineweaver Beck Plot)

Acc. to the graph generated using MS Excel we get the eqn as

$$y = 0.4986x + 0.5026$$

comparing with

$$\frac{1}{v} = \left[\frac{K_m}{V_{max}}\right]\left[\frac{1}{[S]}\right] + \frac{1}{V_{max}}$$

$$\frac{K_m}{V_{max}} = 0.4986$$

$$\frac{1}{V_{max}} = 0.5026$$

$$V_{max} = 1.98965 \ \mu mol/min$$

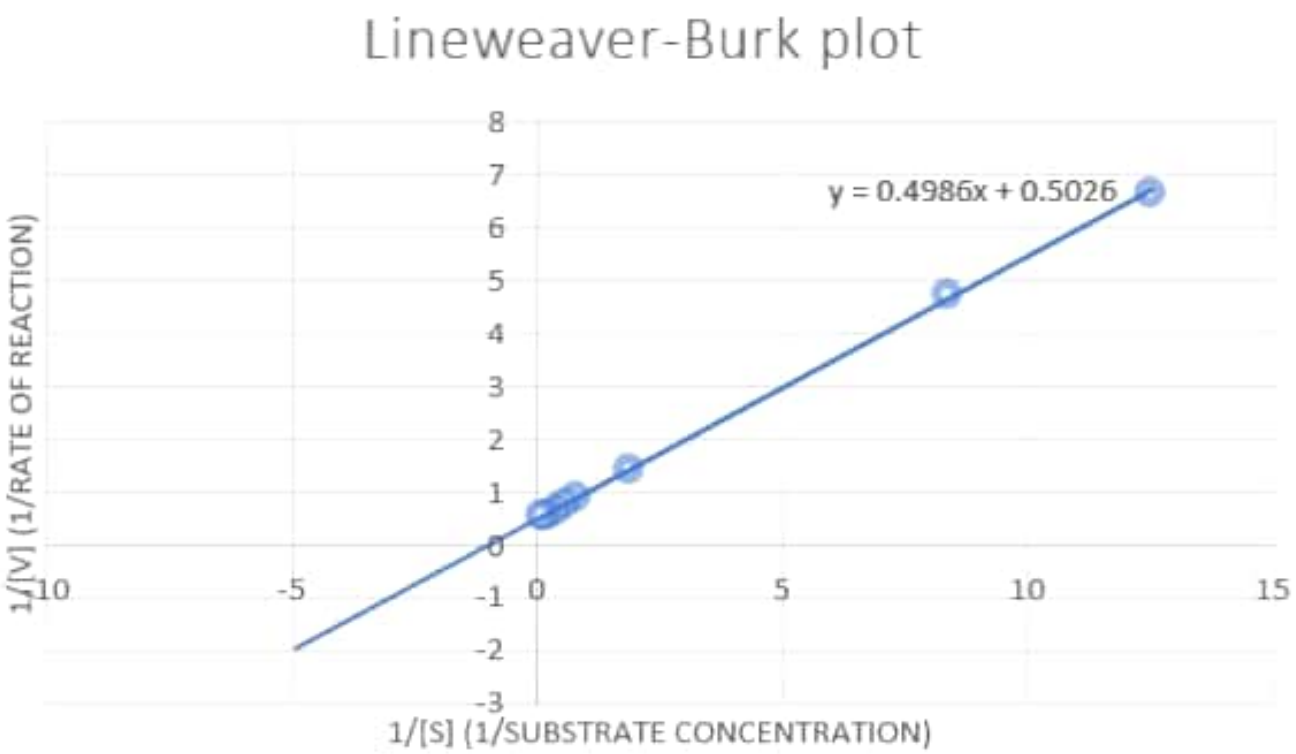
$$K_m = 0.992039 \ \mu M$$

Total Error in Km = $\frac{0.9}{0.9920} \approx 10\%$ (90 → 100, 90)

Error in V_{max} = $\frac{1.8}{1.98965} \approx 10\%$ (90 → 100, 100 − 90) again

No, the results don't agree with the estimates made from first graph as it can be due to human error or any calc. mistake.

[S]	v	1/[S]	1/v
0.08	0.15	12.5	6.666667
0.12	0.21	8.333333	4.761905
0.54	0.7	1.851852	1.428571
1.23	1.1	0.813008	0.909091
1.82	1.3	0.549451	0.769231
2.72	1.5	0.367647	0.666667
4.94	1.7	0.202429	0.588235
10	1.8	0.1	0.555556



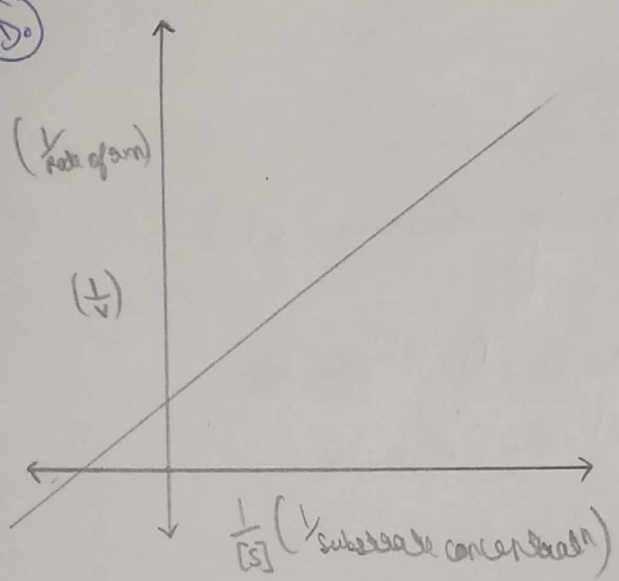
(C.) $K_m' = 3K_m$

So as we know $\frac{1}{V} = \left[\frac{K_m}{V_{max}}\right]\left[\frac{1}{[S]}\right] + \frac{1}{V_{max}}$

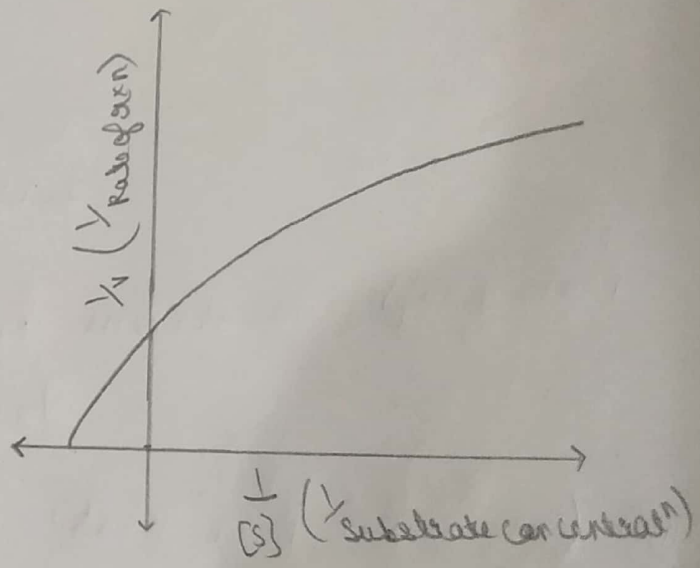
$\frac{1}{V} = \left[\frac{3K_m}{V_{max}}\right]\frac{1}{[S]} + \frac{1}{V_{max}}$ $\quad \frac{1}{V} \propto K_m$

So, we can see that if km is increased by 3 ~~which denotes that~~ showing that the affinity of enzyme is decreased thus, phosphorylation have inhibited the enzyme.

(D.)



unmodified enzyme



Phosphorylated enzyme

As we can see from the graph that in phosphorylated enzyme shows non classical kinetic behaviour over a wide range of substrate concentrations as evidenced by a slightly downward curvature as the substrate concentration is increased. The induced conformational change in enzyme causes the affinity of the paired catalytic site to be reduced by a factor. The induced change also causes an increase in catalytic efficiency such that, when the second substrate molecule binds the rate of hydrolysis is characterized by different higher maximal velocity. This behaviour result from binding site.

(3.)

(A.) Cyanide is a potent cytochrome oxidase (COX) cyanide poisoning is a form of histotoxic hypoxia because this interferes with oxidative phosphorylation. Specifically cyanide binds to the heme binuclear center of COX non-competitive inhibitor that prevents e^-s passing through COX from being transferred to O_2

→ That does not only blocks mitochondrial electron transport chain but also interfere with pumping of proton out of mitochondrial matrix that would otherwise occur at this stage.

→ Cyanide not only interferes with aerobic respiration but also with ATP synthesis pathway it facilitates evening low relationship.

→ Cyanide broad spectrum poison because it inhibits essential axn in to aerobic metabolism. COX found in many form of lives.

→ Susceptibility to cyanide is far from uniform across affected species, plants have alternate e^- transfer pathway available that passes e^- directly from ubiquinone to O_2 which confers cyanide by passing COX.

Ref. → Wikipedia

(13.)

→ Curare has long been used by the Indian of South America for hunting wild game. After the effects of curare ~~was known it was started using in~~ i.e voluntary muscles selectively without affecting the brain and heart it was widely used in general anesthesia frequently with cyclopropane.

→ Curare recently been used in humans to relieve shivering and also in animal experimentation to block shivering to diagnose myasthenia gravis and to manipulate neuromuscular transmission.

→ Curare acts as a neuromuscular blocking agent by binding agent by binding to the acetylcholine receptor (AChR) at the neuromuscular junction and preventing nerve impulses from activating skeletal muscles. Although high doses of curare are lethal low doses will tolerated & effect is completely reversible.

→ In Experiments many have examined dose-dependent effects on both male and female by ~~&~~ monitoring their physical activity and whole body metabolic rate.