Ho Medical ANAESTHESIA

Dr Matthew Ho
BSc(Med) MBBS(Hons) FANZCA

Metabolic Physiology

Physiol-06A13 Describe the factors which influence metabolic rate.

1. Metabolic rate: the rate of energy production by the body per unit time, which is the sum of external work, heat production and energy storage.
    a. Basal metabolic rate is the standardised metabolic rate measured at specific conditions → at physical and mental rest, in thermoneutral zone, 12 hour post-meal.
    b. Normal 2000kcal/day
    c. Measurement:
        i. Direct – measure heat produced in Atwater-Benedict chamber
        ii. Indirect – measure O_2 consumption by closed circuit wet spirometer

2. Factors affecting metabolic rate:
    a. Muscular activity – skeletal muscle is the largest organ source for energy expenditure.
        i. Exercise → ↑10-20 fold energy production by external work and heat.
        ii. Oxygen debt: energy consumption raised after energy to repay O_2 debt.
    b. Feeding status -
        i. Feeding: ↑ metabolic rate by 15% due to food consumption. Specific dynamic action (SDA) measures the ↑ metabolism required for digestion of specific foods – protein 30%, glucose 6%, fats 4%
        ii. Fasting: prolonged starvation > 2 weeks → ↓ BMR by 40% due to ↓ cell mass and tissue metabolism
    c. Temperature:
        i. Ambient temperature: temperature range outside TNZ (27-31°C) causes linear ↑ metabolic rate for thermoregulation. Generally 10% ↑ metabolic rate for each °C outside TNZ.
        ii. Core body temperature:
            1. Hypothermia:
                a. Mild - ↑ metabolic rate (shivering, behavioural change)
                b. Moderate/severe hypothermia – inhibition metabolic pathways → ↓ metabolic rate
            2. Hyperthermia: ↑ metabolic rate by 14% per °C rise.

3. Factors affecting basal metabolic rate (hence metabolic rate):
    a. Body surface area: linear ↑ metabolic rate with BSA, hence most people have BMR corrected to BSA. Normal value is 58W/m^2.
    b. Body fat content:
        i. ↑ fat content → ↓ metabolic rate
        ii. Hence females have ↓ metabolic rate
    c. Age: ↓ metabolic rate with age.
        i. Neonates 2x BMR adult (growth)
        ii. ↓BMR 2% per decade of life (↓ muscle mass)
    d. Hormonal:
        i. Thyroid - ↑ cellular enzymatic activity → ↑ BMR
        ii. Adrenaline - ↑ cellular enzymatic activity → ↑ BMR
        iii. Pregnancy – 20% rise BMR during 2^{nd}/3^{rd} trimester
        iv. Lactation - ↑ BMR

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Physiol-09A15 Describe the formation, fate and role of lactate in energy production.

1. Lactate is a chemical product of the anaerobic metabolism of pyruvate, catalysed by lactate dehydrogenase.
    a. Normal levels 0.5-2mmol/L

2. Formation – from pyruvate metabolism. Pyruvate is an intermediate product of glycolysis. It undergoes the following reaction:

$$\textbf{pyruvate} \Leftrightarrow \textbf{lactate}$$
$$\textbf{NADH} \Leftrightarrow \textbf{NAD}^{+}$$

    a. Pyruvate is formed from several metabolic cycles
        i. Carbohydrate metabolism via Glycolysis: glucose → 2 pyruvate
            1. Yields 4 ATP and 4H
        ii. Lipid metabolism via Gluconeogenesis: glycerol → pyruvate
        iii. Protein metabolism via glucogenic transamination: alanine → pyruvate
    b. Aerobic glycolysis: pyruvate → acetyl CoA → enters the citric acid cycle to produce electrons (H atoms) for oxidate phosphorylation → generation of ATP.
    c. Anaerobic Embden-Meyerhof pathway → no O_2 → no oxidative phosphorylation, citric acid cycle → build up of pyruvate → converted to lactate.
    d. Lactate production under normal circumstances occurs in cells with low O_2 tension and/or absence of mitochondria (for oxidative phosphorylation)
        i. RBCs
        ii. Cornea, Lens
        iii. Renal medulla
        iv. White skeletal muscle cells

3. Fate:
    a. Diffusion out of cells into plasma down concentration gradient → lactic acidosis
    b. Energy source:
        i. Cori cycle: the conversion of glucose → pyruvate → lactate releases 4 ATP molecules (3% energy efficiency). Lactate diffuses into plasma → liver → Gluconeogenesis → reconversion to glucose for further anaerobic metabolism
    c. Gluconeogenesis:
        i. Liver contains enzymes for reconversion back into pyruvate → glucose for re-use as energy and/or storage as glycogen
        ii. This process consumes ATP and O_2 (measured as part of the O_2 debt)

4. Role:
    a. Anaerobic glycolysis: Prevents build-up of pyruvate, and regenerates NAD^+ which allows glycolytic pathway to continue.
    b. Limits anaerobic metabolism due to lactic acidosis in blood (anaerobic threshold). Thus, anaerobic metabolism by lactate production can only provide 60-90 seconds maximal muscle energy contraction.

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Physiol-08B10 Describe sepsis and describe the metabolic consequences of sepsis.

1. The systemic inflammatory response syndrome (SIRS) is the spectrum of disease characterised by two or more of the following:, Temperature > 38°C or < 36°C, HR > 90bpm, RR > 20bpm, WCC >12 x 10^9/L or <4 x 10^9/L
    a. Sepsis is SIRS resulting from infection of which there are varying degrees. Severe sepsis is associated with multi-organ dysfunction, hypoperfusion and/or hypotension (SBP < 100 systolic)
    b. Septicaemia is the systemic disease caused by spread of micro-organisms and their toxins via circulating blood.
2. System consequences:

System	Consequence	Mechanism
Cardiovascular	Vasodilatation, warm peripheries, hypotension Tachycardia	Bacterial endotoxin → damage vascular wall → release of inflammatory cytokines, mediators → vasodilatation, ↑ permeability → fluid extravasation → ↓SVR, intravascular volume → ↓MAP ↓MAP → baroreceptor reflex → ↑SNS
Respiratory	↑respiratory rate, hypoxaemia → ARDS	Alveolar capillary damage → ↑ permeability → fluid extravasation into interstitium → interstitial oedema → hypoxaemia (↓ventilation) Metabolic acidosis → respiratory compensation
Haematological	DIC: Coagulopathy, thrombocytopenia, leucocytosis or leucopenia, microvascular ischaemia.	Capillary endothelial damage → activation 1°/2° haemostasis, complement → platelet and coagulation factor consumption → microemboli → ischaemia. Release of inflammatory cytokines → migration leucocytes. Platelet, coagulation factor, leukocyte consumption → Coagulopathy.
CNS	Confusion CVA	Hypoxaemia, hypotension → ↓ cerebral perfusion → confusion Microvascular infarction → stoke

3. Metabolic consequences:

Consequence	Mechanism
Hypermetabolism	Infection, inflammation → pyrogens → alters posterior hypothalamic set-point setting → fever → ↑O_2 consumption for thermoregulation mechanisms
Anaerobic metabolism	Hypoxaemia (ARDS), ↓perfusion (hypotension) → ↓O_2 delivery to tissues → ↑ anaerobic metabolism by Embden-Meyerhof pathway and Cori cycle → ATP formation via pyruvate → lactate production.
Gluconeogenesis	Anaerobic state → unable to metabolise carbohydrates, fats and protein through Citric acid cycle and oxidative phosphorylation. Glycerol, proteins converted to pyruvate (Gluconeogenesis) → anaerobic conversion to lactate. ↑ glucagon, corticosteroids, catecholamines promote GNG. ↓ insulin → insulin resistance → ↑BSL
Catabolic state	↑energy demand, ↓O_2 supply → breakdown of body glycogen, fats, protein for use in anaerobic glycolysis.
Metabolic acidosis	Hepatic hypoperfusion → ↓ ability to regenerate pyruvate and glucose from lactate → build up lactate → lactic acidosis. System compensatory responses: • Chemical buffering (bicarbonate, protein) • Respiratory hyperventilation (mins → days) • Renal excretion of H^+ (days → weeks)

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Physiol-06B14/96A5/01B3 Compare and contrast the physiological effects of a six hour fast of fluids and food with a twenty four hour fast in a healthy adult.
Physiol-93A1 Discuss the physiological consequences of preoperative fasting (food and water) for 12 hours in a healthy young adult. Include in the discussion the effects on the body of this food and water deprivation and the physiological mechanisms that compensate for them.

Effect	**6 Hour Fast**	**24 Hour fast (and beyond)**
Food (Fuel): all tissues require energy sources for basic metabolic processes. This energy is sourced from food types (carbohydrates, lipids, proteins) which undergo metabolic pathways to produce ATP – the energy currency of the body.		
Scenario	Common – every day between dinner and breakfast	Uncommon – starvation, fasting for GI surgery
Energy requirements	BMR = 2000kcal/day 500kcal/6 hours	BMR = 2000kcal/day
Energy substrate	Glycogen Lactate (secondary)	Lipids (primary) – free fatty acids, glycerol Proteins (secondary) Lactate (secondary)
Metabolic pathways	*Glycogenolysis* converts glycogen → glucose *Glycolysis* converts glucose → pyruvate → production of energy by Krebs cycle and Oxidative phosphorylation. Glycogen stores in liver (100g) and skeletal muscle (400g) last 24 hours. Small amounts anaerobic metabolism by specialised cells (RBC, renal medulla) → lactate production (Embden-Meyerhof).	*Gluconeogenesis* converts free glycerol → pyruvate → glucose *ß-oxidation* converts free fatty acids → acetyl CoA → production of energy by Krebs cycle and Oxidative phosphorylation. (↑FFA released from adipose tissue by HSL) Acetyl CoA → ketone bodies when citric acid cycle is exhausted → circulate and used by tissues as energy substrates → acetyl CoA → citric acid cycle *Glucogenic transamination* converts amino acids (alanine) → pyruvate → GNG → production of energy by Krebs cycle and Oxidative phosphorylation. *Lactate* is formed by anaerobic glycolysis pyruvate → lactate (when ↓O_2 or oxaloacetate) → *Cori cycle* *GNG* → regenerates glucose for fuel. *3-4 days fasting: ↓glucagon levels → ↓GNG. ↑ reliance on lipids (glycerol → glucose, free fatty acids, ketones → acetyl CoA) and lactate (Cori cycle) ↓ reliance protein ↓ BMR 30%
Metabolic consequences	Normal BSL Metabolic homeostasis	↓BSL ↑ketones Lethargy to ↓energy demand
Hormone response	Fasting → glucose uptake by cells for fuel → ↓BSL → ↓Insulin (β-cells), ↑ adrenaline (inhibits insulin release) / cortisol (ACTH stress response), glucagon (α-cells). Adipose → ↓glucose uptake (GLUT-4), ↓LPL, ↑HSL (↑FFA release) Muscle → ↓glycogenesis, ↑glycogenolysis, ↓protein synthesis, ↑ FFA metabolism Liver → ↓glycogenesis, ↑GNG, glycogenolysis, amino acid catabolism, ketone body formation	
Hormone control	↓insulin, ↑ adrenaline, morning cortisol	↓↓insulin, ↑↑glucagon (peaks day 4 fasting), cortisol, catecholamines, GH (rises after 24-48 hours)
Clinical	Hunger,	Hunger, Lethargy

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BSc(Med) MBBS(Hons) FANZCA

features	Ongoing absorption of food from GIT	
Fluids		
Fluid requirements	40mL/kg/day 10mL/kg	40mL/kg/day
Physiological consequences	Minimal at 6 hours	↓circulating volume → ↓Blood pressure → ↓organ perfusion
Hormonal actions	Osmoreceptors: ADH (posterior pituitary) → ↑aquaporin-2 CD → ↑H_2O reabsorption → concentration of urine ADH → vasoconstriction Baroreceptors (low and high pressure) → ↑SNS: ↑RAS → ↑ renin → ↑ angiotensin II → vasoconstriction ↑aldosterone → Na/water retention in CD	
Hormonal control	↑ADH ↑adrenaline	↑↑ADH ↑↑adrenaline ↓ANP, BNP
Cardiovascular consequences	Mobilisation of fluid volume in sphlancnic and skin circulation Maintenance of BP	Tachycardia, ↓urine output, lethargy, ↓insensible fluid loss Usually maintenance of BP
Clinical features	Thirst	Thirst

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BSc(Med) MBBS(Hons) FANZCA

Physiol-07A16 Briefly outline the components of parenteral nutrition, explaining the rationale for the use of each component.

1. Parenteral nutrition: the feeding of an individual intravenously, bypassing the normal enteral route of feeding.
2. Rationale for use:
    a. The use of parenteral nutrition is recommended when caloric requirements cannot be obtained from the enteral route. Often, parenteral nutrition is used to supplement poor oral intake. Occasionally, total parenteral nutrition (TPN) is required for patients who have a prolonged period of no oral intake. Enteral feeding is preferred.
    b. Indications:
        i. Non-functioning GIT (paralytic ileus)
        ii. Pre-emptively in malnourished patients in whom intestinal use is not expected within 7 days of surgery
        iii. Severe mucositis due to chemotherapy
        iv. Severe short-bowel syndrome

3. Components:

Component	**Normal Daily Requirement**	**Rationale**
Water	40mL/kg + losses	Fluid volume loss due to urine output, faeces, drains, fistulas, stomas, wounds. Isotonic solution required.
Electrolytes	Na 1-2 mmol/kg/day K 0.7-1.0 mmol/kg/day PO_4 0.4 mmol/kg/day Ca 0.1 mmol/kg/day Mg 0.1 mmol/kg/day	Renal losses of electrolytes.
Fuel	30kcal/kg/day BMR	20% protein 30% carbohydrate 50% lipid
Carbohydrates	30% calories 3g/kg/day = 1200kcal	Minimal requirement of 150g for brain metabolism 500mL 50% dextrose Modify in diabetics
Lipids 30	50% calories 1.5g/kg/day Intralipid	100g lipid
Protein 20	Amino acids 0.5-1g/kg/day < 3g/kg/day	Essential amino acids Normal cell function, enzyme levels Musculoskeletal integrity (muscle mass)
Vitamins	Vit K 10mg Multivitamins	Essential Vitamins
Minerals	Zn, Cu, I, Mn, Se	
Glutamine		Essential immunonutrient derived from muscle protein breakdown. Given in high risk patients with immunocompromise.

4. Factors affecting nutrition needs:
    a. Age: ↑ requirements (Harris Benedict equation)
    b. Sex: ↑ requirements (Harris Benedict equation)
    c. Desired ↑ weight → ↑ TPN 30%
    d. Disease:
        i. Burns 40% increase
        ii. Sepsis 30% increase

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MAKE-UP: Briefly describe the important features of carbohydrate metabolism

1. Glucose is the final common pathway for transport of all carbohydrates into tissue cells.

2. Storage: upon entering tissue cells, glucose is preferentially stored as glycogen – a large polymer of glucose. Muscle and liver is especially adept at converting glucose → glycogen.
    a. Glycogenesis: the process of glycogen formation from glucose-6-phosphate.
    b. Glycogenolysis: breakdown of cells glycogen stores to re-form glucose by phosphorylase. Stimulated by adrenaline and glucagon.

3. Aerobic Energy release: one mole of glucose releases **38 moles ATP** in several steps of carbohydrate metabolism.
    a. Glycolysis: splitting of glucose → 2 x pyruvate
        i. 10 steps, net gain **2ATP**
        ii. Input: 2ATP
        iii. Output: 4ATP + 4H
    b. 2Pyruvate + 2Co-enzyme A → 2acetyl CoA + $2CO_2$ + 4H
        i. Formation of $2CO_2$ + 4H
    c. Citric acid cycle:
        i. Occurs in matrix of mitochondria
        ii. Input – $6H_2O$ + 2 acetyl-CoA
        iii. Output – $4CO_2$ + 16H + 2Co-A + **2ATP**
        iv. Hydrogen atoms: 24H released by glycolysis + citric acid cycle. 20 of these atoms combine with intermediates
            1. $NAD^+ + H \rightarrow NADH$
            2. $FADH + H \rightarrow FADH_2$
    d. Oxidate phosphorylation: accounts for 90% of total ATP formed from glucose metabolism, occurs in mitochondria. 20H from $NADH/FADH_2$ and 4H (freely released) are used to provide electrons.
        i. Splitting $H \rightarrow H^+ + e^-$
        ii. $4e^- + 4H^+ + O_2 \rightarrow 2H_2O$ (electron transport chain – several intermediaries which accept and then donate electrons. The last and most important is cytochrome oxidase)
        iii. Energy released by electron transport is used to pump H^+ from matrix → intermembrane space (sets up concentration gradient).
        iv. Energy released by flow of H^+ down gradient through ATP synthetase which catalyses ADP → ATP. 3 ATP molecules for every $2e^-$ transported. 66% efficiency.

4. Anaerobic Energy release: occurs when no O_2 available for oxidative phosphorylation, thus end-products of glycolysis and citric acid cycle build up → ↑pyruvate + NADH
    a. Energy inefficient – 3% efficient

b. Anaerobic glycolysis – pyruvic acid converted to lactic acid which diffuses out of cells to ↓pyruvic acid concentration → allows glycolysis to continue.

$$\mathbf{pyruvate + NADH + H^+ \leftrightharpoons lactic\ acid + NAD^+}$$

c. When O_2 source returns, Lactic acid converted by to pyruvate + NADH + H^+ → forms ATP by oxidative phosphorylation → ATP used to convert pyruvate → glucose. This occurs in the liver.

5. Hexose monophosphate shunt: minor mechanism for the oxidation and breakdown of glucose → energy.
   a. Important in liver cells and adipose tissue, where it provides energy independent of citric acid cycle.

$$G-6-P \Leftrightarrow 6PG \Leftrightarrow Ribose-5-phosphate \Leftrightarrow F-6-P$$

   b. Overall:

$$glucose + 12NADP^+ + 6H_2O \Rightarrow 6CO_2 + 12H + 12NADPH$$

      i. NADPH used for fat synthesis from carbohydrates → glucose stored as fat energy
      ii. H used for oxidative phosphorylation → ATP

6. Formation of glucose from fats, carbohydrates, lactate: when glycogen stores fall below normal → derive glucose from amino acids (protein) and glycerol (fat)
   a. Gluconeogenesis:
      i. 60% amino acids by transamination, deamination → pyruvate
      ii. Lactate → pyruvate
      iii. Glycerol → succinyl Co-A → pyruvate
   b. Stimulated by glucagon and steroids

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MAKE-UP: Briefly describe the important features of lipid metabolism

1. Lipids in involved in body metabolism are obtained from the diet and include:
    a. Triglycerides – 3 fatty acids, glycerol backbone
    b. Phospholipids
    c. Cholesterol

2. Lipid transport
    a. Digestion – splitting of triglycerides into monoglycerides and free fatty-acids
    b. Absorption – inside intestinal epithelial cells, resynthesis into triglycerides and packaged as chylomicrons which circulate in the venous system for transport to the liver and adipose tissue
    c. Storage: *Lipoprotein lipase* hydrolyses TGs and phospholipids in chylomicrons → FFAs and glycerol → diffuse into fat (adipose cells) and the liver (hepatocytes) → resynthesised as TGs inside the cells for storage.
    d. Transport: via synthesised lipoproteins which contain variable amounts of TGs, cholesterol, phospholipids and protein. These are formed mostly in the liver, with small amounts of HDL formed in the intestinal epithelium during absorption. The lipoproteins transport and deposit lipid components in the blood and peripheral tissues.

Lipoprotein	Size (nm)	TG	Phospholipid	Cholesterol	Protein	Origin
Chylomicron	75-1000	90	3	5	2	Intestine
VLDL	30-80	55	17	20	8	Liver, intestine
IDL	25-40	40	20	30	10	VLDL
LDL	20	6	21	53	20	IDL
HDL	7.5-10	5	25	20	50	Liver, intestine

    e. Synthesis: the liver can synthesise TGs from carbohydrates; and also cholesterol and phospholipids from FFAs.
    f. Mobilisation: stored fat is hydrolysed by *hormone sensitive lipase* → released and transported as glycerol + FFAs → ionised and carried on albumin. Activation by starvation and diabetes (when carbohydrate metabolism is low).

3. Lipid Metabolism: lipids are oxidised by peripheral tissues to yield energy.
    a. Glycerol metabolism: glycerol converted to glycerol 3-phosphate which enters glycolytic pathway.
    b. Fatty acids: are polymers of acetic acid which undergo β-oxidation in mitochondria to produce many acetyl CoA, with reduction of carbon chain length by 2. Overall, this reaction releases:
        i. H atoms in free F, NADH, $FADH_2$ used for oxidative phosphorylation.
        ii. Acetyl CoA → produces energy via Krebs cycle. Overall, for each mole stearic acid oxidised completely → 146 moles ATP formed.
    c. Ketone bodies: excess acetyl Co-A forms ketone bodies which recirculate from the liver.

$$Acetyl\ CoA + Acetyl\ CoA \Rightarrow acetoacetic\ acid$$
$$\Rightarrow acetone, \beta - hydroxybutyric\ acid, acetic\ acid$$

    These ketone bodies diffuse into liver cells and can be transported to peripheral tissues. Reverse reactions allow further formation of acetyl CoA → Krebs cycle.

$$acetone, \beta hydroxybutyric\ acid, acetic\ acid \Rightarrow acetoacetic\ acid \Rightarrow Acetyl\ CoA$$

    Levels of ketones rise during starvation, diabetes and high fat-content meals as there is a paucity of carbohydrate metabolism. Ketone bodies accumulate when oxaloacetate is exhausted, and cannot combine with Acetyl-CoA to enter the Krebs cycle.

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4. Synthesis of TGs from carbohydrates: high amounts of carbohydrate meals are used immediately, stored as glycogen and also converted to TGs for storage in adipose tissue. This is important as glycogen storage is limited. This process is promoted by insulin.

$$glucose \Rightarrow acetyl\ CoA\ (glycolysis)$$
$$acetyl\ CoA \Rightarrow malonyl\ CoA$$
$$acetyl\ coA + malonylcoA \Rightarrow stearic\ acid$$
$$glycerol \Rightarrow glycerol\ 3\ phosphate \Rightarrow glucose$$

The stearic acid grows in carbon chains and combines with glycerol → triglycerides.

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MAKE-UP: Briefly describe the important features of protein metabolism

1. Proteins in involved in body metabolism are in the form of 20 different amino acids – chemicals with an amino end (-NH_2) and a carboxyl end (COOH). Proteins are chains of amino acids which can link between the amino and carboxyl groups.

2. Source: proteins are derived from the diet -
    a. Digested in the small intestine and stomach into amino acids
    b. Absorbed into the intestinal cells by facilitated diffusion

3. Transport:
    a. Amino acids circulate freely in their ionised state in the plasma, and contributed to negative ion load.
    b. Plasma proteins circulate and can act as a reservoir for use when tissue proteins are low. There are 3 major forms of plasma protein –
        i. Albumin: provides colloid osmotic pressure
        ii. Globulin: natural and acquired immunity, enzymatic function
        iii. Fibrinogen: coagulation and secondary haemostasis

4. Storage: upon entering cells, amino acids combine with one another to form peptide linkages → cellular proteins. Most cellular proteins can be decomposed again into amino acids for release into the circulation when protein levels drop. The liver is a large storage organ for protein, but most other cells can storage and release protein.

$$amino\ acid\ circultion\ \Leftrightarrow protein\ storage$$

This equilibrium is under hormonal control.

5. Synthesis:
    a. Albumin, fibrinogen and 50-80% globulins are synthesised in the liver from amino acids.
    b. 10 amino acids are essential; 10 other amino acids are synthesised in cells from α-keto acids

6. Metabolism
    a. *Deamination* – the conversion of amino acids to stored fat or glycogen. This occurs when there is excess amino acids in cells. Involves the removal of amino groups, and formation of ammonia.
    Deamination → produces NH4 → urea → excretion by kidneys
    b. *Transamination* – interconversion to other amino-acids, carbohydrates and fat, involving the transfer or removal of amino acid groups.
        i. Ketogenic: → other amino acids leucine, isoleucine
        ii. Glucogenic: alanine → pyruvate

$$pyruvate\ \Rightarrow glycogen$$
$$pyruvate\ \Rightarrow acetyl\ CoA\ \Rightarrow fatty\ acids, ketones$$
$$pyruvate\ \Rightarrow citric\ acid\ cycle$$

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MAKE-UP: Briefly describe the role of ATP in metabolism

1. ATP (adenosine triphosphate) is a chemical compound present in the cytoplasm and nucleus of all cells, and acts as the energy currency of the body. All energy required from cellular operation is derived from ATP, and food in cells is oxidised to replenish ATP stores.

2. Structure: adenosine + ribose + 3 phosphate bonds
    a. Each phosphate bond stores energy which is released when broken – 12,000 calories.