

Synthesis of	From	Starting point	Catalyzed by	Where	Committed step	Rate Limiting	Catabolism	Catalyzed by	Products	For
Pyrimidines Uracil Cytosine thymidine	De novo Aspartate Glutamine CO <sub>2</sub>	Formation of Carbonyl phosphate from gln, CO <sub>2</sub> , ATP	Carbonyl Phosphate Synthetase II	Cytoplasm liver	aspartate transcarbamolase catalyzes aspartate to carbamoylaspartate	aspartate transcarbamolase catalyzes aspartate to carbamoylaspartate	Seq hydrolysis Ring opened NH <sub>3</sub> to urea		Malonyl CoA Methylmalonyl CoA → succinyl CoA	FA syn Krebs
Purines Adenine Guanine (produces dGTP for DNA syn)	De novo Folate Glutamine Aspartate Glycine CO <sub>2</sub>	Ribose 5-P from hexose monophosphate shunt			Ribose 5-P reacts with ATP & glutamine to form PRPP		Oxidized in liver	Xanthine dehydrogenase	Uric acid (to urine)	
AA				Liver	Note: Catabolism of AA in liver – most periportal, except glutamate & aspartate. BCAA in muscle.  Note: Pro not stored. Decr pro, decr urea N excretion. Incr pro, incr urea & incr water loss.  Note: Ordinary Careless Crappers Are Also Frivolous About Urination	Note: Catabolism of AA in liver – most periportal, except glutamate & aspartate. BCAA in muscle.  Note: Pro not stored. Decr pro, decr urea N excretion. Incr pro, incr urea & incr water loss.  Note: Ordinary Careless Crappers Are Also Frivolous About Urination	Removal & disposal of amino group by: <b>Deamination</b> H <sub>3</sub> N removed as ammonia Requires B6  Transamination Impt for syn of nonessential AA.  Catabolism of C-skeleton (energy, glucose, ketone bodies, Cholesterol, FA) (Complete oxidation if diets inadeq. in energy)  Disposal of Ammonia <b>1. Ureagenesis</b> O Ornithine C Carbamoyl phosphate C Citrulline A Aspartate A Arginine succinate F Fumurate A Arginine U Urea <b>2. Glutamine synthesis</b>	Dehydratases Lyases Dehydrogenase  Amino transferase (B <sub>6</sub> ) AST (heart) ALT (organ)  Carbonyl phosphate synthetase I	NH <sub>3</sub>  NH <sub>3</sub> + C-skeleton/ α-keto acid  Energy (ATP) CO <sub>2</sub> /HCO <sub>3</sub> <sup>-</sup> , ammonia	N to urea

				Where			Catabolism		Products	For
<u>Metabolism of C-skeleton</u> Glucogenic	Accelerate by glucagon, insulin, cortisol (when not enough CHO, infection, trauma, DM, liver ds.)			Liver kidney	Can undergo gluconeogenesis in liver or kidney	Cholesterol (from acetyl CoA)  FA prod (from acetyl CoA w/ Acetyl CoA carboxylase) if excess energy & pro w/adequate CHO intake.			Can be degraded to pyruvate or intermediates:  Fumarate Succinate Succinyl coA A-ketoglutarate oxaloacetate	Krebs
Metabolism of C-skeleton  Ketogenic Leu & lys the only totally ketogenic AA	Acetyl CoA or acetoacetate	Leu & lys go to acetyl CoA Leu generates HMG CoA					AA catabolized to ketone bodies during inadequate CHO intake		Ketone bodies	
Hepatic metabolism of aromatic AA (has phenol ring)	Phe, Tyr, Trp Partially glucogenic is degraded to fumarate. Ketogenic is catabolized to acetate	Phe can be converted to Tyr (needs/B6) Genetic deficiency of enzyme leads to PKU.  Tyrosine also convert to dopamine & catecholamines (norepi & epi) Thyroid hormones, melanin	Phenylalanine monoxygenase  Trp also converted to serotonin/melatonin  Requires Fe, Cu, Vit C	Liver kidney						

Synthesis of	From					Products				
<u>Tryptophan metabolism</u>	Pyruvate Acetyl CoA NAD, NADPH Serotonin Melatonin	Glucogenic (pyruvate & acetyl coA) Ketogenic  Krebs  Body temp, appetite, anger, aggression Circadian Rhythms, Sleeping patterns				Pyruvate Acetyl CoA NAD & NADPH				
S-containing AA  Methionine Cysteine	Generates Cys, Tau, SAM	SAM is principal methyl donor in body, req'd for syn of carnotine, Creatine, Epinephrine purines, Nicotinimide		Liver	Elevated homocysteine levels increased risk factor for CVD, may develop due to low folate, Vit B <sub>12</sub> or B <sub>6</sub>	Products: Pyruvate (glucogenic) Taurine --> bile Propionyl CoA for β-oxidation of odd # C FA Succinyl CoA --> Krebs  Requires: B6, B12, folate. If low, increased homocystine (CVD, osteoporosis) bec. Can't convert back to Methionine.				
Branched chain AA  Ile, Leu, Val	C-skeleton			Liver minor role in initial metab.	Normally remain in circ., taken up and transaminated by skeletal muscle, heart, kidney and adipose (if needed).  Utilized within tissues, released to circ. Or go back to liver.					



Choline	serine	Acetylcholine Lecithin springomylin	VLDL (fatty liver if not enough choline to make VLDL)							
Metabolism										
Lysine	Liver Totally ketogenic Used for syn of carnitine									
Threonine	Used to syn body pro & high quantities in mucus glycoproteins Metabolized by 3 pathways: 1. Cytosolic threonine dehydratase to succinyl coA (common) 2. Mitoch. Threonine dehydrogenase to pyruvate (conc high) Mitoch. Threonine complex converts threonine to glycine					Threonine, glycine, serine Metab are interrelated.				
Arginine	Catab. Mostly in liver & kidney In kidney, used w/glycine in 1 <sup>st</sup> reaction of creatine syn. In liver, generates urea as part of urea cycle, & ornithine Used for nitric oxide prod. In endothelial cell, cerebellar neurons, neutrophils.									
Histidine	May be catab. To form glutamate. May combine w.β-alanine to form carnosine. Can form histamine – Vit B6 dep decarboxylation									
Glycine & Serine	Produced from one another – req. folate. Hepatic metab. Gly to ser mainly in kidney. Gly needed for syn of creatine, porphyrins, sarcosine, glycocholate Ser used for syn of ethanolamine & choline									
Arg, Glu, Pro, His	Arginine, Glutamate, Proline, & Histidine are Interacting									
Alanine	Impt in intertissue transfer of amino groups generated from AA catab. May travel from muscle to liver. Produce glutamate – may be deaminated to yield ammonia for urea cycle. Can be converted to glucose ( <b>alanine-glucose cycle</b> ) – transport N to liver for conversion to urea while also generating needed substrate. Occurs in low CHO stores (liver glycogen) to maintain blood glucose; eExcessive use for glucose.									
Skeleton muscle & flow of AA	~40% of body pro – in skeletal muscle. AA uptake readily occurs after meal. Asp, asn, glu, leu, ile, val – catabolized more in skeletal muscle. Possess Branched chain amino transferase locted in cytosol & mitochondria – for transaminatio nof BCAA. A-keto acid of BCAA may remain in muscle for further oxidation or transported bound to albumin in blood to other tissues.									
BCAA Metab	Ile, leu, Val – maple syrup urine disease Ile – propionyl CoA carboxylase - -> propionic academia - methylmalonyl CoA mutase --> methylmalonic acedemia									

