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Surplus aminoacids are used as metabolic fuel. The major site of amino acid degradation is the liver. Pathways leading to amino acid degradation are quite alike in most organisms. As is the case for sugar and fatty acid catabolic pathways, the processes of amino acid degradation converge on the central catabolic pathways for carbon metabolism. However, one major factor distinguishes amino acid degradation from other catabolic processes, i.e. every amino acid contains an amino group. As such every degradative pathway passes through a key step in which α-amino group is separated from the carbon skeleton and shunted into the specialized pathways for amino group metabolism.

Two amino acids, glutamate and its amide form glutamine, play crucial roles. Amino groups from amino acids are generally first transferred to a α-ketoglutarate in the cytosol of liver cells to form glutamate. Glutamate is then transported into the mitochondria. In muscle, excess amino groups are generally transferred to pyruvate to form alanine. Alanine is another important molecule in the transport of amino groups, transporting them from muscle to the liver.There is no net deamination in such reactions The effect of transamination reactions is to collect the amino groups from many different amino acids in the form of only one, namely L-glutamate. Cells contain several different aminotransferases, many of which are specific for αketoglutarate as the amino group acceptor. The catalytic versatility of pyridoxal phosphate (PLP)enzymes is remarkable. The PLP enzymes catalyze a wide range of amino acid transformations including decarboxylations, deaminations, racemizations and aldol cleavages. Glutamate is transported from the cytosol to the mitochondria, where it undergoes oxidative deamination catalyzed by L-glutamate dehydrogenase (GD). GD can employ NAD+ or NADP+ as cofactor and is allosterically regulated by GTP and ADP. The combined action of the aminotransferases and GD is referred to as transdeamination. A few amino acids bypass the transdeamination pathway

and undergo direct oxidative deamination. Alanine also plays a special role in transporting amino groups to the liver in a nontoxic form by glucose— alanine cycle. In muscle and certain other tissues that degrade amino acids for fuel, amino groups are collected in glutamate by transamination . Glutamate may then either be converted to glutamine for transport to the liver, or it may transfer its α-amino group to pyruvate, a readily-available product of muscle glycolysis, by the action of alanine aminotransferase. Alanine passes into the blood and is carried to the liver. As in the case of glutamine, excess nitrogen carried to the liver as alanine is ultimately delivered as ammonia in the mitochondria. During a reversal of this alanine aminotransferase reaction, alanine transfers its amino group to α-ketoglutarate, forming glutamate in the cytosol. Some of this glutamate is transported into the mitochondria and acted uponby glutamate dehydrogenase, releasing NH_4^+. Alternatively, transamination with oxaloacetate movesamino groups from glutamate to aspartate, another nitrogen donor in urea synthesis. Vigorously contracting skeletal muscles operate anaerobically and produce not only ammonia from protein degradation but also large amounts of pyruvate from glycolysis. Both these products must find their way to the liver—ammonia for its conversion into urea for excretion and pyruvate for its incorporation into glucose and subsequent return to the muscles. Thus two problems are solved with the glucose—alanine cycle.