Glutamine

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Proliferating cancer cells rely largely on glutamine for survival and proliferation. Glutamine serves as a carbon source for the synthesis of lipids and metabolites via the TCA cycle, as well as a source of nitrogen for amino acid and nucleotide synthesis. To date, many studies have explored the role of glutamine metabolism in cancer, thereby providing a scientific rationale for targeting glutamine metabolism for cancer treatment.

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Hopefully devoted to Q: targeting glutamine addiction in cancer 2017

Over the past 30 years, increasing evidence has shown that many tumours require glutamine as well as glucose for their proliferation and survival.
Until recently, the majority of studies focused on changes in glucose flux, specifically the increased conversion of glucose to lactate (reviewed by Liberti and Locasale (2016)). The observation that mammalian cells rely on both glucose and glutamine (Reitzer et al, 1979; Moreadith and Lehninger, 1984; Board et al, 1990; Yuneva et al, 2007) shifted the focus to a more diverse range of pathways that are rewired in many tumours. Although glutamine is required as an alternative fuel for the tricarboxylic acid cycle (TCA) to produce ATP, it also contributes to a wide range of pathways in cells, producing amino acids, nucleotides and fatty acids as well as playing an important role in reactive oxygen species (ROS) homeostasis, mTOR activation and the hexosamine biosynthesis pathway (Figure 1).
Glutamine is used in the production of glutathione, which helps maintain the redox balance.
ROS homeostasis
Glutamine is also involved in maintaining the redox balance. Reactive oxygen species can be both pro-tumorigenic, through ROS-mediated cell signalling, and highly damaging when in excess (reviewed by Liou and Storz (2010)). Reactive oxygen species are produced from several sources, including the mitochondrial ETC, which leaks electrons to oxygen to generate superoxide. Thus, increased glutamine oxidation can correlate with increased ROS production. However, a number of pathways involved in glutamine metabolism produce products that directly control ROS levels. For instance, glutamine is used to synthesise glutathione, a tripeptide (glu–cys–gly) that neutralises peroxide free radicals. Glutamine also regulates ROS homeostasis through the production of NADPH via GLUD, as well as producing NADPH using malic enzyme 1 (Son et al, 2013).