Glioma stemness refers to the ability of glioma cells to self-renew and differentiate into various cell types within the tumor. This property is similar to that of normal stem cells, which have the ability to self-renew and differentiate into various cell types within the body.

Glioma stem cells are believed to play a critical role in the initiation, progression, and resistance to therapy of gliomas. They have been shown to be more resistant to chemotherapy and radiation than non-stem cells in gliomas. Therefore, understanding the molecular mechanisms underlying glioma stemness is important for developing effective therapies for this devastating disease.

Several factors have been identified as regulators of glioma stemness, including transcription factors, signaling pathways, and microRNAs. For example, the transcription factors SOX2, OCT4, and NANOG have been shown to promote glioma stemness by regulating the expression of genes involved in stem cell self-renewal and differentiation. In addition, signaling pathways such as Notch and Hedgehog have also been shown to play a role in glioma stemness.

Targeting glioma stem cells is an active area of research, with the goal of developing new therapies that can eliminate these cells and improve patient outcomes.