Collagen I is the primary extracellular matrix component of solid tumors and influences metastatic progression. Collagen matrix engineering techniques are useful for gaining insight into how biophysical features of this complex biomaterial regulate cancer cell behavior. Here, we present a new approach to tune collagen fibril architecture without changing matrix density and stiffness by using PEG as an inert molecular crowding agent during gelation and cell embedding. Combining this technique with traction force microscopy, we show that fibril architecture regulates traction and spreading of MDA-MB-231 breast cancer and HT-1080 fibrosarcoma cells. In matrices where fibers are short and pore size is small, cells are confined, fail to stabilize protrusions, and exhibit limited ability to pull on the matrix. Likewise, FAK phosphorylation is low, suggesting that adhesion is limited. Higher reactive oxygen species levels, lowered glucose consumption, and downregulation of mTOR signaling trigger oxidative and metabolic stress consistent with a low-adhesion state. Thus, despite being surrounded by matrix, cells experience a low-adhesion state when confined. In response, cells upregulate Notch signaling and switch into a collective migration and morphogenesis program associated with the expression of cell-cell adhesion proteins, e.g. ICAM1. Most cells form invasive networks reminiscent of aggressive collective migration in tumors, but ~10% of MDA-MB-231s form gland and lobule structures reminiscent of normal breast epithelia. In contrast, cells in non-confining matrices maintain adhesion and migrate as single cells. This study deepens our insight into how the extracellular matrix modulates cancer cell migration and morphogenesis behaviors independently of stiffness and density.