The adipose cells in obese individuals are both greater in number and size than in lean individuals, and the tissue contains a much higher percentage of macrophages. As a result of storing excessive amounts of fat, the stressed adipose cells release inflammation-inducing factors and undergo apoptosis. Both outcomes activate macrophages in a traditional M1 inflammatory state in which they release tumor necrosis factor-α (TNF-α), which recruits and activates additional immune cells to the site. This low level sustained inflammation causes tissues to become resistant to insulin, the first step in developing diabetes.