Extracellular zinc is known as a potent endogenous modulator of a variety of ion channels including many types of voltage-gated potassium channels. Here we report on the inhibitory effect of zinc on voltage-gated Kv1.3 channels in human T lymphocytes (TL). The application of 10 and 20 µM Zn caused a concentration-dependent shift of the activation midpoint of the whole-cell currents from $-19.65 \pm 1.03$ mV (mean±S.E.) under control conditions to $9.84 \pm 0.66$ mV upon application of 20 µM Zn. This effect was saturated at zinc concentrations higher than 20 µM. The activation shift was accompanied by a considerable slowing of the activation rate, whereas the channel closing rate was not significantly affected upon zinc treatment. The inactivation midpoint also shifted from $-53.06 \pm 0.44$ mV under control conditions to $-36.05 \pm 0.48$ mV upon application of 100 µM Zn, whereas the channel inactivation rate was not significantly affected upon Zn treatment. The whole-cell potassium currents were reduced to about 70% of their control values, but this effect showed no clear concentration dependence in the zinc concentration range from 10 to 100 µM. By contrast, raising the zinc concentration to levels above 100 µM produced an inhibition of the whole-cell currents in a concentration-dependent manner. The channels were half-blocked at zinc concentration of 277 µM and the Hill slope coefficient was calculated to be 1.35±0.11. The inhibitory effect of zinc was not complete at micromolar concentrations - raising the zinc concentration to 2.6 mM blocked ca. 82% of the currents. The remaining current was shown to be the potassium current. This inhibitory effect was neither accompanied by a shift of the activation and inactivation midpoints nor by a slowing of the channel activation rate. Our results demonstrate that extracellular zinc inhibits the activity of TL Kv1.3 channels at micromolar concentrations. It is suggested that zinc acts on two independent binding sites on the channels. Binding to one site that is saturated at concentrations higher than 20 µM affects the channel gating. Binding to another site, which occurs at concentrations higher than 100 µM, produces an additional inhibitory effect on the currents without affecting the channel gating.