During hibernation, some mammals show low body temperatures (< 10 °C). Tissues from hibernators exhibit cold resistance even when the animal is not hibernating. Mice can also enter hypothermic fasting-induced torpor (FIT), but the cold resistance of FIT has never been related to their tissues. Here, we show that an inbred mouse STM2 exhibits lower body temperature during FIT than C57BL/6J or MYS/Mz. Thus, STM2 resists the cold more than other strains. Analysis of strain-specific mouse embryonic stem (ES) cells shows that STM2 ES cells are more cold-resistant than others, rely on the oxidative phosphorylation (OXPHOS) pathway but respire independently of the electron transfer chain complex I in the cold. We also found that the liver of STM2 uses OXPHOS more in cold than other strains. This study demonstrates that an organismal phenotype associated with torpor can be effectively studied in an *in vitro* setup using mouse cells.