Receptor-like kinases (RLKs) are known to be involved in the recognition of pathogen-associated molecular patterns (PAMPs) and subsequently activate resistance pathways against broad classes of pathogens. While initiation and maintenance of defense pathways is critical for survival, mechanisms to damp down these responses are just as necessary though currently not as well understood. We have identified \textit{CRLK1}, an Arabidopsis RLK that is highly induced by chitin at early time points and localizes to the plasma membrane. Knock-out mutants in \textit{crlk1} are more susceptible to both biotrophic and necrotrophic fungal pathogens though the response of the mutants to bacterial pathogens is unaffected. Interestingly expression of \textit{MAPK3}, an important positive regulator of innate immunity, is increased in \textit{crlk1} mutants. Our data show that CRLK1 is essential for the establishment of defense against biotrophic and necrotrophic fungi and that the mutation in \textit{CRLK1} does not fully block chitin-enhanced Arabidopsis resistance. We show that CRLK1 is a functional kinase \textit{in vitro} and its kinase activity required the presence of manganese. Overexpression of a 35S:CRLK1::GUS fusion protein in Arabidopsis confers enhanced resistance to the powdery mildew pathogen \textit{Golovinomyces cichoracearum}. In addition, \textit{CRLK1} induction by chitin is increased in \textit{mapk3} and several \textit{wrky} mutants indicating that \textit{CRLK1} may be repressed by \textit{MAPK3} and \textit{WRKY} transcription factors \textit{in planta}. The results presented provide important information about the function and regulation of CRLK1 in Arabidopsis.