The formation of spores is critical for survival of *Clostridium difficile* outside of the host gastrointestinal tract. Persistence of *C. difficile* spores greatly contributes to the spread of *C. difficile* infection (CDI), and the resistance of spores to antimicrobials facilitates the relapse of infection. Despite the importance of sporulation to *C. difficile* pathogenesis, the molecular mechanisms controlling spore formation are not well understood. The initiation of sporulation is known to be regulated through activation of the conserved transcription factor, Spo0A. Multiple regulators influence Spo0A activation in other species; however, many of these factors are not conserved in *C. difficile*, and few novel factors have been identified. We investigated the function of a protein, CD1492, which is annotated as a kinase and originally proposed to promote sporulation by directly phosphorylating Spo0A. We found that deletion of *CD1492* results in increased sporulation, indicating that CD1492 is a negative regulator of sporulation. Deletion of CD1492 also resulted in decreased virulence in the hamster model of CDI. Further, the *CD1492* mutant demonstrated effects on gene expression that are not associated with Spo0A activation, including lower *sigD* and *rstA* transcription, suggesting that this protein interacts with factors other than Spo0A. Altogether, the data indicate that CD1492 negatively affects sporulation and positively influences motility and virulence. These results provide further evidence that *C. difficile* sporulation is regulated differently from that of other endospore forming species.