        - After octanoyl is passed to an acceptor domain by LipB, it is altered to form lipoic acid, a fatty acid essential to the metabolism of *Mycobacterium tuberculosis*.
    - Active site: Cys 176 and Lys 142(nuc in acid/base reaction)
    - Mutated Lip B: without key residues, protein doesn't work.
- **Fts Z**
    - Dimer → subunit a has important stuff (not b).
    - Involved in bacterial cell division which leads to tuberculosis (TB) growth
    - A polymer of FtsZ units is the Z-ring:
        - Contraction of Z-ring leads to cytokinesis, and subsequent splitting of the daughter cells
    - T3 Loop and binding of GTP:
        - The T3 loop begins in an open state, without GTP bound on subunit A
        - The T3 loop then binds GTP_y at the y-phosphate
        - Binding induces conformational changes causing the T3 loop to convert from the open to closed state (which is more stable)
        - *This allows the T3 loop to adopt a straight longitudinal dimer conformation.*
        - Interactions with the T7 loop at the GTPase site on neighboring subunit B causes GTP hydrolysis, which creates a bent longitudinal dimer form
        - *This generates the contraction force necessary for cytokinesis*

- **Ddl**
    - Composed by two amino acid chains
    - Joins 2 molecules of D-al together → helps bacteria make cell wall.
    - DCS→ anti TB drug: looks like d-al→ fit in
    - Binding site
        - 1) Arg 316, Glu 336, Gly 123,
        - 2) Tyr 277, Met 342, Ser 341
    - Nitrogen involved in hydrogen bonding
    - Bits of the protein move constantly
- **Rip A and Rip B**
    - Help bacteria replicate/ divide → cleave filament →splitting up cell wall so bacteria can split into two (catalytic triad to break peptide bond)
    - Rip A posses a larger subunit, has N terminal (functions better)
        - Cys 383 → nucleophile
        - His 432→ acts as a base (nitrogen)
        - Glu 444
    - Rip B (hydrophobic) may have mutated from a duplication of Rip A
        - Cys 152