CLAIMS

What is claimed is:

1. Use of a genetically modified donor bacterium configured to express a heterologous molecule in the manufacture of a medicament for the treatment of Early Mortality Syndrome (EMS) causing bacterial pathogen of a species of *Vibrio* in shrimp comprising:

- a genetically modified donor bacterium configured to express one or more of the following:

  - a heterologous anti-sense RNA (asRNA) polynucleotide that is complementary to the mRNA of an essential gene of an EMS-causing bacterial pathogen wherein said heterologous asRNA polynucleotide comprises SEQ ID NO. 3 or a sequence having approximately 85% or more homology with SEQ ID NO.: 3; or
  - a first heterologous quorum quenching molecule configured to inhibit one or more exogenous autoinducer-1 (AI-1) molecules, encoded by a heterologous *aidH* gene wherein said heterologous *aidH* gene comprises a nucleotide sequence according to SEQ ID NO. 1, or a sequence having approximately 75% or more homology with SEQ ID NO. 1; or
  - a second heterologous quorum quenching molecule configured to remove exogenous autoinducer-2 (AI-2) molecules from the environment and transport them into said genetically modified donor bacterium is encoded by an lsr operon operably linked to a promotor that is configured to express at least one ATP-binding cassette transporter (ABC transporter) and at least one chaperone protein that actively pump exogenous autoinducer molecule AI-2 wherein said lsr operon comprises the nucleotide sequence according to SEQ ID NO.: 2 or sequence having approximately 75% or more homology with SEQ ID NO. 2; and

- wherein said genetically modified donor bacterium is to be administered to a target host that is infected by, or susceptible to infection by said EMS-causing bacterial pathogen.

2. Use of the genetically modified donor bacterium of claim 1, wherein said shrimp comprise shrimp raised in aquaculture.

3. Use of the genetically modified donor bacterium of claim 1 wherein said first heterologous quorum quenching molecule configured to inhibit one or more exogenous AI-1 molecules comprises a homoserine lactonases (AHL lactonase) configured to inactivate exogenous AI-1 molecules.

4. Use of the genetically modified donor bacterium of claim 3 wherein said AHL lactonase configured to inactivate exogenous AI-1 molecules comprises an AHL lactonase configured to inactivate exogenous AHL class QS molecules.

5. Use of the genetically modified donor bacterium of claim 4 wherein said exogenous AHL class QS molecules comprise exogenous Hal-1 molecules.

6. Use of the genetically modified donor bacterium of claim 1 wherein said genetically modified donor bacterium comprises a genetically modified donor microorganism selected from the group consisting of: a symbiotic genetically modified donor bacteria, a shrimp probiotic genetically modified donor bacteria, an shrimp symbiotic genetically modified donor bacterium an shrimp endosymbiotic genetically modified donor bacteria, an shrimp enteric genetically modified donor bacteria, Ag1, *Salmonella typhimurium*, *Bacillus subtilis, Enterobacter, E. coli*, or other symbiotic bacteria.

7. Use of the genetically modified donor bacterium of claim 1 and further comprising providing a therapeutically effective amount of said genetically modified donor bacterium to a target host wherein the therapeutically effective amount results in a reduction in mortality due to EMS caused by one or more EMS-causing *Vibrio*.

8. Use of the genetically modified donor bacterium of claim 1 and further comprising providing a therapeutically effective amount of said genetically modified donor bacterium to a target host wherein the therapeutically effective amount results in a reduction in biofilms or expression of pathogenesis genes including those involved in toxin production caused by one or more EMS-causing *Vibrio*.

9. Use of the genetically modified donor bacterium of claim 1 and further comprising providing a therapeutically effective amount of said genetically modified donor bacterium to a target host wherein the therapeutically effective amount results in a reduction in pathogenicity of one or more EMS-causing *Vibrio*.