**NIH Project Summary/Abstract Template**

*The Project Summary/Abstract is required for all NIH applications. It is limited to 30 lines of text and must follow NIH font, margin, and formatting requirements. Think of the summary as a brief, yet detailed account of your proposed research; furthermore, this document will help NIH staff to identify the most appropriate study section to review your application[[1]](#endnote-1).*

*The project summary is a succinct and accurate description of the proposed work and should be able to stand on its own (separate from the application). This section should be informative to other persons working in the same or related fields and understandable to a scientifically literate reader. Avoid both descriptions of past accomplishments and the use of the first person. Please be concise.*

***Content:***

* *State the application's broad, long-term objectives and specific aims, making reference to the health relatedness of the project (i.e., relevance to the mission of the agency).*
* *Describe the research design and methods for achieving the stated goals.*
* *Be sure that the project summary reflects the key focus of the proposed project so that the application can be appropriately categorized.*
* *Do not include proprietary, confidential information or trade secrets in the project summary. If the application is funded, the project summary will be entered into an NIH database and made available on the NIH Research Portfolio Online Reporting Tool (RePORT) and will become public information***[[2]](#footnote-1)***.*

Information to Include:

* Begin by noting the project’s broad, long-term objectives, particularly as they relate to human health applications. What research design/methodology will be used to address these objectives?
* Secondly, what is the project’s central hypothesis? Does this build upon previous research the PI/co-PIs and/or others have conducted?
* Thirdly, what are the project’s specific aims?
* Lastly, what aspects make the project particularly unique, innovative, and/or critically important? For what health-related areas will this project advance knowledge?

Writing Tips:

* Norins and Matheson[[3]](#footnote-2) suggest using “certain key words so the [Scientific Review Group] SRG staff can readily assign your application… SRG members who are not primary reviewers probably will rely heavily on your summary to understand your proposal during the group’s general meeting to discuss application fundability.”
* Write your Project Summary/Abstract last. After developing and writing the Specific Aims and Research Strategy documents, you will have a clearer, big picture view of your project that will allow you to write a strong, concise summary.
* Review the NIH Institute, Centers, and Offices’ (ICOs) [websites](https://www.nih.gov/institutes-nih/list-nih-institutes-centers-offices) to see if there are specific suggestions/requirements regarding what you should include in this section.
* Norins and Matheson state that a “good place to begin your summary is to get your reviewers’ attention by answering four questions:
  1. What is the problem or need that your proposal will address?
  2. Why is it so important that it must be resolved? In other words, what is the significance?
  3. Why are you the only person or group, or best-suited one, who can resolve the problem or need?
  4. What is your proposed solution to address the problem?”

While there is no exact template for how to write the Project Summary/Abstract, examples are provided below from Norins and Matheson, who draw upon elements NIAID indicates are important for applications. Review your chosen [Institute or Center](https://www.nih.gov/institutes-nih/list-nih-institutes-centers-offices) to see if they provide best practices or specific criteria for this document.(All information is directly quoted2; *blue words/phrases* draw attention to language that guides the reader to know, rather than infer, the writer’s purpose):

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| **Proposal Title: Molecular Mechanism of Phosphate Signaling in E. Coli** | |
| **NIAID-significant topics** | **Summary** |
| Significance of the proposed research | *The objective of the proposed research is to understand how* information concerning environmental phosphatelevels is transmitted through the phosphate signaltransduction pathway to control gene expression inEscherichia coli. At the heart of this pathway are theresponse regulator PhoB and the histidine kinase PhoR. *A great deal of work has already been accomplished* elucidating the mechanisms which these two proteinsfunction. *The focus of this proposal is to understand how* two auxiliary proteins, PstSCAB2 and PhoU, are involved [i]n the sensing of phosphate and the control of this signaling pathway. |
| Innovation & unique features of the proposal | *Given the history of research into phosphate regulation, it is somewhat surprising that fundamental questions still exist as to how* cells sense phosphate and how thatinformation is transmitted within a cell. PstSCAB2 is aphosphate transporter that also functions as the sensorfor the system. PhoU controls the activity of PstSCAB2and is required for the transmission of the signal fromPstSCAB2 to PhoR. |
| Methodology or Specific Aims | *Our first hypothesis is that* the PstSCAB2 transporter transduces information about environmental phosphate levels through conformational changes that are inherent to the transport process. *Our second hypothesis is that* PhoU transmits this information by specifically interacting with a particular PstSCAB2 conformation and mediates protein/protein interactions with PhoR. *We plan on addressing these hypotheses through* both genetic and biochemical approaches. Mutant versions of the transporter will be isolated that “lock” it into various conformations. We will use these versions of the transporter to trap complexes with other proteins using co-elution and co-immunoprecipitation experiments and as well as bacterial two-hybrid analysis. *These* *approaches should prove to be complementary to one another and will provide the greatest opportunity to observe* protein/protein interactions within the proposed signaling complex.  *In the analysis of* the role of PhoU, constitutive signaling mutants will be isolated in the absence of the transporter and studied for interactions with PhoR. *In addition,* highly conserved amino acid residues of PhoU, which must be important to its function, will be mutated and studied for function and cellular localization. PhoUGFP fusions will also be constructed and used to study localization. |
| Re-emphasis of the proposal’s innovation | *The proposed work is important because* these signaling proteins *are essential* for bacteria to survive changing environments – including the human immune system. This feature combined with their absence in higher eukaryotes, makes this signaling pathway *a target for the development of* new antimicrobial drugs. *An increased understanding of these* signal transduction proteins *may assist in* the rational design of drugs to combat pathogens. |

Project Summary/Abstract from a successful R15 grant. This summary is structured, with subtitles in bold. The authors do not end with a revisiting of the innovation in the proposal, instead highlighting it with specific formatting.

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| **Proposal Title: Structural Characterization of the M. tuberculosis Thioredoxin**  **System** | |
| **NIAID-Significant**  **Topics** | **Summary** |
| Significance of the proposed research | The thioredoxin system is ubiquitous, and *plays an essential role in* maintaining thiol/disulfide redox homeostasis in cells. The *Mycobacterium tuberculosis* *(M. tb)* thioredoxin system *has recently been suggested as* a target for anti-tuberculosis (TB) drug development, by disrupting its ability to protect M. tb from the oxidative attacks of macrophages. |
| Innovation & unique features of the proposal | *But, while* thioredoxin systems from some bacteria (ex. *E. coli*) and human are well-characterized, the mycobacterialsystem *is not*. The *M. tb* system is comprised of threethioredoxins (TrxA, TrxB and TrxC) and one thioredoxinreductase (TrxR). Drug discovery efforts targeting this  system are hindered by the lack of available structures for TrxA and TrxB and of their complexes with TrxR. *Furthermore,* the differential roles of the three *M. tb* thioredoxins *are not known*, *with some publications suggesting that* TrxA may even be non-functional.  **The *objective*** *of this project is to define* different structural/functional properties of the three *M. tb* thioredoxins, and *to determine if* TrxA is truly cryptic. *We will use these structures as a foundation for* structure based identification of inhibitors of the entire M. tb thioredoxin system, or specific complexes.  ***Expected outcomes and impact:*** *This project will produce* a structural characterization of the M. tb thioredoxin system, and inhibitors as chemical genetic probes of function and anti-TS drug leads. |
| Methodology or Specific Aims | ***Our Aims Are to:***  **1. Determine solution structures for all M. tuberculosis**  **thioredoxins.** Determine 4 NMR structures: TrxA and TrxB in both redox states (thiol/disulfide), and compare to our TrxC structures.  **2. Determine structural models of *M. tuberculosis***  **TrxR/TrxN (where N=A, B) complexes, and compare**  **to TrxR/TrxC.** (a) Establish if and how TrxR binds the two TrxN’s (both redox states). (b) Determine structural changes in TrxN, induced by TrxR binding. (c) Determine dynamics changes in NADPH cofactor (bound to TrxR), induced by TrxN binding. (d) Construct structural models for the two TrxN/TrxR complexes based on NMR chemical shift perturbations, for various dead end complexes to mimic intermediates in the catalytic cycle.  **3. Identify inhibitors of the *M. tuberculosis* TrxR/TrxN system.** (a) Using the 3-dimensional structural models for the M. tuberculosis TrxR/TrxN system (andcomparable M. smegmatis structures), computationallydock compounds to identify candidate inhibitors, (b)  test candidate inhibitors in both NMR binding (titration) and enzymatic inhibition assays, to determine affinity, and (c) test compounds that have Kd < 50 μM in MIC (minimum inhibitory concentration) assays initially with M. smegmatis (then M. tuberculosis). |
| Re-emphasis of the proposal’s innovation | [Not done in this proposal.] |

1. Norins, L., & Matheson, S. (2014). NIH R15 Grant Application Mentor: An Educational How-to Manual, (2nd edition). Bonita Springs, FL: Principal Investigators Association [↑](#endnote-ref-1)
2. *NIH Research Instructions*. (2018). pp. R-38-R-38. Retrieved from <https://grants.nih.gov/grants/how-to-apply-application-guide.html> [↑](#footnote-ref-1)
3. Norins, L., & Matheson, S. (2014). *NIH R15 Grant Application Mentor: An Educational How-to Manual* (2nd edition). Bonita Springs, FL: Principal Investigators Association. [↑](#footnote-ref-2)