No.	Paper details	Mention the specific contribution of Dr. Ramandeep Singh
1.	Gosain TP, Chugh S, Rizvi ZA, Chauhan NK, Kidwai S, Thakur KG, Awasthi A and **Singh R***. *Mycobacterium tuberculosis* strain with deletions in *menT3* and *menT4* is attenuated and confers protection in mice and guinea pigs. **Nature Communications, 15,** 2024**,** 5467	In this research article, we have shown that *menT3* and *menT4* belonging to *menAT* TA systems are essential for disease pathogenesis. We showed that immunization of animals with the Δ*menT3*Δ*T4* strain is able to generate TH1 response, activated memory response and impart protection against M*. tuberculosis* challenge.
2.	Chugh S, Tiwari P, Suri C, Gupta SK, Singh P, Bouzeyne R, Kidwai S, Srivastava M, Rameshwaram NR, Kumar Y, Asthana S and **Singh R***. Polyphosphate Kinase -1 regulates bacterial and host metabolic pathways involved in pathogenesis of *Mycobacterium tuberculosis*. **Proceedings of the National Academy of Sciences**, 2024, 121(2), e2309664121.	In this research article, we have delineated the mechanisms by which polyP deficiency regulates mycobacterial pathogenesis. We showed that the levels of various virulence associated lipids was reduced in Δ*ppk-1* mutant strain relative to the wild type strain. it was also demonstrated that in comparison to wild type infected animals, Type I IFN-signalling and formation of foamy macrophages were reduced in Δ*ppk-1* mutant infected animals. Further, using target based screening, raloxifene (approved drug) was identified as PPK-1 inhibitor and was able to reduce the growth of intracellular *M. tuberculosis* in mice.
3.	Agarwal S, Sharma A, Bouzeyen R, Deep A, Sharma H, Mangalaparthu K, Datta KK, Kidwai S, Gowda H, Varadarajan R, Sharma RD, Thakur KG and **Singh R.** VapBC22 toxin-antitoxin system from *Mycobacterium tuberculosis* is required for pathogenesis and modulation of host immune response. **Science Advances** 2020, 1-15.	In this research article, we have characterized VapBC22 TA system from *M. tuberculosis*. The authors showed that VapC22 is important for adaptation of *M. tuberculosis* upon exposure to oxidative

		stress. It was also demonstrated that in comparison to wild type strain, Δ*vapC22* was attenuated for growth *in vivo*.
4.	Deep A, Tiwari P, Agarwal S, Kaundal S, Kidwai S, **Singh R***, and Thakur KG*. Structural, functional and biological insights into the role of *Mycobacterium tuberculosis* VapBC11 toxin-antitoxin system: targeting a tRNase to tackle mycobacterial adaptation. **Nucleic acids Research** 21, 2018, 11639-55. ***co-corresponding author.**	In this research article, we have characterized VapBC11 TA system from *M. tuberculosis*. Using RNA-seq data, we have identified the cellular targets for VapC11 toxin. It was also shown that VapBC11 is essential for *M. tuberculosis* to establish disease in guinea pigs. In collaboration with Dr. Krishan Gopal at IMTECH, we have also solved the three-dimensional structure of VapBC11.
5.	Tiwari P, Arora G, Singh M, Kidwai S, Narayan O, and **Singh R**. MazF ribonucleases promote *Mycobacterium tuberculosis* drug tolerance and virulence in guinea pigs. **Nature Communications** 2015; 6(1) 1-13.	In this research article, we have characterized MazF toxins belonging to MazEF TA systems from *M. tuberculosis*. We showed that simultaneous deletion of MazF3, MazF6 and MazF9 impaired survival of *M. tuberculosis* upon exposure to oxidative stress and levofloxacin. We have also shown that MazF3, MazF6 and MazF9 contribute cumulatively to pathogenesis of *M. tuberculosis*.
6.	Agarwal S, Tiwari P, Deep A, Kidwai S, Gupta S, Thakur KG and **Singh R**. System wide analysis reveals differential regulation and in vivo essentiality of VapBC TA systems from *Mycobacterium tuberculosis*. **The Journal of Infectious Diseases** 217 (11), 2018, 1809-20.	In this research article, we have characterised VapC toxins belonging to VapBC TA systems from *M. tuberculosis.* We also showed that a subset of toxins are differentially expressed upon exposure to stress conditions and transcriptional cross-talk exists between TA systems. Using guinea pig model of infection, it

		was demonstrated that VapBC3 and VapBC4 TA systems also contribute to disease pathogenesis.
7.	Singh P, Kumar A, Sharma P, Chugh S, Kumar A, Sharma N, Gupta S, Singh M, Kidwai S, Sankar J, Taneja N, Kumar Y, Dhiman R, Mahajan D and **Singh R***. Identification and optimization of pyridine carboxamide scaffold as a drug lead for *Mycobacterium tuberculosis*. **Antimicrobial Agents and Chemotherapy**, 2024, 68(2), e00766-23.	In the study, we have performed phenotypic based screening and identified pyridine carboxamide as a drug lead for *M. tuberculosis*. We have shown that this drug requires an amidase for activation. The authors also performed a detailed structure activity relationship studies for the lead molecule. Further, it was also shown that this series of molecules possesses a dual mechanism of action. We also show that the optimized lead compound was able to inhibit the growth of intracellular *M. tuberculosis* in mice tissues.
8.	Arora G, Tiwari P, Mandal RS, Gupta A, Sharma D, Saha S and **Singh R*.** High throughput screen identifies small molecule inhibitors specific for *Mycobacterium tuberculosis* phosphoserine phosphatase. **Journal of Biological Chemistry**, 289 (36), 2014, 25149-25165.	In this study, we have biochemically characterized SerB2 enzyme (an enzyme involved in L-serine biosynthesis) from *M. tuberculosis*. Using target based screening, the authors have identified SerB2 specific inhibitors. These molecules were able to inhibit *M. tuberculosis* growth in liquid cultures and in macrophages. Also, molecular docking studies were performed to identify SerB2- small molecule interacting residues.
9.	**Singh R,** Singh M, Arora G, Kumar S, Tiwari P, Kidwai S. Polyphosphate deficiency in mycobacterium tuberculosis is associated with enhanced drug susceptibility and impaired growth in guinea pigs. **Journal of Bacteriology**, 195, 2013, 2839 - 51.	In this study, we have functionally characterized PPK-1 enzyme from *M. tuberculosis*. It was demonstrated that polyP accumulates in *M. tuberculosis* during later stages of growth

		and upon exposure to stress conditions and drugs. We also showed that polyP deficiency enhances the susceptibility of *M. tuberculosis* to front-line TB drugs. Finally, we showed that Δ*ppk-1* mutant strain was attenuated for growth in guinea pigs in comparison to the parental strain.
10.	**Singh R**, Rao V, Shakila H, Gupta R, Khera A, Dhar N, Singh A, Koul A, Singh Y, Naseema M, Narayanan PR, Paramasivan CN, Ramanathan VD and Tyagi AK. Disruption of *mptpB* impairs the ability of *Mycobacterium tuberculosis* to survive in guinea pigs. **Molecular Microbiology,** 50 (3), 2003, 751 – 62.	In this article, we showed that MptpB, secretory tyrosine phosphatase is essential for *M. tuberculosis* pathogenesis. We also showed that MptpB might interfere with IFN-γ mediated signaling in *M. tuberculosis*.