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Protein functionalized nanostructured zirconia based electrochemical immunosensor for cardiac troponin I detection

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We report results of the studies relating to the fabrication of nanostructured zirconia (ZrO_2) based immunosensor for cardiac troponin I biomarker (myocardial infarction) detection. One step, low temperature hydrothermal process has been used for the synthesis of nanostructured ZrO_2 (4.5 nm). These ZrO_2 nanoparticles have been functionalized with 3-amino propyl triethoxy silane (APTES) and thereafter electrophoretically deposited (15 V, 3 minutes) on indium tin oxide coated glass electrode (ITO). EDC/NHS chemistry has been used for covalent immobilization of anti-troponin-I (anti-cTnI) onto APTES/ZrO_2/ITO electrode. Structural, morphological and functional characterization of the synthesized nanoparticles and the fabricated immunoelectrode have been carried out via X-ray diffraction (XRD), transmission electron microscopy (TEM), atomic force microscopy (AFM), fourier transform infrared spectroscopy (FTIR) and cyclic voltammetry techniques. The results of electrochemical response studies of BSA/anti-cTnI/APTES/ZrO_2/ITO immunoelectrode reveal that this smart platform can be used for the detection of troponin I biomarker with a wide linear detection range (0.01 to 100 ng mL^{-1}), high sensitivity (3.7×10^{-4} mA mL ng^{-1} cm^{-2}), remarkable lower detection limit (0.036 ng mL^{-1}) and stability of upto 30 days. The obtained results have been validated through enzyme linked immunosorbent assay (ELISA).

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Identification and characterization of novel drug targets against glutamate racemase of *Mycobacterium tuberculosis*

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There is a pressing need to identify novel drug targets and discover newer antimicrobial inhibitors given the ever-evolving rate of drug resistance against various reported antimicrobials used to treat known infectious diseases. *Mycobacterium tuberculosis*, the causative agent of infectious disease Tuberculosis, has also developed drug resistance against various antibiotics that were used to treat patients. Several reports of multiple drug resistance and extensively drug resistance strains of *M. tuberculosis* are also reported in the literature. Systems biology approaches offer an important platform that facilitates identification of potential drug targets to circumvent the problem of ever increasing drug resistance. Proteins exhibiting high level of conservation among various species could be considered and reported inhibitors against these homologous proteins may be used for their binding and further inhibiting mycobacterial protein. In the present study, we have analyzed the possible mechanism of action of compounds targeting one such protein Glutamate racemase of *M. tuberculosis* (MTB-GR), an enzyme that is involved in the early phases of peptidoglycan biosynthesis. We analyzed known inhibitors of similar protein from among other organisms for their binding capacity with glutamate racemase homology model of *M. tuberculosis*. In this study, protein model building and lead-inhibitor identification was carried out for MTB-GR and the structure was further refined and validated using PROCHECK online tool. Compounds showing activity against glutamate racemase enzymes of other organisms were collected from the literature and were docked into the active site of MTB-GR. We analyzed three inhibitors namely, SIN inhibitor, B08698 and DB08272, reported in the literature. Among these three inhibitors, SIN inhibitor exhibited maximum Ludi score and hydrogen bonding interactions with the key amino acid residues of glutamate racemase. Therefore, SIN inhibitor could act a promising lead compound for the drug design against *Mycobacterium tuberculosis* glutamate racemase enzyme.

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