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Poster Number

36 **A Novel Aptamer-Based Enzymatic Assay for the Diagnosis of Malaria**

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Malaria causes nearly 1 million annual fatalities in lesser developed regions in South East Asia and Africa. Symptoms of malaria are unspecific (fever, vomitting, headache etc.) meaning without laboratory equipment reliable diagnosis is problematic. Point-of-care antibody-based dipsticks are available on the market but the cost and instability of antibodies in heat and humidity limits their widespread use. Aptamers are short oligonucleotide sequences which selectively bind their targets and present as stable and affordable alternatives to antibodies in point-of-care tests. Aptamers against the malaria antigen Plasmodium falciparum lactate dehydrogenase (pLDH) have been developed in our lab with nanomolar Kd affinities and are specific enough to not interact with human LDH isoforms. We have incorporated these aptamers into a novel 96-well plate based malaria test called the aptaMAL. Surface bound aptamers capture pLDH out of blood samples and after wash steps and reagent addition, the solution turns blue if the test is positive. The colour is achieved by coupling a colour changing reaction to the enzyme activity of pLDH. The test has been found to be effective on rat blood samples spiked with the pLDH antigen at very low concentrations (aptaMAL limit of detection 10 ng/mL, typical pLDH concentration in infected blood 3000 ng/mL). This aptaMAL mechanism shows potential to be incorporated into a point-of-care device format which would be affordable, reliable and have no need for any complex equipment or unstable reagents.

37 **Nuclear translocation of HDAC4 in Alzheimer's disease human brain**

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Histone deacetylases (HDACs) play a crucial role in histone modification whose inappropriate function has been linked to Alzheimer's disease (AD). HDAC4, as a member of the family of HDACs, presents predominantly in the cytoplasm of the neurons of the human brain. As our lab has shown previously, its nuclear translocation is mediated by a decreased level of ATM (ataxia telangiectasia [A-T], mutated), a multi-functional protein kinase whose deficiency leads to A-T. Based on the fact that both A-T and AD are neurodegenerative disease involving modification of the histone code, there arises the possibility that ATM may play a role in AD. Using the nuclear translocation of HDAC4 as a marker, previous work has proved the involvement of ATM deficiency in AD diagnosed with clinical dementia rating (CDR) system. Extending that work, we performed immunohistochemistry on the human brain samples grouped by Braak stages. This staging scheme is based largely on the extent and location of deposits of hyperphosphorylated tau. We found a significant decrease in ATM activity in hippocampus between groups of age-matched controls with normal cognition, mild cognitive impairment (MCI) and AD. Consistent with previous work, we found that the CA2 sub-region was the area with the highest levels of nuclear HDAC4. Extending this work, we show for the first time a substantial correlation of this phenotype with the presence of ectopic cell cycle markers. Taken together, the results confirm the importance of the decrease in ATM activity during the pathogenesis of AD.