DELTA-AMINOLEVULINIC ACID DEHYDRATASE (ALAD) POLYMORPHISM IN LEAD EXPOSED BANGLADESHI CHILDREN AND ITS EFFECT ON URINARY AMINOLEVULINIC ACID (ALA)

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Background and objective
Lead has long been recognized as a harmful environmental pollutant. Developing countries like Bangladesh still have higher risk of lead exposure. Previous research suggests that delta-aminolevulinic acid dehydratase (ALAD) genotype can modify lead toxicity and individual susceptibility. As children are more susceptible to lead induced toxicity, the study was to investigate the frequencies of ALAD polymorphism among environmentally lead exposed children in Bangladesh and to determine whether ALAD genotype influenced urinary excretion of aminolevulinic acid (ALA).

Methods
Subjects were elementary school children from a semi-urban industrialized area in Bangladesh. A total of 134 children were studied. Blood and urine were collected to determine the ALAD genotypes, blood lead level (BPb) and urinary aminolevulinic acid (U-ALA).

Results
The mean BPb was found 9.7 µg/dl with 34% participants exceeding 10µg/dl, the CDC recommended level of public health action. In total, the gene distribution for ALAD1-1 was 71%, ALAD1-2 was 26% and ALAD2-2 was 3%. In this study, U-ALA concentration was lower in ALAD2 carriers than ALAD1 carriers at the same exposure level for boys (p = 0.01). But for girls, U-ALA did not differ significantly by genotype (p = 0.61).

Conclusion
This study provides the information about the frequency of ALAD polymorphism in Bangladeshi children for the first time, associated with U-ALA. ALAD2 showed protective effects in terms of U-ALA among the boys exposed to environmental lead.