The metalloid arsenic enters the environment by natural processes (volcanic activity, weathering of rocks) and by human activity (mining, smelting, herbicides and pesticides). Although arsenic has been exploited for homicidal and suicidal purposes since antiquity, its significance as a public health issue arises from its potency as a human carcinogen. In addition, considerable epidemiological evidence shows that chronic exposure to inorganic arsenic contributes to increased risk of other diseases (Hughes et al., 2011). Interest in the biomethylation of arsenic as a factor in its environmental fate and its actions as a toxicant and a carcinogen originated in the 19th century with observations that microorganisms converted inorganic arsenicals used as wallpaper pigments into Gosio gas, a volatile species that is released into the atmosphere and that Gosio gas was trimethylarsine. Subsequent detection of methylated arsenicals in natural waters and in human urine suggested that biomethylation of arsenic was a widespread phenomenon (Cullen, 2008).

These studies piqued interest in understanding the molecular basis of biomethylation of arsenic. Studies in mammals demonstrated methylation of arsenic to be enzymatically catalyzed and culminated in isolation and purification of an arsenic methyltransferase from rat liver cytosol and cloning and expression of cognate genes from rat, mouse, and human (Thomas et al., 2007). This gene was initially identified as *cyt19* but is now designated as arsenic (+3 oxidation state) methyltransferase (*As3mt*). The human *AS3MT* gene (accession number AAI19639.2) encodes a 367 amino-acid protein (33010 Da; EC # 2.1.1.137) that contains sequence motifs commonly found in non-DNA methyltransferases. Putative *As3mt* genes have been identified in genomes of deuterostomes ranging in complexity from purple sea urchin (*Strongylocentrotus purpuratus*) to *Homo sapiens.*