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Review

Arsenic and human health effects: A review

Khaja Shameem Mohammed Abdul [a], Sudheera Sammanthi Jayasinghe [b], Ediriweera P.S. Chandana [a], Channa Jayasumana [c], P. Mangala C.S. De Silva [a]

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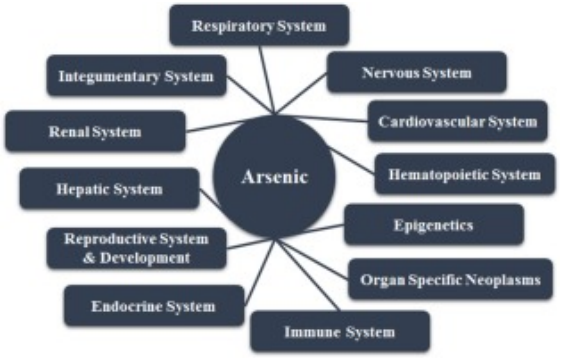
Abstract

Arsenic (As) is ubiquitous in nature and humans being exposed to arsenic via atmospheric air, ground water and food sources are certain. Major sources of arsenic contamination could be either through geological or via anthropogenic activities. In physiological individuals, organ system is described as group of organs that transact collectively and associate with other systems for conventional body functions. Arsenic has been associated with persuading a variety of complications in body organ systems: integumentary, nervous, respiratory, cardiovascular, hematopoietic, immune, endocrine, hepatic, renal, reproductive system and development. In this review, we outline the effects of arsenic on the human body with a main focus on assorted organ systems with respective disease conditions. Additionally, underlying mechanisms of disease development in each organ system due to arsenic have also been explored. Strikingly, arsenic has been able to induce epigenetic changes (in utero) and genetic mutations (a leading cause of cancer) in the body. Occurrence of various arsenic induced health effects involving emerging areas such as epigenetics and cancer along with their respective mechanisms are also briefly discussed.

Graphical abstract



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Abbreviations

iAs, inorganic arsenic; JNK3, c-Jun N-terminal kinase-3; p38MAPK, p38 mitogen activated protein kinase; ROS, reactive oxygen species; CC16, clara cell protein; CVD, cardiovascular diseases; BFD, endemic black foot disease; PVD, peripheral vascular disease; GSTP1, glutathione S-transferase P1; IHD, ischemic heart disease; ECG, electrocardiography; HERG, cardiac potassium channel human *ether-a-go-go*-related gene; UPR, unfolded protein response; T3, tri-iodothyronine; T4, thyroxine; T1DM, type 1 diabetes mellitus; T2DM, type 2 diabetes mellitus; SNP, single nucleotide polymorphism; CAPN-10, clapain-10 gene; NGal, neutrophil gelatinase-assoicated lipocalin; ROS, reactive oxygen species; NF-κB, nuclear factor-κB; TNFα, tumor necrosis factor α; IL-6, interleukin-6; PPARγ, peroxisome proliferator activated receptor γ; JNK, C-Jun N-terminal kinase; CKDu, chronic kidney disease of unknown etiology; ATF-2, activation transcription factor-2; AP-1, activator protein-1; ELK-1, ETS domain containing protein ELK-1; C-Src, proto-oncogene tyrosine-protein kinase Src; JAK, janus kinase; STAT, signal transducer and activator of transcription; MAPK, mitogen-activated protein kinases; EGFR, epidermal growth factor receptor; PI3K, phosphatidylinositol-4,5-bisphosphate 3-kinase; AKT, protein kinase B; PTEN, phosphatidylinositol-3,4,5-trisphosphate 3-phosphatase; VEGF, vascular endothelial growth factor; NOTCH2, neurogenic locus notch homolog protein 2; ADAMTS9, a

disintegrin and metalloproteinase with thrombospondin motifs 9; NRF2, nuclear factor (erythroid-derived 2)-like 2; KEAP1, kelch-like ECH-associated protein-1; CUL3, cullin-3; *DAPK*, death-associated protein kinase; RHBDF1, rhomboid family member 1; Alu, arthrobacter luteus; LINE-1, long interspersed element-1; DNA, deoxyribonucleic acid; HCY, hydrofolatehomocysteine; SAM, S-adenosylmethionine; IARC, International Agency for Research on Cancer; ATP, adenosine triphosphate; UV, ultraviolet; NMSC, non-melanoma skin cancer; BCC, basal cell carcinoma; SCC, squamous cell carcinoma; SMI, arsenic methylation index; CAE, cumulative arsenic exposure

Keywords

Arsenic; Environmental exposure; Toxicity; Organs at risk; Epigenesis; Neoplasms