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Heavy Metal Ion Reducing Microorganisms versus Bioremediation of Key Pollutant Elements in Environment and Foods Affecting Human Health: An Overview

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ABSTRACT

Considering a global issue pertaining to impact of heavy metals on environmental components, this overview covers the action-packed progression of 'Bioremediation' at diversified level. An augmentation of heavy metal in water bodies alongwith the soil in close environs to industries making it lethal and for scheming it, bioremediation is brought into task including a sequence of processes, with the aid of metal resistance microorganisms, followed by accumulation of metal like Zinc, Cadmium and Arsenic. Moreover, amongst the microorganisms, capable of bioremediation of the major pollutant elements, namely, lead, cadmium, arsenic and mercury, encircling their individual allowable limits, in context to foods and beverages, Saccharomyces cerevisiae *has been revealed to be an appealing preference for its exceptionality and being safe for humans, eventually enabling it rationally common and admirable in terms of '**Food Safety**' in the food processing sectors. As there is at a standstill prerequisite for further studies to expand bioremediation technologies in order to find additional biological solutions for bioremediation of heavy metal contamination from different environmental systems, the compilation of data with noteworthy hypotheses taken together with this overview provides new insights into rising a platform to investigate certain novel bioremediation investigational model that could be speedy, accurate and cost effective, eventually affecting human health.*

Keywords: *Bioremediation, biosorption, contaminants, environment, food safety, heavy metals, human health.*

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I. Introduction

In fact, heavy metal effluence is a serious concern because of hazardous impacts at even nano concentrations. Heavy metals are non-biodegradable, bioaccumulate in tissues and are biomagnified along with the trophic levels [1-3]. The nature of heavy metals released from the industrial waste depends on nature of industrial effluent and certain other factors such as the innate chemical profile of the soil, climate, nature, and composition of the soil and other anthropogenic activities in the specific region [2, 4]. Subsequent releases and the entry of heavy metals into the food chain depend on their concentration and uptake by the local flora and fauna [2, 5]. The genetic and epigenetic effects of these elements are associated with an increased risk of different cancer types [1-4, 6]. Epigenetic mechanisms play an equally important if not a more prominent role than genetic events in carcinogenesis. These effects occur most frequently during the early stages of tumor development. Epigenetic measures include reversible modification of histone proteins and CpG islands of gene promoters that affect not only gene expression of germ and somatic cells, but also cause indirect gene-sequence changes [7, 8].

In ranking the carcinogens, heavy metals have been classified by the International Agency for Research on Cancer (IARC) and Environmental Protection Agency (EPA) as the first group, except for selenium that has been listed within group 3 (not carcinogen to humans) of the IARC classification [9]. The impact of heavy metals on the quality of water and soil due to urbanization and industrialization concomitant with Bioremediation applying certain potential devices including the context of '**Food Safety**' has been overviewed under following headings:

(1) Modes of Exposure to Toxicants

Animals including human usually get exposed to the toxicants through: (a) respiratory (for gaseous and particulate matters); (b) the skin (chemicals able to cross skin barrier); (c) digestive tract (for food contaminants). After entering the body the metal deposited in nasopharyngeal, tracheobron chial, or pulmonary compartments may be transported through the mucociliary action to the gastrointestinal tract. Macrophages phagocyte the wandering metals. Food is a principal source of essential and toxic elements. Some ele ments like mercury (Hg) are biologically magnified at higher trophic level. The dietary contribution for toxic metal intake has been extensively studied [10]. If an individual is deficient in minerals and trace elements its body will absorb heavy metals on their place. Every cell membrane breaks down and rebuilds every two weeks but does not release the heavy metals if essential fats are not properly ingested or if poor quality fats are ingested. The liver that performs detoxification 100% of the time cannot perform this important task without a complete profile of essential nutrients.

Chemical elements present in the form of free ions are readily ionized and eventually get absorbed totally by the body. Transition metals readily form stable covalent complexes and generally intermingle as parts of macromolecules (proteins, enzymes, hormones, etc.) according to their chemical uniqueness including oxidation state [1, 2]. The behavior of metal ion release into biofluid is regulated by the electrochemical rule. Released metal ions do not all the time combine with biomolecules to come out toxicity as active ion immediately combine with a water molecule or an anion in close proximity to the ion to form an oxide, hydroxide, or inorganic salt. Consequently, there is only a small chance that the ion will merge with biomolecules to cause cytotoxicity, allergy, and other biological effects [11-13]. Health damage caused by toxic metals may be less (irritation) or acute (mutagenic, teratogenic and carcinogenic). These reactive elements of food fabricate complexes with fiber, reveal low solubility within the intestinal lumen and are weakly absorbed (**Table I**). Absorption of these minerals is augmented at low concentration of fiber, and in the absence of phytates and oxalates in the diet [12]. Micronutrients can interact with toxic metals in the body at several points: ***(a)*** absorption; (b) transport; (c) binding to target proteins; (d) metabolism; (e) sequestration; (f) excretion of toxic metals; and (g) finally in secondary symptoms of toxicity such as oxidative stress [13-17]. The role of oxidative stress in the destruction of immune cells has been elucidated [13-17]. Therefore, a diet poor in micronutrients can lead to enhancement in the toxicity. The prevalence and mortality due to multifactorial polygenic diseases; hypertension, coronary artery disease (CAD), diabetes and cancer vary depending upon genetic susceptibility as well as environmental pollutants generated as a consequence of numerous chemicals, metal ions and metalloids. Speedy

Table I: Food Sources of Toxic Metals

Metal	Food Source
Pb	Egg, cocoa powder, rice, wheat, potato, calcium supplement, smoked food, wine, beer, milk, carrot, raisins
As	Green papaya, rice, tomato, carrot, seafood, Indian mustard, bovine and chicken meat, wine, milk
Hg	Egg, mushroom, seafood, fish oil
Cd	Egg, fish, mushroom, garlic, spinach, wheat, rice, oat, corn, soyabean, peanuts, mushroom

Source: Mudgal *et al*. (2010); Mishra et al. (2021)

changes in diet and lifestyle may manipulate heritability of the variant phenotypes that are dependent on the nutraceutical or functional food supplementation for their expression [18]. It is possible to recognize the interaction of specific nutraceuticals, with the genetic code possessed by all nucleated cells. There is evidence that South Asians have an increased susceptibility to CAD, diabetes mellitus, central obesity and insulin resistance at younger age, which may be due to interaction of gene and nutraceutical (especially micronutrients) environment. These populations appear to have inherited predisposition and may have interaction of internal nutritional status and environmental factors, mainly metal ions. Higher intake of refined starches and sugar increases generation of super oxide anion in the leucocytes and mononuclear cells, and free fatty acids (FFA), as well as higher amount and activity of nuclear fac tor-kB (NF-kB), a transcriptional factor regulating the activity of at least 125 genes, most of which are pro-inflammatory. Glucose intake also causes an increase in two other pro-inflammatory transcription factors; activating pro tein-1 (AP-1) and early growth response protein-1 (Egr-1), the first regulating the transcription of matrix metallo-proteinases and the second modulating the transcription of tissue factor and plasminogen activator inhibitor-1. Refined food, mixed meal induces activation of NF-kB associated with free radicals generation by mononuclear cells. The superoxide anion is an activator of at least two major pro inflammatory transcription factors, NF-kB and AP-1. Increased intake of linoleic acid, saturated fat, trans fat and refined starches and sugars can increase the generation of free radicals and activate the NF-kB, leading to rapid expression of pro-inflammatory genes. It is possible that nutraceuticals; antioxidants, micronutrients, minerals, vitamins, coenzyme Q10 and w-3 fatty acids may inhibit the generation

of super oxide and suppress NF-kB as well as AP-1, and Egr-1 leading to suppression of phenotypic expressions. It is known that genes are important in determining enzymes, receptors, cofactors, structural components involved in regulation of blood pressure, the metabolism of lipids, lipoproteins and inflammatory and coagulation factors that are involved in determining individual risk for vascular diseases and diabetes. It seems that these phenotypic expressions may be silenced by targeting simple sequence differences known as single nucleotide polymorphisms by nutraceuticals and slowly absorbed wild foods rich in micronutrients and antioxidants.

In biological fluids and tissues, the majority of metals and metalloids are not present as free cations. In blood they are generally bound to red cells or to plasma proteins. Lead and cadmium are almost totally bound to red blood cells. The chemical elements bound to plasma proteins constitute the fraction available for transport into and out of the tissues. Albumin, a plasma protein, has an enormous capacity to bind several metals.

(2) Endurable Daily Intake Approach

In view of avoiding undesirable health hazards consequent of "excessive" intake of toxicants (including toxic metals), international and national scientific organisms such as FAO/WHO, FDA, European Union, etc have used the safety factor approach for establishing acceptable or tolerable intakes of substances that exhibit threshold toxicity. The acceptable daily intake (ADI) or Endurable daily intake (EDI) or provisional endurable weekly intakes (PEWI) are used to describe "safe" levels of intake for several toxicants including toxic metals [19]. For chemicals that give rise to such toxic effects, a tolerable daily intake (EDI), i.e. an estimate of the amount of a substance in food, expressed on a body weight basis (mg.kg-1 or mg.kg-1 of body weight) that can be ingested over a lifetime without appreciable health risk. Exposure exceeding the TDI value for short periods should not have deleterious effects upon health. However, acute effects may occur if the TDI is substantially exceeded even for short periods of time. Besides, contaminants possessing very long half-lives can be accumulated in the body and chronic effects are most often observed when critical concentrations are reached in target tissues. The comprehensive account of health hazards rendered principally by aluminium (Al), arsenic (As), cadmium (Cd), lead (Pb), mercury (Hg), Selenium (Se) and Lethium (Li) is represented as follows:

***(a) Aluminium*:** The compounds of this element have a wide range of applications in different industries, including cosmetics, and food additives [20]. Aluminum-induced carcinogenesis is related to its ability to bind to the estrogen receptor and mimic estrogen functions, therefore its named metallo-estrogen [20]. Metallo-estrogen triggers expression in genes that contain estrogen responsive-element (ERE) on their promoters. In mammary gland cells, this gives rise to an increase in the number of divisions of breast cells, thus increasing replication errors in cancer-related genes [21]. It has been shown that if antiperspirants containing aluminum applied on the skin around the underarm and breast areas are not effectively washed, some aluminum salts remains in the area. This gives rise to continuous exposure, and enhances the risk of breast cancer [22].

***(b) Arsenic*:** Arsenic is mostly known as an epigenetic carcinogen metalloid when in the form of an inorganic compound. Trivalent arsenite (As+3) has more carcinogenic properties than the pentavalent arsenate (As+5) [23, 24]. Trivalent arsenic can bind with high affinity to thiol groups of proteins and reduced glutathione (GSH) [25]. Long time uptake of drinking-water containing low levels of arsenite, induces carcinogenesis in skin, lung, bladder, and kidney tissues, resulting from alteration in multiple signaling pathways [26].

***(c) Cadmium*:** Certain compounds of cadmium (Cd) are highly toxic to humans. Cadmium is employed in several industrial proc esses such as: (a) protective coatings (electroplating) for metals like iron; (b) preparation of Cd-Ni batteries, control rods and shields within nuclear reactors and television phosphors. Cadmium is a cumulative toxicant and carcinogenic that affects kidneys, generates various toxic effects in the body, disturbs bone metabolism and deforms reproductive tract as well as endocrine system. There are several morpho pathological changes in the kidneys due to long-term expo sure to cadmium. Increasing intakes of zinc can reduce the renal toxicity of cadmium. An exposure to cadmium increases calcium excretion thus causes skeletal demineralization, probably leading to increases in bone fragility and risk of fractures [27]. Cadmium and its compounds are currently classified by IARC as a Group 1 carcinogen for humans. Occupational human exposure has been correlated with lung cancer. Cadmium exposure, during human pregnancy, leads to reduced birth weights and premature birth [28].

***(d) Lead*:** Lead (Pb) is used in storage batteries, cable coverings, plumbing, ammunition, manufacture of tetraethyl Pb, sound absorbers, radiation shields around X-ray equipment and nuclear reactors, paints, while the oxide is used in producing fine "crystal glass" and "flint glass" with a high refractive index for achromatic lenses, solder and insecticides. Lead enters the human body in many ways. It can be inhaled in dust from lead paints, or waste gases from leaded gasoline. It is found in trace amounts in various foods, notably fish, which are heavily subjected to industrial pollution Plants can absorb Pb from soils and from a PbEt4 traffic-induced air pollution (90 % of total Pb emissions into the atmosphere). Pb can contaminate water and consequently enter the

aquatic food chains [29].

***(e) Selenium*:** Dietary selenium (Se) supplementation with different origin and chemical forms is generally use for overcoming selenium deficiency and maintaining high productive and reproductive performance of farm animals. Excess amount of selenium is found as pro-oxidant and can be toxic for all animal species and man depending on the dose and duration of intake. The mechanism of selenium toxicity not known exactly but there are several proposals as oxidative stress mechanism, which supported by in vitro and in vivo results [30].

***(f) Mercury*:** Mercury (Hg) and its compounds are highly toxic, especially methylmercury - a potent neurotoxin. It has caused a significant number of human fatalities in several accidents around the world. Due to its wide dispersion through the atmosphere, Hg is considered a global pollutant, being deposited even in remote pristine aquatic systems, where it is biomagnified through the food chain. Hg and its compounds are highly toxic, have wide dispersion through the atmosphere. It is biomagnified through the food chain. Hg use in dental amalgams, thermometers, barometers, and the development of large-scale industrial processes (e.g. chlor-alkali plants and PVC production) and release into the environ ment. Hg occurs in nature in mineral, cinnabar, metacinnabar and hypercinnabar. Diet can be the main source of inorganic and organomercurials especially seafood while dental amalgams are the main exposure source to elemental Hg. Mercury is organomercurial in the form of methylmercury, which have toxicological uniqueness. Mercurial fungicides treated wheat seeds cause poisoning and death of 5,000 to 50,000 people. Tokuomi *et al.* [31] were the first to describe the symptoms of methylmercury poisoning. Thus, the symptoms were named the Hunter-Russell syndrome. Elemental Hg can be oxidized to Hg^{2+} that accumulates preferentially in the kidneys. The increased excretion of low molecular-weight proteins confirmed at low-level exposure, and related to damage to the renal tubes. It is a potent neurotoxin to human due to their ability to cross the blood-brain barrier. It is absorbed in the gastrointestinal tract, immediately entering the blood stream. It readily passes the placental barrier affecting the developing nervous system of the foetus.

***(g) Lithium*:** Lithium (Li) is transferred in the food chain from soils via flora and fauna to human beings. Till date, it is not measured as an essential element for animal and human. The postulated normative lithium necessities amount to < 100 μg Li/day in man. Numerous actions of lithium are significant for its therapeutic effects. These complex effects stabilize neuronal activities, support neuronal plasticity and provide neuroprotection. Three interacting systems appear most vital: (a) modulation of neurotransmitters by Li likely readjusts balances between excitatory and inhibitory activities and thus may contribute to neuroprotection; (b) Li modulates signals impacting on the cytoskeleton, a dynamic system contributing to neural plasticity. (c) Li adjusts signaling activities regulating second messengers, transcription factors and gene expression. Neuroprotective effects may be derived from its inflection of gene expression. These findings recommend that Li may exert some of its long term beneficial effects in the treatment of mood disorders via underappreciated neuroprotective effects [32].

***(h) Nickel*:** Water-insoluble nickel compounds including nickel sulfides, disulfides, and oxides readily enter the cell and are very potent carcinogens [33]. In contrast, water-soluble nickel compounds including acetate, chloride, nitrate, and sulfate do not enter the cells as readily as water-insoluble nickel compounds [34]. The increase in the usage of nickel compounds and the spread of nickel due to its dissolution from nickel ore-bearing rocks are the main causes of nickel presence in the environment. The primary source of nickel in drinking-water is the leaching of metals in water network [34]. However, food is the major source of nickel exposure in the non-smoking, non-occupationally exposed population, but nickel absorption from water, was significantly higher than absorption of nickel from beverages like tea, coffee, or orange juice and milk [35]. Ni2+ induces carcinogenesis through several processes including DNA hypermethylation (H3K9 mono- and dimethylation), DNMT inhibition, DNA mutation, ROS generation, inhibiting histone H2A, H2B, H3 and H4 acetylation, converting the tumor suppressor genes to the heterochromatin, and substantial increases of the ubiquitination of H2A and H2B [36]. Therefore, nickel plays an important role in the suppression or silencing of genes.

***(i) Chromium*:** Trivalent chromium is an epigenetic carcinogen factor since it can form stable compounds with macromolecules such as DNA and cysteine residue of proteins and glutathione [37]. The trivalent form of chromium cannot pass the cell membrane; however, the hexavalent salts are able to enter the cell and are converted to the trivalent form [38]. Thus, depending on the situation, reducing agents can affect carcinogenic properties of chromium and inside the cell, chromium (VI) can be converted to a carcinogen. During Cr (VI) reduction, many compounds such as oxygen radicals, DNA inter-strand cross links (ICLs), and single-strand breaks (SSBs) may form. ICLs act as physical barriers to DNA replication and transcription events, thus inducing apoptosis [39]. The chromium carcinogenicity, particularly in lung epithelial cells and fibroblasts, is imposed through hypermethylation of CYP1a1 promoter. Chromium recruits histone deacetylase 1 (HDAC1) and DNMT1, especially to CYP1a1 promoter, and this assembly recruits BP1 and inhib its CYP1a1 gene expression [39]. CYP1A1 is important in the metabolism of carcinogens such as polycyclic aromatic hydrocarbons (PAHs) and heterocyclic amines that are widely distributed widely in our environment through

auto mobile exhausts, cigarette smoke, charcoal-broiled cooking, and industrial waste. In contrast to other cytochrome P450 enzymes such as epoxide hydrolase and dihydrodiol dehydrogenase that are involved in PAH- and Benzo(a)pyrene induced carcinogenesis, CYP1A1 inhibits PAH carcinogenesis. Thus, inhibition of CYP1A1 by chromium leads to the production of a PAH [38]. PAHs have an important role in the activation of cytosolic ligand-activated transcription fac tor named aromatic hydrocarbon receptor (AhR) [40]. After formation, the PAH-AhR complex is transferred into the nucleus. In the nucleus, PAH is detached from the complex and AhR binds to its nuclear partner, Arnt. This new complex acts as a transcription factor and interacts with DRE of CYP1A1 gene, leading to the activation of CYP1A1 gene expression, thus causing bioactivation of exogenous procarcinogens of both hepatocellular and lung carcinomas [41]. It is interesting that PAH through binding to transcription factor Ah R, activates CYP1a1 gene expression, and CYP1A1 inhibits PAH carcinogenesis, but in the presence of Cr, the promoter of CYP1a1 is inactivated and PAH can act as carcinogens. Benzo(α) pyrene is also a member of polycyclic aromatic hydrocarbon (PAHs) family that is metabolically transformed from its pro-carcinogenic status to the carcinogenic metabolite (BP-7,8-dihydrodiol-9,10- epoxide (BPDE)), that can bind covalently to DNA and form BPDE–DNA adducts and reactive oxygen species. BPDE activates apoptosis through p53 –independent and – dependent manner [42]. P53 dependent Cr-induced apoptosis takes place by increasing p53 phosphorylation at serine 392, as well as up-regulation of pro-apoptotic gene bcl-XS, and caspase-7, and down-regulation of several anti-apoptotic genes from Bcl2-family (bcl-W and bcl-XL), and bax. These apoptotic events result in the destruction of the mitochondria and release of cytochrome c [43-45]. Moreover, Cr induces the ATM protein production, which phosphorylates and acti vates Chk2 protein. The phosphorylated Chk2 in turn phos phorylates and activates p53. The phosphorylated p53 does not bind to MDM2 protein [45]. Cr exposure at very high concentrations activates all subclasses of MAPK through phosphorylation; therefore, Cr acts as a MAPK kinase and increases survival/proliferation in a dose-dependent manner. This function is associated with its ability in ROS generation [46].

(3) Bioremediation Approach for Heavy Metal Pollution in Soil, Water and Air

Bioremediation has been considered as one of the safer, cleaner, cost effective and environmental friendly technology for decontaminating sites which are contaminated with wide range of pollutants [47]. The term bioremediation has been introduced to describe the process of using biological agent to remove toxic waste from environment. Bioremediation is the most effective management tool to manage the polluted environment and recover contaminated soil [47]. The process of bioremediation uses various agents such as bacteria, fungi, algae and higher plants as major tools in treating heavy metals present in the environment [3, 47-49]. Bioremediation, both In situ and ex-situ have also enjoyed strong scientific growth, in part due to the increased use of natural attenuation, since most natural attenuation is due to biodegradation [48]. Bioremediation and natural attention are also seen as a solution for emerging contaminant problems. Microbes are very helpful to remediate the contaminated environment [47, 48]. Soil heavy metal pollution is difficult to control due to its strong toxicity, wide distribution, and easy transformation [49]. The remediation of heavy metal contaminated soil is a long-term and arduous process. Remediation technology is also constantly developing, from a single remediation with a single effect to a combination of advantages. The restoration is shifting from high-cost technologies with obvious side effects to low-cost, green and environmentally-friendly technologies, and new and more efficient restoration materials are continuously synthesized from ordinary materials. Among all remediation technologies, compared with physical and chemical remediation methods, microbial remediation can be carried out in situ, saves treatment costs, has little impact on the environment, is environmentally friendly and economical, is an enhancement of natural processes and thus generally does not produce secondary pollution, so it has a wide range of application prospects. But meanwhile, there are disadvantages such as long time consumption, specificity in most cases, and small remediation scope. Therefore, when selecting remediation technology, environmental, time, and economic factors should be considered comprehensively to choose the most suitable remediation plan [3, 47-51].

Mercury removal from synthetic wastewater using a bioreactor has been systematically documented [52]. The wastewater bioremediation is reliant on various factors e.g. pH [53]. The pH affects their bioavailability by affecting the solution chemistry through processes like complexation, hydrolysis, redox as well as precipitation [54]. Microbial biomass surface area and pretreatment processes (modifying the surface area) tend to influence the bioremediation process [54]. Microbial biomass may be required to be immobilized in matrices like alginate and silica gel to develop a suitable commercial biosorbent with appropriate strength and porosity [55]. Encapsulation commences physicochemical stability as well as heat resistance. Encapsulated *Agrobacterium sp*. in alginate with nanoparticles of Fe has revealed excellent adsorption ability for continuously five cycles [56]. Poor selectivity and hurdles in recycling biomass are some of the limitations of the process. Bioremediation has further been mediated through microbial biofilms with high resistance and tolerance for metal ions.

The air pollution of toxic heavy metals has been considered one of the most significant environmental issues which have accelerated noticeably due to altering industrial activities [57]. Various most common techniques, strategies, and biological approaches of heavy metal bioremediation have been practiced so far [57]. Besides, certain specific roles of microorganisms in the bioremediation of heavy metals in polluted air have been well discussed [58]. Advanced methods of heavy metal remediation include physicochemical and biological methods; the latter can be further classified into in situ and ex situ bioremediation. The *in situ* process includes bioventing, biosparging, biostimulation, bioaugmentation, and phytoremediation. *Ex situ* bioremediation includes land farming, composting, biopiles, and bioreactors.

(4) Bioremediation of Heavy Metals in Food Industry

Heavy metals have been known as natural elements in the Earth's crust, likely to enter human food via industrial or agricultural processing, in the form of fertilizers and pesticides [59]. These elements are basically not biodegradable. Some heavy metals are recognized as pollutants and are toxic, and their bioaccumulation in plant and animal tissues can result in undesirable effects on humans; thus, their amount in water and food should always be under control [60]. There have been always regular practices

to investigate the conditions for the bioremediation of heavy metals in foods. Various physical, chemical, and biological techniques have been used to reduce the heavy metal content in the environment. During the last decades, bioremediation procedures using plants and microorganisms have generated interest to researchers for their advantages such as being more specific and eco-friendly [61]. The major pollutant elements in foods and beverages are lead, cadmium, arsenic, and mercury, which encompass their own permissible limits. Amongst the microorganisms that are capable of bioremediation of heavy metals, *Saccharomyces cerevisiae* has been shown to be an interesting choice for its special uniqueness and being safe for humans, ultimately enabling it reasonably common and valuable in the food industry [59, 60]. Its mass production as the byproduct of the fermentation industry and the low cost of culture media are the additional advantages. The ability of this yeast to get rid of an individual separated element has also been extensively studied. In countries with high heavy metal pollution in wheat, the application of *S. cerevisiae* is an indigenous solution for overcoming the problem of solution [59].

(5) Strengths and Weaknesses of Bioremediation

Bioremediation is a straightforward procedure used by several researchers in the waste treatment process for contaminated environments such as soil and water. The microbial organisms that degrade the contaminant augment in numbers and release harmless products. The residues for the treatment are generally harmless products, namely, carbon dioxide, water and cell biomass. Bioremediation is of basically very less effort, less laborious, as well as cost effective compared to other methods that are in practice for the removal of hazardous waste. Besides, bioremediation is ecofriendly, sustainable, reasonably easy to implement, and useful for the total destruction of a wide range of contaminants [62]. Many hazardous compounds can be altered into harmless products. Furthermore, bioremediation can be implemented on the site of contamination itself without causing a principal disruption of standard activities. There is no need of transporting huge numbers of waste off-site, there is no latent human health risk, and the environment will continue uncontaminated. The majority of the disadvantages of bioremediation narrate to it requiring a longer time to be accomplished as compared with other options, namely, excavation and removing pollutants from the site. Moreover, there is a complexity of bioremediation in treating inorganic contaminants and in ascertaining whether contaminants have been perfectly destroyed or not. In addition, there is a sluggishness of highly chlorinated materials biodegradation and generation of extra toxic or carcinogenic by-products [63]. Lastly, the products of biodegradation sometimes become more toxic than the original compound. Its biological processes are also highly specific with efficient site factors including the presence of microbial populations, growth conditions, and quantity of nutrients concomitant with pollutants [58, 64, 65].

II. Conclusion

Bioremediation technique is still a useful, natural, and environmentally friendly process in which the polluted environment is biologically biodegraded. Microorganisms play a pivotal role in the removal of heavy metals pollutants. The heavy metals (e.g., mercury, silver, lead, cadmium, and arsenic) exert toxic effects on living cells. Examples of degradative aerobic bacteria are *Pseudomonas*, *Alcaligenes*, *Sphingomonas*, *Rhodococcus*, and *Mycobacterium.* Anaerobic bacteria have also been used for the bioremediation of biphenyls, dechlorination, and chloroform. In addition, fungus microorganisms can efficiently degrade numerous toxic environmental pollutants. Bioremediation is of very less effort, less laborious, cost effective, ecofriendly, sustainable, and comparatively easy to implement. The

majority of the disadvantages of bioremediation narrate to the slowness and time-consumption; moreover, the products of biodegradation occasionally become extra toxic than the original compound. Bioremediation may be restricted by irregularity and uncertainty of totality. Furthermore, the performance evaluation of bioremediation might be complicated as there is no up to standard endpoint. Further, the major pollutant elements in foods and beverages are lead, cadmium, arsenic, and mercury, which encompass their own permissible limits. Amongst the microorganisms that are capable of bioremediation of heavy metals, *Saccharomyces cerevisiae* has been shown to be an interesting choice for its special uniqueness and being safe for humans, ultimately enabling it reasonably common and worthy in terms of '**Food Safety**' in the food processing sectors. As there is still requirement for more studies to develop bioremediation technologies in order to find more biological solutions for bioremediation of heavy metal contamination from diverse environmental systems, the collection of data concomitant with significant hypotheses together with this overview provides new insights into developing a platform to explore certain novel bioremediation experimental model that could be rapid, precise and cost effective, ultimately affecting human health.

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Conflicts of Interest

Authors declare that they have no conflicts of interest.

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