Review Form 1.6

Journal Name:	**Asian Journal of Biochemistry, Genetics and Molecular Biology**
Manuscript Number:	**Ms_AJBGMB_93278**
Title of the Manuscript:	**Chromium induced developments of diseases and their inhibitions**
Type of the Article	**Review Article**

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This journal's peer review policy states that **NO** manuscript should be rejected only on the basis of '**lack of Novelty'**, provided the manuscript is scientifically robust and technically sound.
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PART 1: Review Comments

	Reviewer's comment	**Author's comment** *(if agreed with reviewer, correct the manuscript and highlight that part in the manuscript. It is mandatory that authors should write his/her feedback here)*
Compulsory REVISION comments		
Minor REVISION comments	The author (s) should input the following corrections. **ABSTRACT** Line 4: as an essential Line 5: hormones Line 8: reduced/altered antioxidant/immune response activities, Line 11: immunotoxicity, or **INTRODUCTION** Line 1: in crust, is found Line 5: when exposed to humans mainly through inhalation, dermal contact, and Line 6: the development Line 9: DNA, RNA, and proteins, Line 10: form Line 13: formation Line 17: excessively Line 18: stress, Line 19: system; damage; formation Line 21: transcription, and Line 23: concentrations, ~~the~~ Line 24: concentrations, damage, cells, Line 26: reduction, which finally Line 28: extent Line 29: size Line 30: application, antibiotics, Line 31: electrons, neutralizing free radicals, Line 32: Some Line 33: pterostilbene, Line 34: anti-inflammatory, Line 35: immunotoxicity, **2.1 Role of chromium in pathogenesis** Line 3: to Line 4: effects Line 6: transport Line 7: causing, cytotoxic effects **2.2 Permeability of chromium into the cell membrane** Line 2: anion Line 9: phagocytosis, **2.3 Chromium-VI metabolism by intracellular / extracellular reductions** Line 1**:** Cr-IV, Line 3: allowing Line 8: (GR), Line 11: catalase, Line 12: oxygen Line 13: damage, Line 14: over-expression Line 17: formation	

	Line 18: protein, damage **2.4 Chromium-VI-induced oxidative and nitrative stresses / damages** Line 2: usage Line 3: generation Line 9: to generate Line 10: damage. stress Line 11: damage Line 14: peptides, to form Line 18: surfaces, membranes, **2.5 Cr-VI-induced DNA damage** Line 3: lesions, resulting in the formation Line 4: cross-links, and DNA-protein cross-links, succeeding in the inhibition of replication, mutagenesis, **2.6 Cr-VI-induced DNA damage with GSH** Line 1: as an antioxidant as well as a metal, group, having a high Line 2: for metals. to form Cr-V, Cr-IV, Line 3: through the donation Line 4: signal. to form Line 5: Cr-V-GSH complex then reacts with H_2O_2 to produce Line 6: via a Fenton-type reaction to lead to DNA damage. Line 8: further reacts, generate a di-sulfide Line 9: in turn, Line 12: production of HO. Line 16: phosphates, leading Line 17: replication. **2.7 Cr-VI-induced DNA damage with ascorbate** Line 7: to form dimers. Line 8: react with which Line 11: enhance the Cr-III Line 13: DNA, resulting in the resistance to dissociation **2.8 Cr-VI-induced DNA damage with antioxidant NADPH/NADH enzymes** Line 2: occur through the donation of two electrons directly to form Cr-III line 5: breaks line 8: as the glucose-6-phosphate **2.9 Cr-VI-induced DNA damage with mitochondrial electron transport chain (ETC) Enzymes** Line 1: deplete a higher **2.10 Cr-VI-induced epigenetic alterations** Line 3: genes, correlated Line 4: genes' expressions Line 8: exposed to Cr-VI Line 9: expression **2.11 Chromium-induced immune-responses and inflammations** Line 5: prosthesis-implant-exposure, indicating systemic immune-suppression. Line 10: concentrations **2.12 Cr-VI-induced mutagenicity and tumor formation** Line 1: A few Line 2: and eventual Line 5: complexes, which occur Line 6: In the presence Line 7: exposed to Cr-VI Line 8: At a lower Line 9: at a higher Line 10: non-oxidative Line 11: dose-dependent **2.13 Cr-VI-induced gene expression**	

	Line 2: exposure, and Line 3: Acute, short-term exposures (2-3 h) to high Cr-VI dosages cause different Line 4: A few investigators have performed experiments on Line 6: metabolism, and Line 13: A few Line 14: have significantly decreased Line 16: of the ErbB2 Line 17: basal level, indicating Line 18: expression. Other investigators have demonstrated that the exposure Line 27: regulate gene expression, Line 29: improve the ROS Line 30: metabolism, and **2.14 Cr-VI-induced survival signaling pathways** Line 1: A few Line 2: MAPKs, and extracellular signal-regulated Line 3: A few Line 6: has Line 8: A study Line 12: transducers of the Src Line 13: Fyn, and Line 14: cells may selectively activate Fyn and initiate an interferon-like Line 16: Other studies have Line 18: airway Line 19: studies have Line 20: may lead to increased Line 22: genes, and Line 23: MEK/ERK-independent signaling pathways. A few Line 24: that exposure to Cr-VI **2.15 Cr-VI-induced DNA-lesions repair and side effects** Line 2 and progression Line 12: may inhibit OGG1 Line 15: of the BER/APE Line 17: studies have Line 25: deletion/insertion Line 26: replication, homologous recombination (HR), methylation, base oxidation, and other Line 28: to a few types of cancer, resistance to chemotherapeutic components, and Line 30: a high Line 33: formation, implicating Line 34: exposed to Cr-VI Line 36: cycle, resulting **2.16 Cr-VI-induced neurotoxicity** Line 1: chromium has shown Line 2: neurotransmitter inhibition and neuronal cell damage Line 3: that chromium-exposure may inhibit acetylcholinesterase (AChE) activity, Line 4: leading to cholinergic and dopaminergic neuronal cell damage, astrogliosis, and Line 7: A few **3. APPLICATIONS OF THERAPEUTIC MOLECULES / CARGOS ON CHROMIUM-EXPOSED CELLS** Line 2: through the removal Line 3: metals, and Line 4: catalase (CAT), and Line 6: glutathione, and lipoic acid), melatonin, and other compounds. Line 7: that SOD Line 13: of a poorly	

	Line 14: as an antioxidant Line 15: through the conversion Line 17: acid, which is used Line 19: that vitamins C and E Line 21: A few reports have elucidated that reduced glutathione (GSH) has the Line 23: under an oxidative Line 24: oxidation Line 28: form-dihydrolipoic Line 29: exo-genous antioxidants, and Line 30: injury, and Line 32: have shown that carotenoids (β-carotene) may act as antioxidants for their Line 37: damage Line 38: cells, and Line 41: atoms Line 42: metals, preventing Line 43: concentrations, they may Line 44: cancer, and Line 47: arrest, and Line 48: A few Line 51: has Line 57: A few Line 60: as an antioxidant Line 61: expression Line 64: protecting the Line 69: tri, and Line 77: complexes-I Line 78: of the mitochondrial Line 81: damage. Line 82: A few reports have shown that propyl thiouracil is capable of reducing Cr-VI Line 84: Several other reports Line 87: A few Line 89: radicals **4. CONCLUSIONS AND FUTURE PERSPECTIVES** Line 2: mutagenesis, and Line 3: pathways, leading Line 4: neurotoxicity, and carcinogenesis, while its chronic exposure may lead to damage Line 5: as the liver, kidney, brain, and lungs, causing the development Line 7: damage, apoptosis or various signaling pathways, Line 9: concentration, including Line 10: modulate Line 12: immunotoxicity, and carcinogenicity, including Line 13: tissues, or Line 15: insolubility, and drug-resistance, and to target formulated low quantities Line 17: pharmacokinetics, and	
Optional/General comments		

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PART 2:

	Reviewer's comment	**Author's comment** *(if agreed with reviewer, correct the manuscript and highlight that part in the manuscript. It is mandatory that authors should write his/her feedback here)*
Are there ethical issues in this manuscript?	*(If yes, Kindly please write down the ethical issues here in details)*	

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