RESEARCH ARTICLE

Genomic insights into chromium resistance mechanisms in *Citrobacter* spp.

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Cr(VI) is a highly toxic environmental pollutant, and microbial reduction represents a promising bioremediation strategy. This study investigated the Cr(VI)-reducing capability of *Citrobacter freundii* A1B10 and elucidated its genomic determinants. When *C. freundii* A1B10 was inoculated in LB medium containing 8.5 mg/L Cr(VI), the residual Cr(VI) was 3.48 ± 1.31 mg/L after 40 h incubation. Whole-genome sequencing of *C. freundii* A1B10 showed 4,927,083 bp with 4,631 coding genes and a GC content of 52.11%. Genomic analysis identified critical determinants for Cr(VI) resistance including sulfate (*ettA*, *cysA*, *cysW*, *cysU*, *cysP*), phosphate (*pstS*, *pstC*, *pstA*, *pstB*, *phoU*), and molybdate (*modF*, *modC*, *modE*, *modB*, *modA*) transporters, which implicated that CrO_4^{2-} was taken *via* competitive ion transport. Reduction pathways involved chromate reductase (*chrR*), cytochrome c (*ccmH*, *ccmF*, *ccmE*, *ccmG*, *nrfE*, *napC*, *napB*, *nrfB*, *torC*, *torA*, *torY*, *torZ*, *fbcH*, *petA*), nitrite reductases (*nirD*, *nirB*, *nrfA*), proton transport proteins (*atpB*, *atpE*, *atpF*, *atpH*, *atpA*, *atpG*, *atpD*, *atpC*), and cytochrome oxidases (*appX*, *cydB*, *cydA*, *cydX*, *cyoD*, *cyoB*, *cyoA*, *cyoC*). These systems collectively facilitated extracellular proton/electron transfer and intracellular Cr(VI)-to-Cr(III) reduction, thereby mitigating chromium toxicity. This study provided a genomic blueprint for *C. freundii* A1B10 of Cr(VI) resistance, highlighting multi-channel transport and enzymatic reduction mechanisms.

Keywords: Cr(VI); *Citrobacter freundii*; whole-genome sequencing; reduction.

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Introduction

Chromium (Cr), a critical industrial metal, is extensively utilized in electroplating, tanning, metallurgy, and pigment manufacturing, resulting in annual emissions of 250 pounds into the atmosphere and 64,500 pounds into aquatic systems [1]. Among its compounds, hexavalent chromium (Cr(VI)) has emerged as a significant environmental and health hazard due to its strong oxidizability, high mobility, and bioaccumulation potential [2]. Cr(VI) enters ecosystems *via* industrial wastewater, exhaust gases, and solid waste, persisting in soil and aquatic environments [3]. Toxicological studies revealed that Cr(VI) toxicity originated from its ability to penetrate cell membranes, where it generated reactive oxygen species (ROS), triggering DNA strand breaks, protein oxidation, and lipid peroxidation, ultimately resulting in apoptosis or carcinogenesis [4, 5]. To address chromium contamination, conventional physicochemical remediation technologies including chemical reduction, adsorption

precipitation, and ion exchange have been widely employed [6]. Chemical reduction methods utilize reducing agents such as Fe^{2+} and sodium thiosulfate ($Na_2S_2O_5$) to convert Cr(VI) into less toxic Cr(III) [7, 8]. However, excessive reductants may induce secondary pollution [9]. Adsorption techniques, while effective for Cr(VI) removal from aqueous systems, face challenges such as high regeneration costs of common adsorbents like activated carbon and nanoscale zero-valent iron and the risk of heavy metal re-release during spent adsorbent disposal [10]. These limitations have driven researchers to explore more sustainable microbial remediation strategies.

The cornerstone of microbial remediation technology lies in leveraging the metabolic capabilities of specific bacterial strains to achieve efficient detoxification of Cr(VI) [11]. Research on microbial chromium resistance mechanisms serves as the core driver for advancing microbial remediation strategies for heavy metal pollution [12]. Currently identified microbial chromium resistance mechanisms include biosorption, biological reduction, and biomineralization [13-15]. For example, *Aspergillus niger* immobilizes Cr(VI) by forming stable complexes *via* polysaccharides and proteins on hyphal surfaces [16]. *Shewanella oneidensis* MR-1 reduces Cr(VI) to Cr(III) through a cytochrome c mediated extracellular electron transport chain [17]. *Pseudomonas putida* catalyzes the stepwise reduction of Cr(VI) *via* the NADPH-dependent ChrR enzyme with its antioxidant system mitigating oxidative stress during the process [18]. Under heavy metal stress, *Ochrobactrum tritici* 5bvl1 synthesizes polyphosphate to bind Cr(III), forming insoluble extracellular phosphate complexes that enhance community resistance [19]. By elucidating chromium resistance mechanisms in metal-tolerant microorganisms, researchers can optimize strain screening, genetic engineering, and remediation process design [20]. The revelation of these microbial chromium resistance mechanisms significantly enhance the efficiency, stability, and applicability of microbial remediation technologies, driving their development for practical environmental applications [21]. In recent years, *Citrobacter* spp. has garnered significant attention in heavy metal pollution remediation due to its exceptional hexavalent chromium (Cr(VI)) reduction efficiency and remarkable environmental adaptability [22]. However, current research predominantly focuses on phenotypic characterization, while systematic elucidation of the molecular mechanisms underlying Cr(VI) reduction and the regulatory networks of resistance genes remain scarce. This knowledge gap critically hinders its application in engineered remediation systems.

This study employed whole-genome analysis to elucidate Cr(VI) uptake, efflux, and intracellular-extracellular reduction mechanisms in the chromium-resistant *C. freundii* A1B10. The results of this study provided a theoretical reference for systematically elucidating the chromium resistance mechanisms of chromium-resistant microorganisms. Meanwhile, this study also provided possible molecular targets for molecular biology approaches to optimize the chromium-resistant performance of *Citrobacter* spp.

Materials and methods

Strain and growth medium

Citrobacter freundii A1B10 was purchased from China General Microbiological Culture Collection Center (CGMCC, Beijing, China) with the CGMCC Number of 1.12836. Luria–Bertani (LB) medium was used as the basal growth medium comprising 10 g tryptone, 5 g yeast extract, and 10 g NaCl in one liter volume. All reagents were sourced from Sinopharm Chemical Reagent Co. (Shanghai, China). LB media were adjusted to pH 7.2 prior to autoclaving, and the bacteria were incubated at 30 ± 1°C.

Determination of Cr(VI) reduction efficiency

C. freundii A1B10 was cultured in Cr(VI)-supplemented LB medium at 30 ± 1°C under shaking conditions of 180 rpm for 96 h. Uninoculated medium served as the abiotic

control. Residual Cr(VI) concentrations were quantified using the diphenylcarbazide spectrophotometric method [23]. An appropriate amount of colorless sample was taken, diluted to the mark in a 50 mL colorimetric tube, followed by addition of 0.5 mL sulfuric acid solution and 0.5 mL phosphoric acid solution, then 2 mL diphenylcarbazide chromogenic reagent. Absorbance was measured at 540 nm using a Thermo Scientific Multiskan SkyHigh Spectrophotometer (Thermo Fisher Scientific, Waltham, Massachusetts, USA). A calibration curve (R^2 = 0.9999) was generated using Cr(VI) standards (0 – 20 µg/mL).

Genomic DNA extraction

C. freundii A1B10 was cultivated in LB medium at 30 ± 1°C, 180 rpm, for 24 h. The bacterial cells were harvested by centrifugation at 1,500 g for 10 min at 4°C with subsequent removal of supernatant. Bacterial DNA was purified using the Quick-DNA Miniprep Plus Kit (Zymo Research, Tustin, California, USA) with purity and integrity being assessed by agarose gel electrophoresis and quantified *via* Qubit 4.0 Fluorometer (Thermo Fisher Scientific, Waltham, Massachusetts, USA).

Library construction and sequencing

Libraries for single-molecule real-time (SMRT) sequencing were constructed with an insert size of 10 kb using the SMRTbell® Template Prep Kit (version 1.0) (PacBio, Menlo Park, California, USA). Sequencing libraries were generated using NEBNext® Ultr™ DNA Library Prep Kit for Illumina (NEB, Omaha, Nebraska, USA) following manufacturer's instructions. The genome of *C. freundii* A1B10 was sequenced by Novogene (Sacramento, CA, USA).

Whole genome map

GeneMarkS (version 4.17) (http://topaz.gatech.edu/GeneMark/) was used to predict the coding genes of the sequenced genome, while Clusters of Orthologous Groups (COG) (http://www.ncbi.nlm.nih.gov/COG/) was employed to predict gene functions. The assembled genomic sequences derived from sequenced samples were integrated with coding gene prediction results and visualized using Circos software (version 0.69) (https://circos.ca/) , ultimately generating a comprehensive whole genome map.

Results

Cr(VI) reducing capacity of *C. freundii* A1B10

The results demonstrated that, after 40 h incubation of LB media containing 8.5 mg/L Cr(VI) with/without inoculated *C. freundii* A1B10, the residual Cr(VI) concentration was reduced from 8.5 mg/L to 3.48 ± 1.31 mg/L in the LB media with *C. freundii* A1B10, while the residual Cr(VI) concentration remained basically unchanged in the LB media without *C. freundii* A1B10 (Figure 1). The results confirmed that *C. freundii* A1B10 had the ability to reduce Cr(VI).

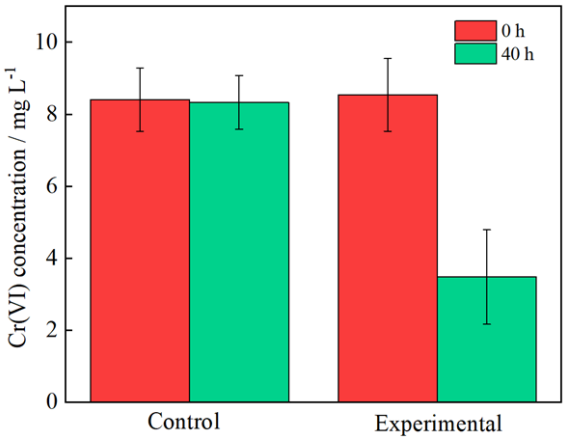

Figure 1. The Cr(VI) reduction in LB media containing Cr(VI) concentration of 8.5 mg/L without inoculated *C. freundii* A1B10 (Control) and with inoculated *C. freundii* A1B10 (Experimental).

Whole-genome sequencing of *C. freundii* A1B10

The results showed that the estimated genome size was 4,927,083 base pairs (bp) with a gene number of 4,631 and a GC content of 52.11%. A total of 4,308 genes from *C. freundii* A1B10 strain were annotated with COG annotation, accounting for 93.03% of the total number of genes. The number of genes with unknown functions was the highest with a total of 323

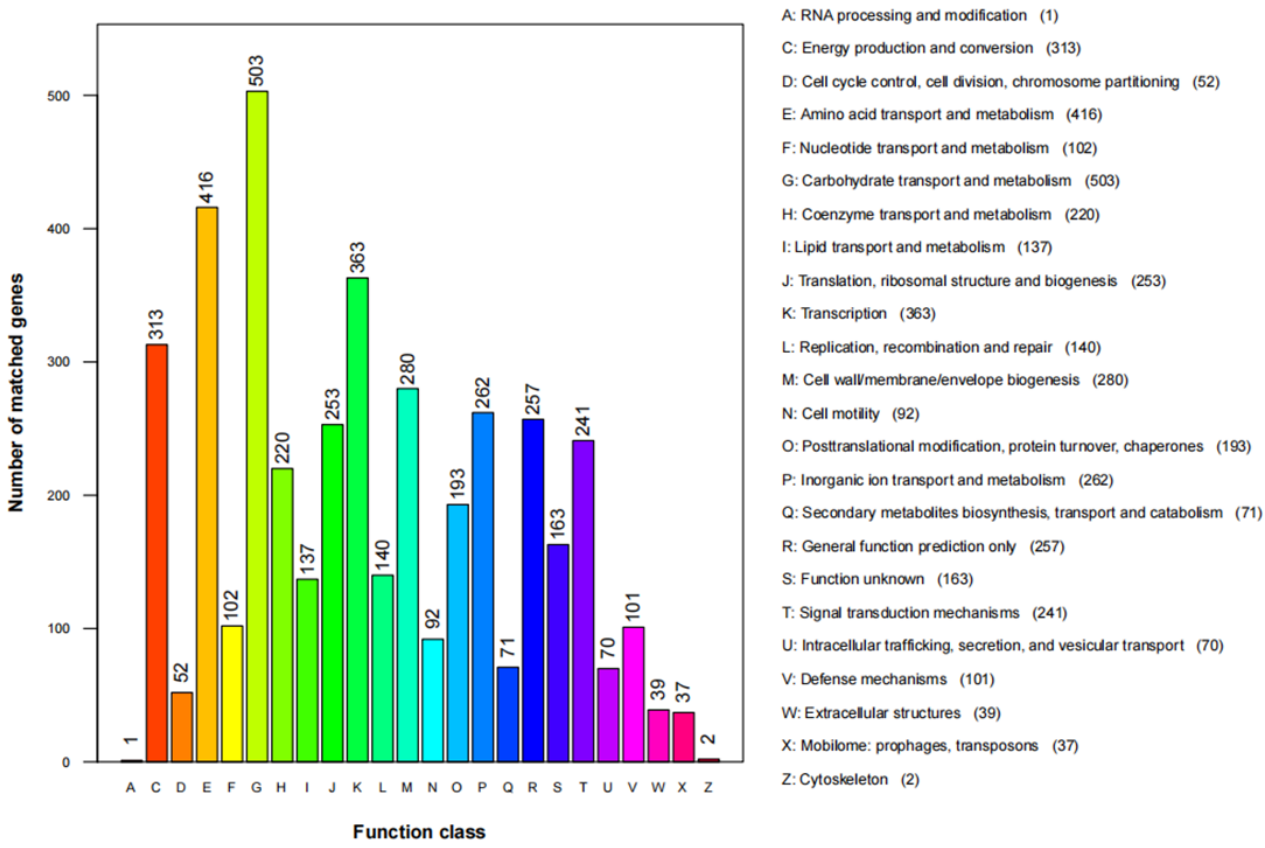

Figure 2. Gene function of *C. freundii* A1B10 based on COG database.

genes, accounting for 6.97% of the annotated genes. The functional classification of COGs related to heavy metal reduction was analyzed, and the results revealed that energy production and conversion, amino acid transport and metabolism, carbohydrate transport and metabolism, coenzyme transport and metabolism, translation, ribosomal structure and biogenesis, transcription, cell wall/membrane/envelope biogenesis, inorganic ion transport and metabolism, general function prediction only, signal transduction mechanisms accounted for 313, 416, 503, 220, 253, 363, 280, 262, 257, 241 genes, respectively, representing 7.27%, 9.66%, 11.68%, 5.11%, 5.88%, 8.43%, 6.50%, 6.08%, 5.97%, 5.59% of the total number of annotated genes (Figure 2). The whole-genome map of *C. freundii* A1B10 genes was then drawn (Figure 3). The whole-genome DNA sequence of *C. freundii* A1B10 was submitted to National Center for Biotechnology Information (NCBI) (https://www.ncbi.nlm.nih.gov/) with the BioSample accession number of SAMN47739824.

Cr(VI) resistance genes of *C. freundii* A1B10

Previous studies have identified multiple molecular determinants implicated in microbial Cr(VI) resistance mechanisms including sulfate transporters, phosphate transporters, molybdate uptake systems, chromate reductase, cytochrome c complexes, nitrite reductase, and proton-coupled electron transport systems [24-26]. Guided by this mechanistic framework, a comprehensive genomic survey was conducted to delineate the Cr(VI) resistance determinants in *C. freundii* A1B10. The results showed that the sulfate transport system proteins were encoded by genes *ettA*, *cysA*, *cysW*, *cysU*, and *cysP* in *C. freundii* A1B10. The phosphate transport system proteins were encoded by genes *pstS*, *pstC*, *pstA*, *pstB*, and *phoU* in *C. freundii* A1B10. Similarly, molybdate transport system components were

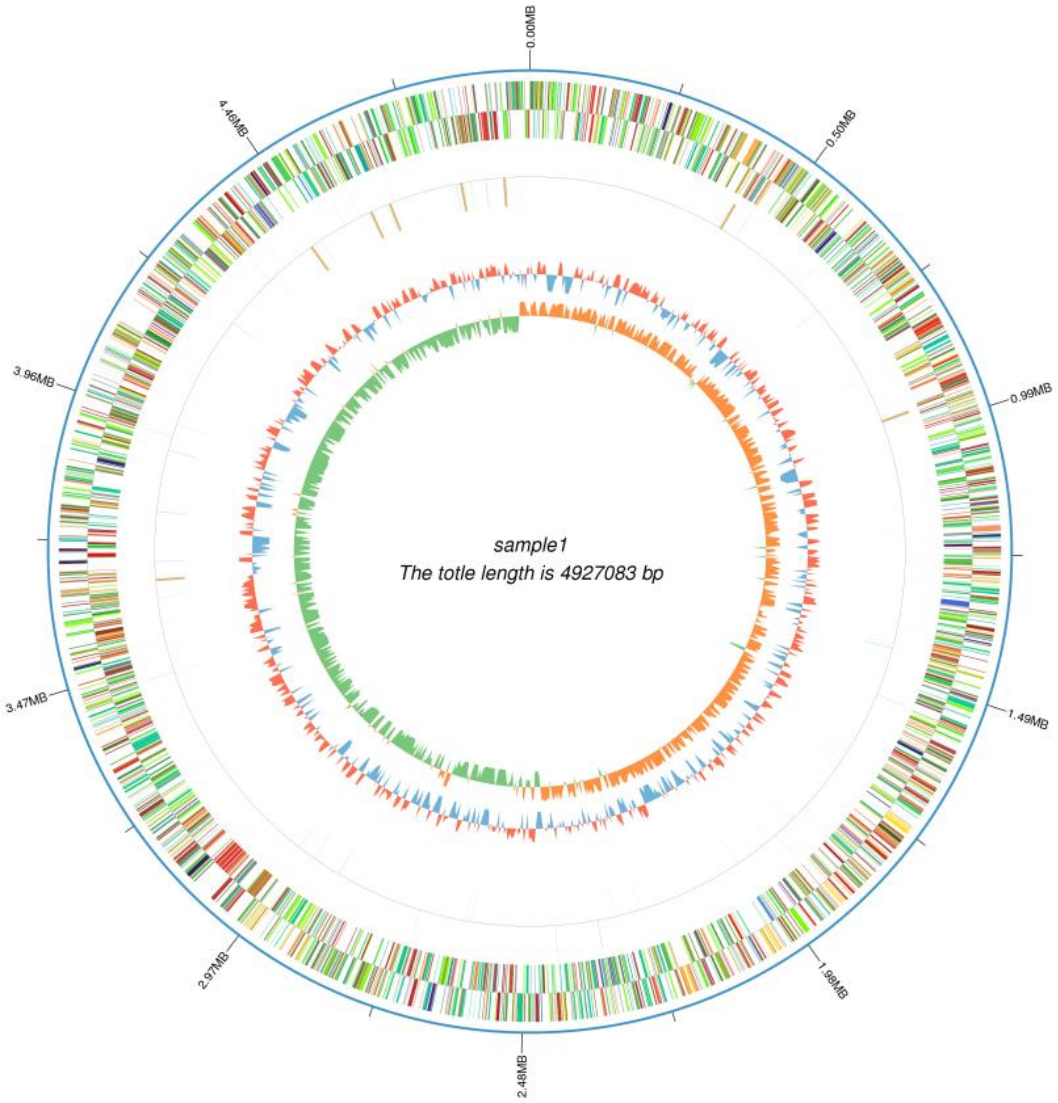

Figure 3. Whole genome map *C. freundii* A1B10. The visualization displayed genomic position coordinates in the outermost layer with successive inward layers representing functional gene annotations (COG), ncRNA distributions, and genomic GC content profiles from periphery to center.

encoded by the genes *modF*, *modC*, *modE*, *modB*, and *modA* in *C. freundii* A1B10. Chromate reductase production was directed by *chrR*, while nitrite reductase synthesis involved the *nirD*, *nirB*, and *nrfA* genes in *C. freundii* A1B10. Genes *ccmH*, *ccmF*, *ccmE*, *ccmG*, *nrfE*, *napC*, *napB*, *nrfB*, *torC*, *torA*, *torY*, *torZ*, *fbcH*, and *petA* in *C. freundii* A1B10 could encode cytochrome c. Genes *atpB*, *atpE*, *atpF*, *atpH*, *atpA*, *atpG*, *atpD*, and *atpC* in *C. freundii* A1B10 could encode proton transport proteins. The cytochrome oxidase systems in *C. freundii* A1B10 demonstrated distinct genetic organization with cytochrome bd oxidases being encoded by the genes *appX*, *cydB*, *cydA*, and *cydX*, while cytochrome o oxidases production involving the genes of *cyoD*, *cyoB*, *cyoA*, and *cyoC* (Table 1).

Discussion

In this study, *C. freundii* A1B10 was able to reduce Cr(VI), which was the same as that of *Citrobacter* sp. reported by Li *et al*. [22]. This might involve complex intracellular and extracellular reduction mechanisms [6, 14]. Therefore, the genes related

Table 1. Chromium resistance related genes.

Gene name	Gene production	Gene function
ettA	sulfate-transporting ATPase	Sulfate transport
cysA	sulfate transport system ATP-binding protein	
cysW, *cysU*	sulfate transport system permease protein	
cysP	sulfate transport system substrate-binding protein	
pstS	phosphate transport system substrate-binding protein	Phosphate transport
pstC, *pstA*	phosphate transport system permease protein	
pstB	phosphate transport system ATP-binding protein	
phoU	phosphate transport system protein	
modF, *modC*	molybdate transport system ATP-binding protein	Molybdate transport
modE, *modB*	molybdate transport system regulatory protein	
modA	molybdate transport system substrate-binding protein	
chrR	chromate reductase, NAD(P)H dehydrogenase (quinone)	Chromate reductase
nirD	nitrite reductase (NADH) small subunit	Nitrite reductase
nirB	nitrite reductase (NADH) large subunit	
nrfA	nitrite reductase (cytochrome *c*-552)	
ccmH, *ccmF*, *ccmE*, *ccmG*, *nrfE*, *napC*, *napB*, *nrfB*	cytochrome *c*-type protein	Cytochrome *c*
torC, *torA*, *torY*, *torZ*	trimethylamine-N-oxide reductase (cytochrome *c*), cytochrome *c*-type subunit	
fbcH	ubiquinol-cytochrome *c* reductase cytochrome *b*/*c*1 subunit	
petA	ubiquinol-cytochrome *c* reductase iron-sulfur subunit	
atpB atpE atpF, *atpH*, *atpA*, *atpG*, *atpD*, *atpC*	F-type H+-transporting ATPase subunit a	Proton transport
appX, *cydB*, *cydA*, *cydX*	cytochrome *bd*-II ubiquinol oxidase subunit	Electronic efflux
cyoD, *cyoB*, *cyoA*, *cyoC*	cytochrome *o* ubiquinol oxidase subunit IV	

to chromium resistance in *C. freundii* A1B10 were analyzed by whole genome sequencing to elucidate this mechanism.

Transmembrane transport is a critical pathway for Cr(VI) entry into biological cells. Previous studies have demonstrated that Cr(VI) enters cells in the form of chromate ions (CrO_4^{2-}) through inorganic anion transport pathways, leading to DNA damage and cellular metabolic dysfunction *via* complex competitive inhibition effects [27]. The mechanism by which CrO_4^{2-} enters cells *via* sulfate transport systems is well established [28]. Due to the structural similarity between CrO_4^{2-} and sulfate ions (SO_4^{2-}), CrO_4^{2-} competes with SO_4^{2-} for transport carriers to gain intracellular access [29]. Notably, CrO_4^{2-} transmembrane transport exhibits multi-channel characteristics. Plant studies have confirmed that CrO_4^{2-} can enter cells through phosphate transporters [30]. Compared to $H_2PO_4^-$, CrO_4^{2-} shows higher chemical structural compatibility with phosphate transporters and forms stable binding interactions, facilitating its entry *via* these transporters [31]. Additionally, CrO_4^{2-} and molybdate ions (MoO_4^{2-}) share structural similarities, both adopting tetrahedral configurations. The Mo-O bond length (1.79 Å) is comparable to the Cr-O bond length (1.686 Å), suggesting potential involvement of molybdate transport systems in cellular Cr(VI) uptake [32, 33]. Studies indicated that the expression levels of microbial molybdate transporter genes *modA*, *modB*, *modC* were significantly upregulated in the presence of Cr(VI) [34], which implied that CrO_4^{2-} might enter cells *via* molybdate transporters, while microorganisms might upregulate molybdate transporter expression to compensate for the demand of MoO_4^{2-} transport channels to maintain molybdenum homeostasis. Therefore, in *C. freundii* A1B10, the genes encoding sulfate transporters (*ettA*, *cysA*, *cysW*,

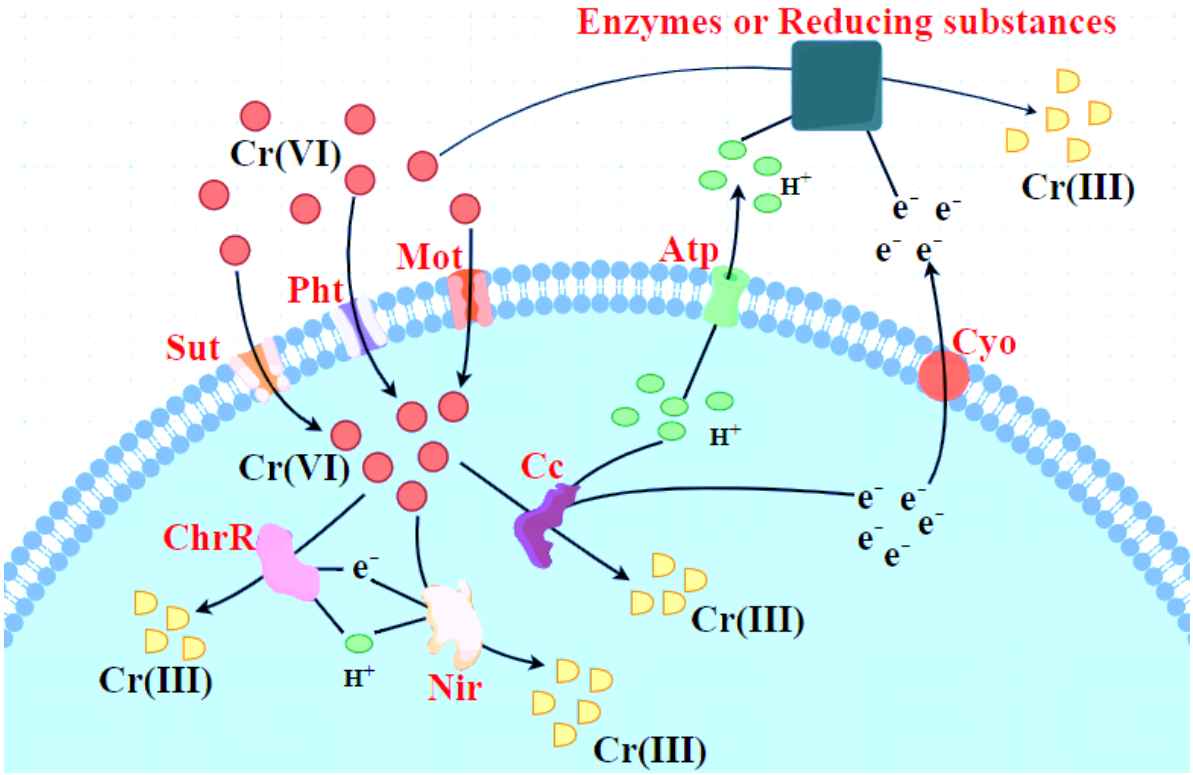

Figure 4. Schematic model of *C. freundii* A1B10 to reduce Cr(VI). Sut: sulfate transport system protein. Pht: phosphate transport system protein. Mot: molybdate transport system protein. Atp: proton transport protein. Cyo: cytochrome oxidase. ChrR: chromate reductase. Nir: nitrite reductase. Cc: cytochrome c.

cysU, *cysP*), phosphate transporters (*pstS*, *pstC*, *pstA*, *pstB*, *phoU*), and molybdate transporters (*modF*, *modC*, *modE*, *modB*, *modA*) are associated with cellular Cr(VI) uptake.

Microbial reduction of Cr(VI) can be categorized into extracellular and intracellular reduction. Numerous studies have demonstrated that most microorganisms can secrete chromate-reducing enzymes to reduce highly toxic Cr(VI) to less toxic Cr(III) [35, 36]. Regardless of whether intracellular reduction or extracellular reduction occurred, the process of Cr(VI) reduction by microorganism requires the participation of H^+ and electrons [37]. The genes *atpB*, *atpE*, *atpF*, *atpH*, *atpA*, *atpG*, *atpD*, *atpC* can encode proton transport proteins in *C. freundii* A1B10. The genes *appX*, *cydB*, *cydA*, *cydX*, *cyoD*, *cyoB*, *cyoA*, *cyoC* can encode cytochrome oxidase in *C. freundii* A1B10. These genes may play an important role in maintaining intracellular and extracellular proton and electron balance and promoting extracellular hexavalent chromium reduction. However, there are few reports on extracellular chromate reductase. It has been found that cytochrome c, quinone oxidoreductase ChrR and nitrite reductase have chromate reducing activity in the cell, which can reduce Cr(VI) to Cr(III). Therefore, the ChrR encode gene *chrR*, the cytochrome c encode genes *ccmH*, *ccmF*, *ccmE*, *ccmG*, *nrfE*, *napC*, *napB*, *nrfB*, *torC*, *torA*, *torY*, *torZ*, *fbcH*, *petA*, and the nitrite reductase encode genes *nirD*, *nirB*, *nrfA* in *C. freundii* A1B10 may play the role of reducing Cr(VI) to Cr(III) in the cell to reduce the toxicity of Cr(VI) to cells. Based on the above analysis, the mechanism diagram of reduction of Cr(VI) by *C. freundii* A1B10 was speculated (Figure 4). The sulfate transporters (Sut), phosphate transporters (Pht), and molybdate transporters (Mot) are associated with cellular Cr(VI) uptake. The proton transport proteins (Atp) and cytochrome oxidase (Cyo) play

an important role in maintaining intracellular and extracellular proton and electron balance and promoting extracellular hexavalent chromium reduction. The ChrR, cytochrome c (Cc), and nitrite reductase (Nir) play the role of reducing Cr(VI) to Cr(III) in the cell.

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