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Short term effects of crude oil, vanadium and nickel intoxication on rats liver antioxidant defence system

Abstract. Lipid peroxidation (LPO) and alterations in cellular systems protecting against oxidative damage were determined in the liver of male albino rats, 4 weeks after intraperitoneal (i.p) injection of 0.5 ml/kg bw of crude oil and/or oral consumption of 150 mg V /L or 180 mg Ni /L. After subjecting the rats to crude oil or vanadium or nickel, LPO level and serum alanine-(ALT) and aspartate-(AST) transaminases activities were raised ($p<0.01$) but the antioxidant enzymes, superoxide dismutase (SOD) and catalase (CAT) activities were decreased ($p<0.01$) significantly. Significant increase ($p<0.001$) was observed in the glutathione content in nickel-treated rats whereas glutathione content in crude oil or vanadium-treated rats was significantly ($p<0.001$) inhibited. Glutathione-S-transferase (GST) activity was found to be increased ($p<0.001$) after crude oil exposure while vanadium or nickel exposure decreased ($p<0.001$) the GST activity. In conclusion, crude oil, vanadium and nickel treatments caused profound cell damage as indicated by increased LPO in liver and leakage of intracellular enzymes, ALT and AST to the blood; concomitant exposure to crude oil and vanadium exacerbated this effect in a synergistic manner. Stimulation of GSH synthesis by administration of nickel reduced crude oil induced toxicity. The toxicity induced by simultaneous administration of vanadium, nickel and crude oil is less than single toxicity of these metals and crude oil.

Keywords: *c*rude oil, vanadium, nickel, antioxidant enzymes, lipid peroxidation, rat.

Oil is a necessity in our industrial society, and a major sustainer of our lifestyle. Crude oil is composed of complex mixtures of a vast number of individual chemical compounds, the bulk of which are hydrocarbons. However, all crude oils also contain traces of characteristic metallic compounds such as nickel and vanadium [1]. It is nearly impossible to avoid exposure to hydrocarbons from petroleum products, whether it is from gasoline fumes at the pump, spilled crankcase oil on asphalt, solvents used at home or work, or pesticide applications that use petroleum products as carriers [2]. Wastewaters released by oil-processing and petrochemical enterprises contain large amounts of toxic derivatives, such as polycyclic and aromatic hydrocarbons, phenols, sulfides, and heavy metals [3]. Vanadium and nickel metals not only are present in residual waters from the oil industry but also can be present as particulate matter in inhaled air [4] and as environmental heavy metals emitted from automotive materials [5]. Both metals are also present as aerosols or in the ashes of thermal power stations operating on oil fuel [6]. The recovery of heavy metals from industrial residues is an important task for environmental and economic reasons [7]. Since vanadium and nickel are components in the sample, the final toxic response to crude oil might be due to the synergistic or antagonistic action between vanadium and nickel.

Considering the above- stated, the toxic effect of

crude oil, vanadium and nickel in liver of rats after separate and combined exposures was planned to be carried in the present research by using antioxidant indices and marker enzymes of tissue damage (aminotransferases) as biochemical indicators.

Materials and methods

Chemicals: Kits for glutathione (GSH), superoxide dismutase (SOD), catalase (CAT), glutathione-S-transferase (GST), aspartate aminotransferase (AST) and alanine aminotransferase (ALT) were obtained from Cayman chemical, E. Ellsworth Road, Ann Arbor, USA. Ammonium metavanadate (NH4VO3), nickel sulfate (NiSO4.7H2O) and all the other chemicals used were purchased from high commercial company from Almaty, Kazakhstan. Fresh crude oil was obtained from the oil field Biikzhal, western Kazakhstan.

Animals treatment: Adult male albino rat weighing 230–245 g were used for experimental purposes. The animals were housed in plastic cages at room temperature 20 ± 2, in a photoperiod of 12:12 light/dark cycle and were acclimatized for 2 weeks prior to the start of the experiment. Rats were maintained on commercial pellet diet (protein 18%, fat 6%, fiber 6%, carbohydrates 56%, calcium 0.6%, moisture 10% and ash 11%) and water ad libitum. The study was conducted in accordance with the "Guide for the care and use of laboratory animals"

The animals (six per group) were randomly divided into seven groups. The first group received water to drink and served as a control, the second group received only crude oil (0.5 ml/kg bw, i.p 5 times per week), the third group received daily water solution of ammonium metavanadate (AMV; at a concentration of 150 mg V/L), the fourth group received daily water solution of nickel sulfate (NiSO4; at a concentration of 180 mg Ni/L). The fifth group received crude oil and AMV at the same doses as in the group 2, 3 for crude oil and AMV, the six group received crude oil and NiSO4 at the same doses as in the group 2, 4 for crude oil and NiSO4 and the seventh group received crude oil, AMV and NiSO4 at the same doses as in groups 2, 3, 4 for crude oil, AMV and NiSO4. All rats were weighted at the beginning of the exposure and then the body weight was checked weekly and again when killed. The doses for crude oil, vanadium and nickel were chosen on the basis of our previous study, [8]. Rats were anaesthetized with light ether and blood samples were collected by direct puncture the abdominal aorta to be used in estimation of serum activities of ALT and AST then the animals were sacrificed by xsanguinations under light anesthesia. Livers were removed immediately and processed for estimating enzymes activities.

Enzyme and non-enzyme assays. The content of malondialdehyde was determined in the liver spectrophotometrically at wave length 532 nm according to the method of Burlakova et al., [9]. Superoxide dismutase (SOD) was assayed at wave length 450 nm by the method described by Marklund [10]. Catalase (CAT) was determined at wave length 510 nm according to the method described by Aebi [11]. Glutathione (GSH) was determined at wave length 405 nm using the method described by Baker et al., [12]. Glutathione-S-transferase (GST) was determined at wave length 340 nm by the method described by Habig et al., [13].

Statistical analysis. All data expressed as mean ± SE and statistical analysis was made using the Statistical Package for Social Sciences (SPSS 18.0 software and Microsoft Excel 2010). For tests, analysis of differences between groups consisted on a one-way analysis of variance (ANOVA) with repeated measures, followed by post-hoc comparisons (LSD test). All data are expressed throughout as an arithmetic mean ± standard error (SE). Differences were considered statistically significant at $p<0.05$ [14].

Results

In the visual observation, control animals remained healthy and there was no mortality throughout the experimental period. Animals treated with vanadium alone or in combination with crude oil suffered from dehydration, weight loss and even death while animals treated with nickel or crude oil showed no treatment-related clinical observations. The changes in body weight are summarized in Table 1. There was a progressive and significant ($p<0.001$) increase in the body weight of control rats whereas crude oil, vanadium and nickel treated rats showed a significant ($p\leq0.001$) decrease in body weight. The decrease in body weight by crude oil treatment was more pronounced by vanadium co-treatment and was ameliorated by nickel co-treatment. The simultaneous administration of nickel, crude oil and vanadium decreased the deleterious effect of crude oil and vanadium combination.

Changes in serum marker enzymes

Table 2- shows the levels of serum marker enzymes in control and experimental rats. Crude oil, vanadium and nickel treatment caused abnormal liver function in rats as demonstrated by increased activities of serum hepatospecific enzymes ALT and AST ($p\leq0.001$) after the treatment. The induction in ALT and AST was more pronounced by crude oil and vanadium combination and was ameliorated by crude oil and nickel combination.

Table 1 -The changes in body weight gain 4 weeks after crude oil, vanadium, and nickel exposure

Treatment / Weight	Control	C.O 0.5 ml/kg	AMV 150 mg V/L	N.S 180 mg Ni/L	C.O + AMV	C.O + N.S	C.O+AMV+N.S
Initial weight (g)	235±1.77	245.5±2.31	234±2.03	237±1.86	243.5±1.91	236.5±1.77	242±2.13
Final weight (g)	262±1.98†	229±2.77†	161±2.25†	216±1.75†	184.5±2.83†	231.5±2.70	207.17±2.52†

Values are statistically significant (p<0.05) comparison with initial weight mean ± SE for six rats; **C.O** = crude oil; **N.S** = nickel sulfate; **AMV** = ammonium metavanadate; †P≤0.001

Table 2 - The effect of crude oil, vanadium and nickel on serum aminotransferases activities 4 weeks after exposure

Treatment / Enzyme	Control	C.O 0.5 ml/kg	AMV 150 mg V/L	N.S 180 mg Ni/L	C.O + AMV	C.O + N.S	C.O+AMV+N.S
ALT (mMol/h*L)	0.578±0.03	0.890±0.03†	1.167±0.02†	1.923±0.02†	1.334±0.04†	0.677±0.02**	0.961±0.02†
AST (mMol/h*L)	0.584±0.03	1.015±0.03†	0.988±0.03†	0.863±0.02†	1.171±0.04†	0.683±0.02**	0.713±0.012†

Values are statistically significant (p<0.05) mean ± SE for six rats; **C.O** = crude oil; **N.S** = nickel sulfate; **AMV** = ammonium metavanadate; **P ≤0.01; †P≤0.001

Alteration in liver lipid peroxidation (LPO)

As shown in Table 3, a significant increase in liver malondialdehyde (MDA) after crude oil, vanadium and nickel treatmentwas observed (p≤0.001). Crude oil induced lipid peroxidation was marked by vanadium co-administration (p≤0.001) and slightly improved by nickel co-administration (p≤0.001). However, simultaneous administration of crude oil, vanadium and nickelsignificantly (p≤0.01) recovered the MDA content.

Alteration in liver antioxidant enzyme and non-enzyme assays

As demonstrated in Table 3, a significant decrease in rat liver CAT activity occurred subsequent to crude oil (p≤0.001), vanadium (p≤0.001) and nickel (p≤0.05) treatment. When crude oil was administrated in combination with vanadium or nickelor both salts, the CAT activity significantly induced by (p≤0.01, 0.05, and 0.01) respectively. Crude oil or vanadium or nickel treatment resulted in significant decrease in SOD activity (p≤0.001). The combination treatment had more impact than separate treatment. The administration of crude oil or vanadium to rats has been shown to reduce the hepatic glutathione levels. The reduction in the GSH level was higher in vanadium-treated rats (p≤0.001) than in crude oil- treated rats (p≤0.001). There was a trend towards glutathione induction in rats treated with nickel (p≤0.001). Nickel administration in combination with crude oil or vanadium was increased significantly (p≤0.001). Crude oil and vanadium combination potentiated the depletion in glutathione level (p≤0.001), while the administration of nickel in combination with crude oil and vanadium slightly ameliorate the glutathione level (p≤0.001) than that obtained by crude oil and vanadium combination. GST activity showed a significant (p≤0.001) increase in rat liver after crude oil treatment. However all other treated groups showed significant (p≤0.001) decrease in the liver GST activity.

Table 3 - In vivo effect of crude oil, vanadium and nickel on rat antioxidant parameters and lipid peroxidation after 4 weeks of exposure

Parameters / Treatment	MDA content (nmol/g)	SOD activity (U/ml)	CAT activity (U/g)	GST activity (nmol/min/ml)	GSH content (μM)
Control	0.069±0.0012	0.075±0.0034	3.877±0.021	245.87±1.63	19.59±0.345
C.O (0.5 ml/kg)	0.089±0.0029 †	0.058±0.0028†	3.712±0.031†	265.96±2.60†	17.94±0.289†
AMV(0.15 mg V/ml)	0.083±0.0026†	0.052±0.0021†	3.721±0.029†	159.11±1.93†	10.51±0.291†
N.S (0.18 mg Ni/ml)	0.092±0.0038†	0.054±0.0025†	3.775±0.035*	201.53±1.89†	21.35±0.297†
C.O + AMV	0.093±0.0024†	0.049±0.0020†	4.011±0.029**	187.31±2.41†	8.89±0.320†
C.O + N.S	0.089±0.0016†	0.036±0.0022†	3.981±0.034*	225.69±1.95†	21.88±0.335†
C.O+AMV+N.S	0.058±0.0021**	0.044±0.0014†	4.005±0.033**	221.12±1.58†	15.07±0.234†

Values are statistically significant (p<0.05) mean ± SE for six rats; C.O = crude oil; N.S = nickel sulfate; AMV = ammonium metavanadate; *P ≤0.05;**P ≤0.01; †P≤0.001

Discussion

Body weight changes are indicators of adverse effects of drugs and chemicals and it will be significant if the body weight loss occurred is more than 10% from the initial body weight [15]. In this experiment, crude oil, vanadium and nickel groups were weighed less than the control group. A consistent reduction in body weight by vanadium has also been reported by Thompson and McNeill [16]. The decreased body weight in our study is in good agreement with some previously published articles by Kechrid et al., [17] who have also reported decreased body weight in nickel treated rats. This reduction in weights might be due to low food consumption and reduction in protein levels. As the nickel ions have a higher affinity for proteins and amino acids and have shown to produce oxidation of proteins in cells [18]. The decrease in body weight by crude oil is reported also by Adedara et al., [19]

Several of soluble enzymes of blood serum have been considered as indicators of the hepatic dysfunction and damage. The increase in the activities of these enzymes in plasma is indicative for liver damage and thus causes alteration in liver function. In our study, the increased activities of ALT and AST in serum obviously indicate that liver is susceptible to crude oil, vanadium and nickel induced toxicity. This increase could be attributed to the hepatic damage resulting in increased release of functional enzymes from biomembranes or its increased synthesis [20]. This elevation of serum liver enzymes is similar to that reported by Adedara et al., [19], Sidhu et al., [21] Elshaari et al. [22]. The results obtained regarding the activities of SOD, CAT and GST and the concentration of GSH and MDA (an indicator of lipid peroxidation) in the liver clearly indicate that crude oil, vanadium and nickel are able to induce the oxidative stress during repeated separate administration as well as during co-exposure. One of the various markers of oxidative stress is malondialdehyde (MDA) one of the end products of lipid peroxidation. Many authors reported that, vanadium administration enhance the lipid peroxidation in in vivo conditions [23]. Furthermore, the MDA-enhancing effect of crude oil that was supported by Adedara et al., [19] Corroborate with our findings [24] noted high level of lipid peroxidation in the liver with an increased concentration of H2O2 followed by a reduction in the activity of enzyme catalase which is an H2O2 scavenging enzyme by nickel. Herein, the combined crude oil and vanadium treatment had more impact than the separate treatment and crude oil and nickel combination had less impact than the separate treatment whereas the simultaneous administration of crude oil, vanadium and nickel significantly recovered the LPO level. The failure of elevation of LPO by simultaneous administration of crude oil, vanadium and nickelon the other hand, could be attributed to the sufficient capacity of the tissue basal GSH and GSTs in eliminating the toxic intermediates produced by them.

Mammalian cells are equipped with both enzymic and non-enzymic antioxidant defences to minimize the cellular damage caused by interaction between cellular constituents and oxygen free radicals (OFRs) [25]. Antioxidative defense mechanisms dispose of free radicals/ROS by directly scavenging them or by interrupting the already occurring lipid peroxidation chain reaction to limit their tissue damage. Among the well-known biological antioxidants, SOD and its two isozymes, and catalase have a significant role. SOD spontaneously dismutates (O2-).anion to form O2 and H2O2. The decrease in the activity of this enzyme in this study could result in the accumulation of O2.□ within the cell. One important reaction of O2.□ is with H2O2 to form the hydroxyl radical (OH.), which is the most potent oxidant known [26]. Hence the diminishment of SOD by crude oil, vanadium and nickel could be a reflection of their insult which would make target tissues more vulnerable under oxidative stress.

Catalase is a common enzyme found in nearly all living organisms that are exposed to oxygen, where it catalyzes the decomposition of hydrogen peroxide to water and oxygen [27]. Inhibition of the CAT activity by crude oil or vanadium or nickel alone indicates H2O2 in the rats liver had accumulated to cause toxicity to the enzyme, while the induction of CAT activity by combined xenobiotic treatment might reflect an adaptive response of the organism or a loss of compensation mechanism to the pollution stress.

GSH is a well-known free-radical scavenger and a potent inhibitor of LP [28]. Under oxidative stress GSH levels are suppressed due to the loss of compensatory responses and oxidative conversion of GSH to its oxidized form [29]. Because of its importance in the detoxification mechanism, its depressed levels in crude oil or vanadium toxic conditions would, therefore, lead to decreased detoxification capacity of liver. In the other hand nickel treatment increased the GSH content. A higher intracellular GSH concentration caused by nickel may reduce damage and promotes better survival under the conditions of oxidative stress.

Glutathione S-transferases (GST) are antioxidant enzymes that play important roles in the cellular detoxification, excretion of environmental pollutants as well as protection against oxidative stress by their

ability to conjugate GSH with compounds containing an electrophilic center [30]. We have observed increase in GST following crude oil treatment. The induction of GST as a major antioxidant produced by the cell, protect it from free radicals. These highly reactive substances, if left unchecked, will damage or destroy key cell components (e.g. Membranes, DNA). This agreed with the earlier work reported by Xiao et al., [31]. Generally, depletion of GST activity in the liver homogenates of vanadium or nickel treated rats was observed. This depletion may be due to the fact that toxic intermediates produced in the liver during contaminant metabolism may inactivate the enzyme, resulting in reduced GST activity levels in this organ.

Conclusion

Vanadium was toxic and it toxicity was augmented by crude oil co-administration while nickel or crude oil alone does not pose an apparent evident toxic insult to rats. The severe depletion of GSH level and GST activities may result in the enhancement of LP and the disturbance of membrane integrity in response to crude oil or vanadium treatment. The continuation of depressions of GSH and GSTs by combination treatment of these xenobiotics may imply that the onset of recovery of detoxication pathway needs a longer period upon combined treatment in the liver. An amelioration of oxidative damage noted by the combined crude oil and nickel treatment compared to crude oil-induced hepatic injury indicates – an antagonism between these xenobiotic. The recovery of hepatic injury seems to be accompanied by a slight amelioration in LPO lipid.

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Егеуқұйрықтар бауырының антиоксиданттық қорғау жүйесіне шикі мұнай, ванадий және никельдің қысқа мерзімді әсері

4 апта ішінде құрсақішілік концентрациясы 0,5 мл / кг мұнаймен және ауыз суда ерітілген концентрациясы 180 мг Ni/л никельмен, 150 мг в V/л ванадиймен бөлек және бірге уландырылған ақ егеуқұйрықтардың бауырында липидтердің асқын тотығының (ЛАТ) мөлшері және антиоксиданттық жүйедегі өзгерістер анықталды. Бауырдағы ЛАТ мөлшерінің және АЛТ және АСТ ферменттерінің қандағы белсенділігінің өсуі, мұнай, никель және ванадийдің бөлек уландыруы клеткалардың зор зақымдалуына әкелетінін көрсетеді. Ванадий мен мұнайдың бірлескен әсері синергетикалық қасиетке ие болып, токсикалық нәтижені күшейтеді. Никельдің әсерінен GSH синтезінің белсенденуі мұнайдың уыттылығын төмендетеді. Ванадий, никель және мұнайдың бірлескен әсерінің уыттылығы осы заттардың бөлек әсерінің уыттылығынан төмен болып шықты.

Түйін сөздер: шикі мұнай, ванадий, никель, антиоксидантты ферменттер, липидтердің асқын тотығуы, егеуқұйрықтар.

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Кратковременное действие сырой нефти, ванадия и никеля на антиоксидантную защитную систему печени крыс

Перекисное окисление липидов (ПОЛ) и изменения в антиоксидантной системе были определены в печени белых крыс самцов, получавших раздельно и совместно в течение 4-х недель внутрибрюшинно сырую нефть в концентрации 0,5 мл / кг и ванадий и никель в питьевой воде в концентрациях 150 мг в V/л и 180 мг Ni/л . Можно заключить, что раздельное воздействие сырой нефти, никеля и ванадия вызывали сильное повреждение клеток, о чем свидетельствует увеличение уровня ПОЛ в печени и повышение активности внутриклеточных ферментов АЛТ и АСТ в крови. Совместное действие ванадия и сырой нефти проявляет синергизм и усугубляет этот эффект. Стимуляция синтеза GSH, вызванное воздействием никеля, уменьшает индуцированную воздействием сырой нефтью токсичность. Совместное воздействие ванадия, никеля и сырой нефти характеризуется меньшей токсичностью по сравнению с раздельным воздействием металлов и сырой нефти.

Ключевые слова: сырая нефть, ванадий, никель, антиоксидантные ферменты, перекисное окисление липидов, крыса.