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LIVER AND KIDNEY FUNCTIONS IN URANIUM MINING WORKERS

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ABSTRACT

Uranium mining workers are exposed to external gamma radiation, some B-radiation, to air beam of radon and radon daughters. This is in addition to ore dust containing members of uranium decay series. The intake of radionucleoids are mainly represented by inhalation and / or ingestion and then pass into the blood and body fluid, and from there to the systemic organs. The major biological effect is imposed to the kidney, liver, bone marrow and body fluid. The effect depends on both physical and biological factors .

The level of uranium in the blood and urine of twenty U-mining workers have been investgated in addition to five controls. The kidney and liver functions are tested as well. The data revealed that these are the most affected organs .

INTRODUCTION

uranium is radiochemically and chemically toxic and both U^{235} and U^{238} are radiocarcinogens .

Like any other hard rock mining, uranium is exracted by deep

mining or open pit method during which miners are exposed to external γ - radiation, some B-radiation, air beam radon and radon-daughter and decay products Po^{218} and Bi^{214}.

Absorption of uranium salts may occur by inhalation or by ingestion. 95% of retained uranium in the body is deposited in bone. Extraction is mainly by the kidney. As all uranium isotopes in nature are radioactive, the hazards of high intake of uranium are two fold chemical toxicity and radiological damage.

The changes in the kidney occured when the uranium concentration exceeds 1 mg / kg. Absorption, retention and excretion of uranium are dependent upon its chemical form, (Scott, 1973).

There are two hazards connected with exposure to uranium compounds. The renal damage caused by the chemical toxicity of the soluble uranium compounds and the injury caused by the ionizing radiation resulting from the disintegration of the uranium isotopes (Hursh and Spoor, 1973). The corresponding functional changes in the kidney are characterized by protein - urea, impaired diodrast and PAH clearance, and increased clearance of aminoacids and glucose. Inuline and creatinine often remain virtually normal through after severe damage they may decrease. All changes reflect damage to the lower - third of the proximal convoluted segment of the proximal tubules.

The present work deals with the effect of the inhaled uranium

bearing dust on the uranium mining workers and its impact on liver and kidney functions as well as some chemical parameters in urine .

MATERIALS AND METHODS

Serum and urine levels estimated in twenty uranium mining workers by means of laser induced fluorescence technique . The apparatus used is laser uranium analyzer of Scintrex and is characterized by its highest accuracy (Aly and Anwar, 1992). The sensitivity of the method used is in the ppb range. Controls considered of five ordinary workers far from uranium compounds and dust exposure .

Ten ml venous blood were collected with disposable sterile syringes. Five ml blood were treated directly for uranium estimation, the other 5 ml were centrifuged for serum separation and kept at - 20°C in plastic vials until used .

The activities of SGOT and SGPT were determined following the method of Reitman and Frankel, (1957). The levels of serum uric acid (Kajeyama, 1971), urea (Chaney, et al., 1962) and creatinine (Bartels, 1972) were determined .

A representative sample of 24 hours collected urine was analyzed for proteins and glucose using strips of Compur 9 Test produced by Boehringer. The data were statistically examined according to Snedecor and Cochran, (1967) .

RESULTS AND DISCUSSION

The range, mean and standard error of uranium in blood and urine are presented in Table (1) .

Statistical treatment showed a significant " t " test value ($p < 0.01$) for blood uranium and a highly significant "t" test value ($p < 0.001$) for urine uranium. Figs. (1) and (2) showed the distribution of uranium in both blood and urine respectively .

All the twenty uranium mining workers examined were within the normal levels of uranium in both blood (0.1 - 0.8 ug/L) and urine (0.015 - 7.0 ug / L). The normal picture is correlated with what already known from records that those workers are not frequently subjected to long term exposure beside the good and high ventillation rate efficientcy which reduce radon daughter concentrations in

Table (1) : Concentartion of uranium in blood and urine for mining workers and control groups .

	Parameter	Mining Workers group	Control group	" t " value
Blood uranium	$\bar{X} \pm SE$	0.50 ± 0.04	0.22 ± 0.03	3.48*
(ug / L)	Range	0.26 to 0.80	0.20 to 0.32	
Urine uranium	$\bar{X} \pm SE$	2.48 ± 0.23	0.13 ± 0.02	4.83**
(ug / L)	Range	1.19 to 5.24	0.09 to 0.18	

* $P < 0.01$ ** $P < 0.001$
$\bar{X}$ = mean SE = standard error

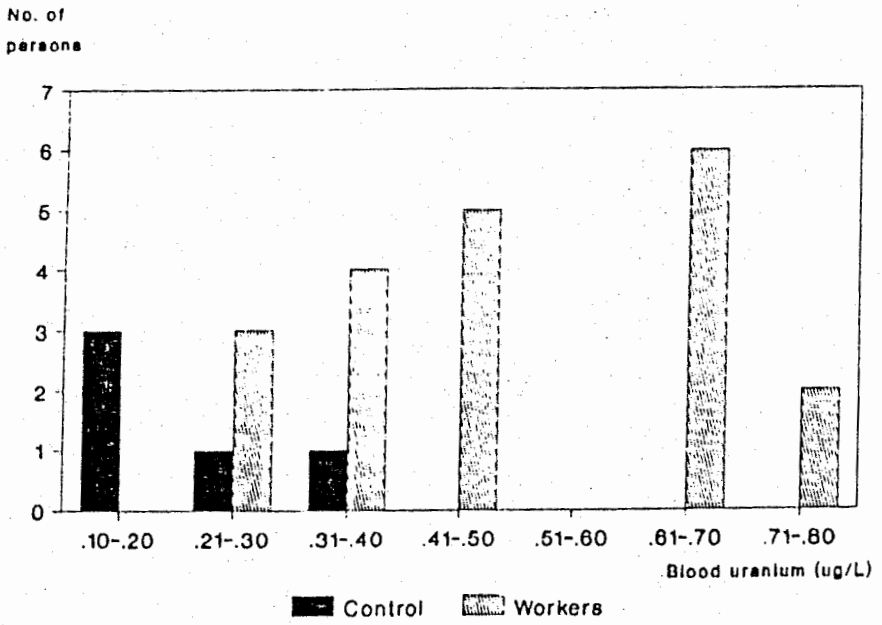

Fig (1): Distribution of Uranium in blood for mining workers and control group .

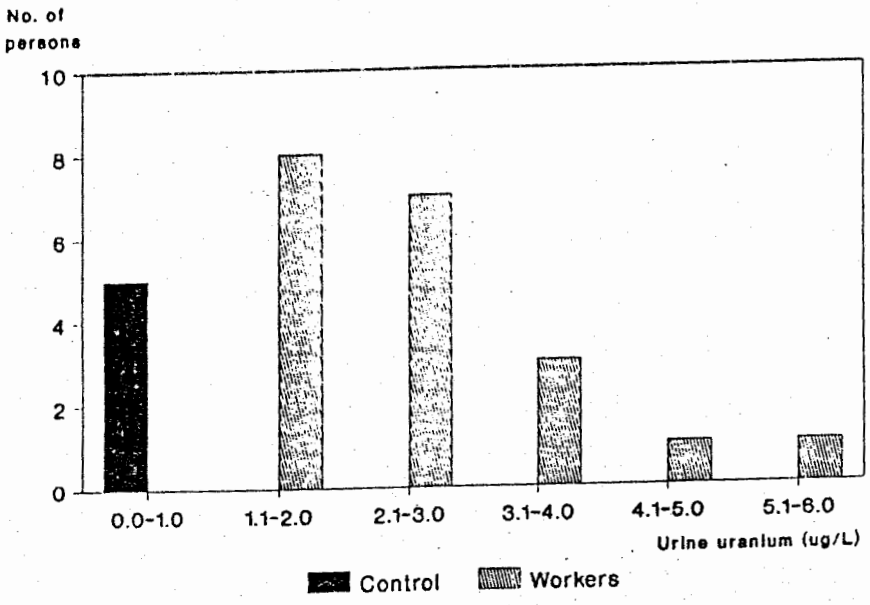

Fig (2): Distribution of Uranium in urine for mining workers and control group.

the exploration mine atmosphere. However, ventillation may increase the dispersion of uranium minerals and silica containing dust (International Commission on Radiological Protection, 1977) . Due to the high density of uranium containing particles, most particles would have an aerodynamic size which will not permit them to be carried to the prephiral part of the lung (Harris, 1961) . Only 1-5 % of uranium containing dust will penetrate to the pulmonary region, the rest will be deposited in the upper respiratory tract and eventually be swallowed (Schultz, 1968) . 95 % of retained uranium in the body is deposited in bone . Excretion of uranium compound is mainly by the kidney .

From the previously mentioned physiological aspects, the normal level of uranium in the blood and urine of those miners may be explained .

It is well known that the critical organ for the chemical uranium toxicity is the proximal tubule of the kidney. The concentration of uranium in kidney is mainly dependent on the solubility of the uranium compounds to which the individual is exposed. The changes of the kidney occurred when the uranium concentration exceeds 1 mg / kg body weight. Because the amount of inhaled uranium is not lethal, regeneration of the renal injured epithelium begins after 2 - 3 days, regrowth is complete within 2 - 3 weeks but this new epithelial lining differs morphologically from normal tubular epithelium and consequently tke kidney function may be greatly affected (Heid, et

al, 1975) .

Table (2) shows the data of kidney and liver functions parameters in serum of the investigated workers, and the results of the statistical treatment are given as well . The " t " test values are significant in case of SGPT, uric acid, urea, and creatinine and non significant for SGOT.

Table (2) SGOT, SGPT, Uric Acid, Urea and Creatinine in serum of the uranium mining workers and the control groups.

	Parameter	Mining Workers group	Control group	" t " value
SGOT (ug / ml)	$\bar{X} \pm SE$	35.85 ± 1.44	23.20 ± 3.01	2.73
	Range	23 to 46	16 to 32	
SGPT (ug / ml)	$\bar{X} \pm SE$	40.40 ± 1.12	23.80 ± 3.02	3.15*
	Range	29 to 50	1 to 31	
Uric Acid (mg%)	$\bar{X} \pm SE$	7.41 ± 0.25	5.76 ± 0.48	2.89*
	Range	5.9 to 10.5	4.1 to 6.9	
Urea (mg%)	$\bar{X} \pm SE$	40.85 ± 2.63	25.20 ± 3.84	2.94*
	Range	17 to 65	15 to 38	
Creatinine (mg%)	$\bar{X} \pm SE$	1.42 ± 0.12	0.84 ± 0.09	3.12*
	Range	0.8 to 3.1	0.6 to 1.1	

* $P < 0.01$

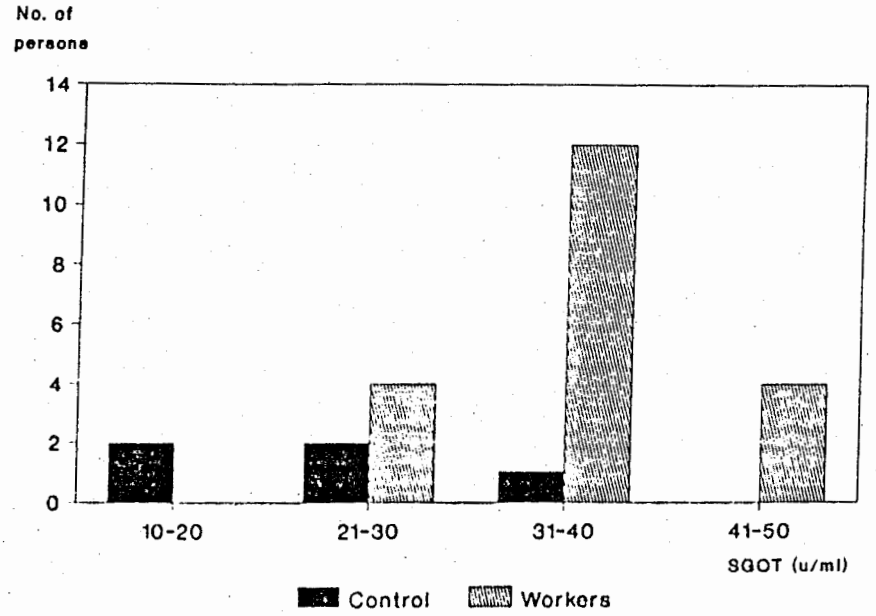

Fig. (3) Distribution of SGOT for mining workers and control group.

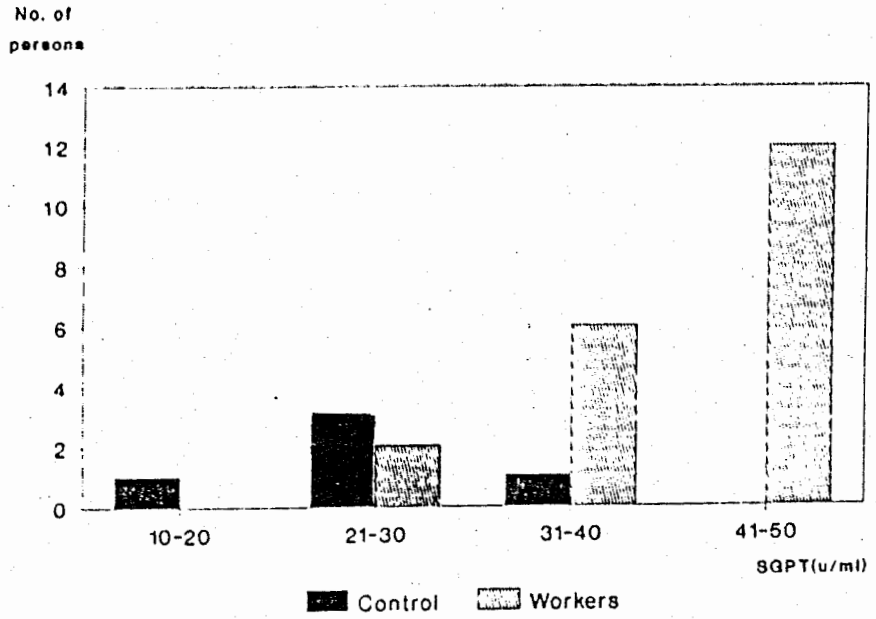

Fig. (4) Distribution of SGPT for mining workers and control group.

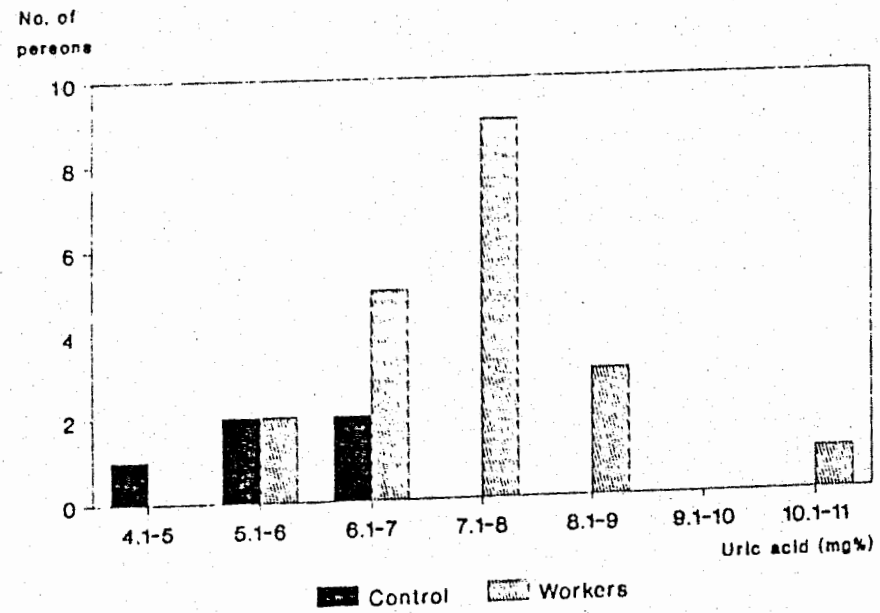

Fig. (5) Distribution of Uric acid for mining workers and control group.

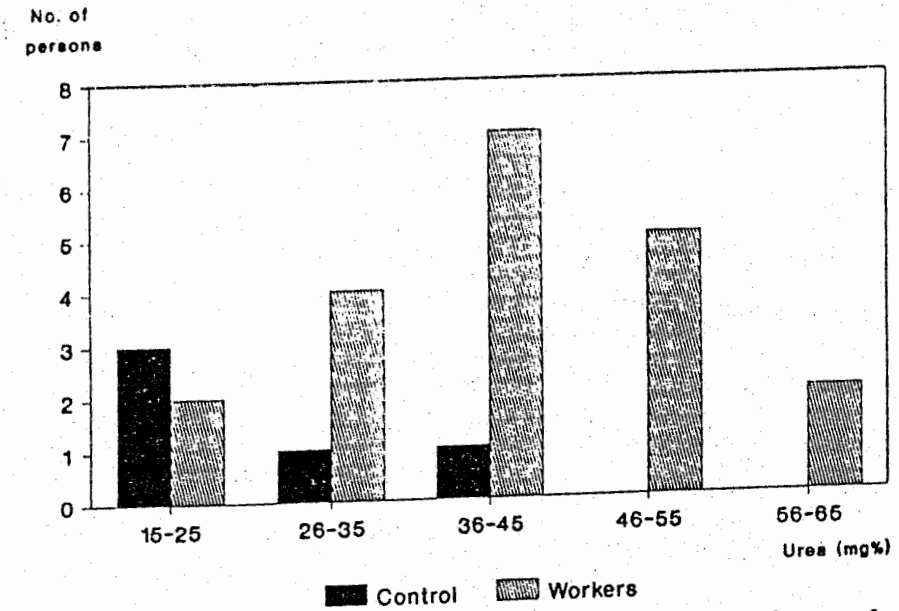

Fig. (6) Distribution of Blood urea for mining workers and control group.

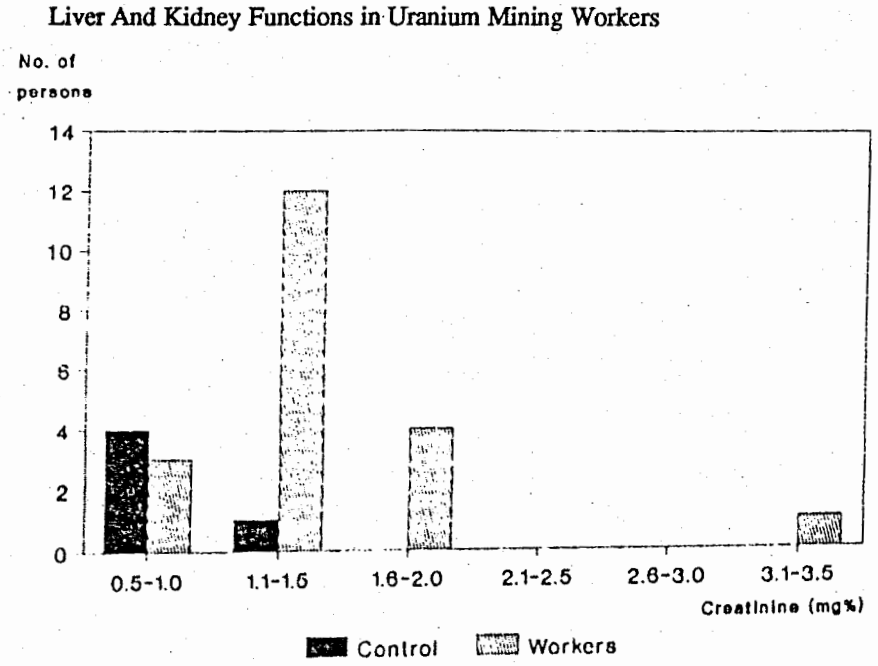

Fig. (7) Distribution of mining workers and control group.

Figs (3-7) show the histograms of kidney and liver functios parameters distribution.

The liver enzymes Table (2) do not show any abnormalities in the investigated workers. This is in full agreement with the fact that uranium is not included among hepto toxine metals which give rise to effects ranging from abnormalities in hepatic enzyme levels to clinical jaundice. Besides the primary target of the uranium compounds is known to be the kidney and death may result from renal failure . Table (3) shows the data of urine proteins and glucose.

Table (3) Urine proteins and glucose in the investigated mining workers and control group .

Mining workers group			Control group		
Serial No.	Proteins	Glucose	Serial No.	Proteins	Glucose
1	-	-	1	-	-
2	-	+	2	-	-
3	-	-	3	-	-
4	+	+	4	-	-
5	-	-	5	-	-
6	-	-			
7	+	++			
8	+	+			
9	-	-			
10	-	-			
11	-	-			
12	-	-			
13	-	+			
14	+	++			
15	++	++			
16	-	-			
17	-	-			
18	-	-			
19	-	-			
20	-	+			

- = negative
+ = positive
++ = positive in excess

Among the twenty uranium miners, only five workers have the signs of kidney disorders showing high values of all kidney function parameters, besides those workers have also a positive abundance of both glucose and proteins. Disorder of the kidney may be attributed to renal injury done by the excretion of uranium compounds.

The obtained results are in full agreement with the findings of Durbin and Wrenn, (1975).

SUMMARY AND CONCLUSION

Analysis of uranium, kidney function parameters and liver enzymes have been carried out in the blood of twenty uranium mining workers and five controls .

Among the obtained data only the kidney function parameters in five miners showed abnormal values indicating kidney disorder. Besides Uranium levels in the blood of the miners didn't exceed the normal levels stated by (Scott, 1973) which reflect no recent exposure and can be used for routine checking of exposure conditions .

According to the already known that the safety precautions against radiation hazards are well stablished in Uranium mines in Egypt. So the assumption of kidney disorder due to renal injury from uranium compounds excretion is cancelled and the disorder may be due to the severe conditions of living in the desert which affect the kidney function . However, seasonal follow up is still needed and recommended ,

REFERECES

Aly, M. M. and Anwar, S. M., (1992) : New method for estimation of microamounts of uranium in blood and urine. Sci. J. Fac. Sci. Menoufia Univ. Vol. VI, P353 - 370 .

Bartels, H. (1972) : Colourimetric method for creatinine determination. Clin. Acta, 37, p. 193-197 .

Chaney, A. L., Marbach, C. P. and Fawcett, J. K. (1962). A colourimetric method for the determination of blood urea concentration. J. Clin. Chem. 8-130 .

Durbin, P. V. and Wrenn, M. F., (1975) in : Conferehce on Occupational Health Experience with Uranium, pp. 67 - 129, U. S. Energy Research and Development Administration, ERDA (Available from National Technical Information Service, U. C. Department of Commerce .

Harris, W. B. (1961) In : Inhaled particles and vapours (C. N. Davies, ed.) . Vol. 1, pp. 209 - 215. Pergamon Press, London.

Heid, K. R., Walsh, W. P. and Houston, I. R. (1975). In : Conference on Occupational Health Experience with uranium, pp. 297 - 322, U. S. Energy Research and Development Administration, ERDA (Available from National Technical Information Service, U. S. Department of Commerce) .

Hursh, J. B. and Spoor, N. L. (1973). In : Handbook of experimental Pharmacology, Vol. 36. Uranium, Plutonium, Transplutonic Elements (H. C. Hodge, J. N. Standard and J. B. Hursh, eds), pp. 197 - 240, Springer - Verlag, New York .

International Commission on Radiological Protecion (1977). Radiation Protecion in Uranium and other Mines. ICRP publica-

tion, 24; Oxford, New York, Frankfurt, Pergamon Press .

Kajeyama, K., (1971). Enzymatic colourimetric method for the determination of uric acid. J. Clin. Chem. Acta, 31, p. 421 .

Schultz, NB. (1968). Proceeding of the First International Congress of Radiation Protection , 2 nd Ed., pp. 1205 - 1217. Pergamon press, London .

Scott, L. M. (1973) in : Hand book of Experimental pharmacology, vol. 36. Uranium, Plutonium and Transplutonic Elements (H. C. Hodge, J. N. Standard and J. B. Hursh, Eds). Springer Velag, New York .

Snedecor, G. W. and Cochran, V. G. (1967). Statistical Methods. 6 th Ed. Iowa State University Press. Iowa, U. S. A.

Reitman, S. and Frankel, S. (1957) A Colourimetric method for the determination of oxaloacetic and glutamic pyruvic trans - aminases. Am. J. Clin. Path. 28, 56 .

دراسة كفاءة الكبد والكلى لعمال مناجم اليورانيوم

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هيئة الطاقة الذرية هيئة المواد النووية

يتعرض العاملون بمناجم اليورانيوم إلى أخطار أشعة جاما وأشعة بيتا وإلى غاز الرادون ووليدات سلسلة تحلله الإشعاعية . هذا بالإضافة إلى دخول حبيبات تراب الخام الذى يحتوى على اليورانيوم ووليدات سلسلة تحلله الإشعاعية أيضاً عن طريق الإستنشاق أو عن طريق الجهاز الهضمى ثم تسلك طريقها إلى الدم ثم إلى أعضاء الجسم التى تتأثر به . ومن أكثر أجزاء الجسم تأثراً بهذه الدورة الكبد والكلى والنخاع الشوكى وسوائل الجسم المختلفة .

وفى هذا البحث تم تحديد نسبة اليورانيوم فى دم عشرون عامل من عمال مناجم اليورانيوم . وكذا قياس كفاءة الكبد والكلى لهم وتم مقارنتها بنتائج تحليل دم مجموعة لاتعيش هذه الظروف للمقارنة . وقد دلت النتائج على وجود بعض الخلل فى وظائف الكلى لعدد خمسة عمال وقد تم قياس مستوى نسبة اليورانيوم فى دم هذه المجموعة وجد أنها كلها لم تتعدى الجرعات المسموح بتواجدها فى دم الإنسان مما يؤكد عدم تعرض هؤلاء الأشخاص لأى إشعاعات حديثة .

وبمراجعة ظروف تشغيل هؤلاء العمال واحتياطات الوقاية الموجودة والمصممة بعناية بمناجم اليورانيوم فقد تم استبعاد امكانية تأثر الكلى نتيجة لخروج المواد المشعة عن طريق البول وقد يرجع هذا الخلل فى وظائف الكلى إلى ظروف معيشة هؤلاء العمال بالصحراء .

هذا ونوصى بتتبع حالة العمال الصحية دائما بالإضافة إلى قياس مستويات نسبة اليورانيوم فى الدم والبول الذى يعد مقياس دقيق لمدى تعرض العامل للأخطار سالفة الذكر ولحجب الخطر عنهم وسرعة علاجهم .