1. INTRODUCTION

The paint used for coating automobile bodies contains toxic substances. Many solvents used are toxic; it is the same for certain other components of the paint: binders, pigments, additives … (1) (2) (3)

These toxic substances can enter the body through various routes: respiratory, digestive or skin. There is therefore a risk of poisoning by these substances, a risk which must be avoided by the use of suitable protective equipment. (4)

On the other hand, the level of plasma creatinine is an important indicator of kidney function. The noticeable increase in serum creatinine indicates renal dysfunction (5).

By measuring the plasma creatinine, we want to check if there are any “automobile” painters who could be victims of kidney poisoning, which could be a sufficient argument for first of all, educate the painters themselves, but also those in government about the risks involved in the profession of “automobile” painter.

Unfortunately, in our town of Lubumbashi, and perhaps in our country, “automobile” painters work without any protection, they have no masks, no gloves and even less appropriate clothing. There is therefore a real risk that "automobile" painters become intoxicated by various substances contained in the paint.

In the present work, we attempt to assess this risk by assaying plasma creatinine in a few individuals in this occupational category.

2. MATERIAL AND METHODS

2. 1. Study environment and Sampling

Our sample was made up of 35 “automobile” painters met in different paint shops in the city of Lubumbashi. They were all male and between 18 and 65 years old.

In addition to the painters, we formed a control sample, made up of individuals claiming to be in good health and not having (permanent) professional contact with the paint in the fluid state. The witnesses were also male and chosen from the same age group as the painters, between 18 and 65 years old.

2.2. Investigation methods for renal functional exploration

The investigations were carried out in the form of a comparative study, alongside the painters, we also assayed creatinine in a sample made up of control individuals, who do not work in painting, who do not have permanent contact with fluid paint and claim to be in good health.

Renal functional exploration essentially meets two objectives:

- Evaluate the overall function of the kidney and specify the importance of a possible nephrotic reduction.
- Study in isolation the major renal tubular functions: the function of concentration and dilution of urine, the acidification function, the reabsorption function (diffusion of different constituents contained in the urinary filtrate: glucose, sodium, etc.) and the secretion function.

Most of these are urinary examinations: diuresis, elimination of H ions by measuring pH, reabsorption of glucose by glycosuria (5).

1) Concentration of plasma creatinine as an index of filtration rate glomerular (7).

To overcome errors related to the 24-hour urine collection, it was proposed to establish two alternatives:

 The first is to measure renal creatinine clearance from plasma creatinine using the Cockcroft - Gault formula:

$$\textbf{Creatinine clearance} = \frac{[140 - \text{age (year)}] \times \text{weight (kg)}}{7.2 \times \text{creatinine plasm.(gm/l}}$$

The Cockcroft -Gault formula was obtained from a population free from kidney and liver disease. If the creatinine is expressed in µmol / L, the factor 7.2 is replaced by 0.814.
However, this formula has some drawbacks:
- Established in healthy men, it is not evaluated in women or in the subject by state of renal failure. If it is used in women, multiply the result obtained by 0.854 by assuming that for equal weight, creatinine production is 15 % indoor.
- In addition, this formula is inapplicable in patients deviating from the usual standards, for example obese or edematous patients, as well as those in whom the mass muscle is very weak.
- The second alternative is quite simply to be satisfied with the serum creatinine, to interpret it and to draw the consequences from it in relation to the reference values.
- This is why, in view of all this drawback, the determination of plasma creatinine is currently the test widely used to assess renal function since its value reflects the glomerular filtration rate.
However, a substantial fraction of the creatinine excreted in the urine arises from a secretion process in the proximal tubule such that in healthy subjects, renal creatinine clearance overestimates glomerular filtration rate by 10 to 30 %. .

This situation worsens considerably when renal function deteriorates, and in severe renal impairment, creatinine clearance can overestimate the filtration rate by 180-200 %. The situation is greatly complicated by the fact that the degree of tubular secretion of creatinine is variable and unpredictable, particularly in severe renal failure.

Conversely, the filtered creatinine can be passively reabsorbed by the tubule, especially when the urine output is very low (less than or equal to 0.5 ml per minute).
The amplitude of this phenomenon remains low, however, of the order of 5 to 10 % at most of the filtered creatinine, and can be avoided by ensuring sufficient diuresis.

Creatinine is a very sensitive marker for well-established renal failure; it is not for early renal failure, since its value only regularly becomes abnormal for glomerular filtration close to 60 ml / minute (i.e. higher creatinine levels at 2 mg / ml corresponding to a nephrotic reduction of approximately 50 % (9). Thus, when the creatinine is, even if only slightly high, it means that the kidney is very sick because, as said previously, the nephrotic reduction will already be

greater than 50 %. Hence creatinine is a more specific measure of renal exploration; compared to uremia which itself reflects variations both in the liver and in the kidney (10).

2.2.1. Plasma Creatinine Assay Method (9)

A. Principle of the Method

Creatinine reacts with picric acid in an alkaline medium, forming a yellow-orange complex, the coloring intensity of which, measured with a photometer, is directly proportional to the concentration of creatinine present in the biological specimen. This is the reaction described by Jaffé, and the method is called Jaffé. We used the kinetic variant of the method.

B. Procedure

At the wavelength of 492 nanometer, at a temperature of 37 ° C, optical path cuvette 1 cm, zero the instrument with distilled water, then pipette the solutions into a cuvette, as seen in the next board :

Table 1: Procedure for the determination of plasma creatinine

	White	Standard	Sample
Standard	-	100 µl	-
Sample	-	-	100 µl
Working solution	1 ml	1 ml	1 ml

Mix and start the stopwatch. Place the mixture in the photometer, read absorbance 1 (abs 1), at the 30th second and absorbance 2 (abs 2), at the 90th second counting from the addition of the sample.

C. Calculation of Creatinine Concentrations

The variations in optical densities must first be calculated, both for the standard and for the samples.

ΔAbs sample = Abs2 sample - Abs1sample

ΔAbsstand = Abs2 stand - Abs1 stand

The concentration of creatinine is obtained by the relationship:

$$\mathbf{C = Abs_{Sample} / Abs_{standard} \times Standard\ concentration}$$

2.2.2 Statistical methods used for the analysis of the results (11)

We used Student's t test. Student's "t" test is a hypothesis test of the difference between means. The null hypothesis is not rejected (therefore accepted) when there is no significant difference between the means and it is rejected when there is a significant difference.

The protocol for calculating the "t" is as follows:

$\overline{X} = \sum \frac{Xi}{n}$ $\overline{X}$: Arithmetic mean

$\sigma = \sqrt{\frac{\sum (Xi - \overline{X})}{n-1}}$ σ : Difference - kind

t= Student's reduced variable

$t = \frac{\left|\overline{X}_1 - \overline{X}_2\right|}{\sigma\sqrt{\frac{1}{n_1} + \frac{1}{n_2}}}$ With $\sigma = \sqrt{\frac{(n_1 - 1)\sigma_1^2 + (n_2 - 1)\sigma_2^2}{n_1 + n_2 - 2}}$

σ = standard deviation of common estimate.

$\overline{X}_1$ = mean of the sample of painters

$\overline{X}_{2=}$ mean of the sample of witnesses

n_1 = Workforce of painters

n_2 = Workforce of witnesses

3. RESULTS AND DISCUSSION

3.1. Presentation of the results

We assayed plasma creatinine in 35 “automotive” painters, all male, and in 36 control individuals, also male. The blood specimens analyzed were sera, and the samples were taken on an empty stomach. The assay results observed are shown in Tables II and III.

Table 2. Plasma creatinine assay results in painters "Automobile"

N°	**N ° Seniority in the profession**	**Creatinine level (mg / dl)**
1	10 years	1.1
2	3 years	1.3
3	25 years	1
4	50 years	1.1

5	30 years	0.9
6	11 years	0.9
7	20 years	1
8	15 years	1.2
9	25 years	1.1
10	27 years	1.1
11	8 years	1.2
12	11 years	1
13	1 year	1.1
14	18 years	1.1
15	10 years	1.7
16	17 years	1.1
17	19 years	1.1
18	6 years	1.2
19	2 years	1.2
20	18 years	1.4
21	2 years	1
22	2 years	1
23	10 years	1.4
24	3 years	1.2
25	4 years	1
26	18 years	1.2
27	24 years	1.1
28	5 years	1.4
29	20 years	1.1
30	27 years	1.2

31	25 years	1.2
32	20 years	1.5
33	2 years	1.1
34	27 year	1.9
35	3 year	1.4
	Mean	**1.2**
	Standard deviation	**0.21**

Table 3. Results of plasma creatinine assay in controls

N°	**Creatinine level (mg / dl)**
1	0.97
2	0.97
3	1.1
4	1.1
5	1
6	1.4
7	1
8	0.9
9	1.3
10	1.2
11	1.5
12	1.6
13	1.1

14	1.2
15	1.1
16	1.1
17	0.93
18	1.1
19	1
20	1.1
21	1.2
22	1
23	0.97
24	1.2
25	0.8
26	0.97
27	0.8
28	1.2
29	1.3
30	0.8
31	0.93
32	0.93
33	2.1
34	0.93
35	0.9
36	1.2

	Mean 1.1
	Standard deviation 0.063

3.2. Results analysis

3.2.1. Comparison of Means of Plasma Creatinine Levels

We compared the means of creatinine levels between painters and controls by the "t" test to see if their difference is significant or not.

Null hypothesis: H0 = X1 = X2: the painters have the same concentrations as the controls.

So the difference is not significant.

Alternative hypothesis: H1 = X1 ≠ X2 or X1> X2: painters have plasma levels of creatinine higher than those of the controls so the difference is significant.

$$t_{calculé} = \frac{\left|\overline{X}_1 - \overline{X}_2\right|}{\sigma\sqrt{\frac{1}{n_1} + \frac{1}{n_2}}} = 1{,}83$$

The t distribution table gives the threshold of 5 %, with 69 degrees of freedom t = 1.994

We deduce the acceptance interval of the null hypothesis H_0 at 95% given by t tabulary (t theoretic) belongs to the interval [-1.994; + 1.994].

Now we see that t calculated = 1.83 belongs to this interval.

Hence the null hypothesis is not rejected (accepted) and the H_1 hypothesis is rejected. There is therefore no statistically significant difference between the averages of the creatinine levels of the painters and the controls. We can therefore deduce that the painters and the witnesses have the same plasma concentrations of creatinine

Table IV: Student's "t" statistics

	Workforce	**Mean**	**ddl**	**$t_{calcule}$**	**$t_{theoretical\ 5\ \%}$**	**Interpretation**
Painters	35	1.2	69	1.83	2.00	N.S*
Witnesses	36	1.1				

ddl : degree of liberty

N.S* : non-significant difference

3.2.2. Comparison of the proportions of abnormal creatinine levels between painters and witnesses.

This analysis is no longer necessary, because there is no abnormal plasma creatinine value, neither among the painters, nor among the witnesses.

4. DISCUSSION OF RESULTS

A. The Choice of the Method of Investigation.

For at least two reasons, we have chosen to refer the analytical methods on painters to the results obtained on a group of witnesses made up of individuals claiming to be in good health, and not having direct contact with automobile paint, rather than referring to the reference values as described in the literature (12):

• The reference values depend on a large number of factors: race, sex, geographical situations, eating habits, socio-economic situation, and there is nothing that guarantees us that the reference values indicated in the literature conforms to our biological status.

• In this particular case of the creatinine dosage, the delicacy of the analysis (strict observation of temperatures and incubation times) means that very often the working conditions differs somewhat from those in which the reference values were established. . Therefore, the reference to these values can introduce a bias in the analysis and in the interpretation of the results.

For these two reasons, we preferred to use witnesses because living in the same conditions as painters, and their blood specimens rigorously analyzed under the same conditions as those of painters can provide comparable data.

B. Results

Using Student's test, we compared the means of the creatinine levels between the painters and the controls. We found that at the significance level 0.05 and with 69 degrees of freedom, the t tabulary was 1.994, while the t calculated was 1.83.

This allows us to say that there is a 95 % probability that the plasma creatinine levels in painters are not different from those seen in control individuals. We observe that both painters and controls have plasma creatinine levels within the reference range as given in the literature (12).

These results tend to show us that "automobile" paint, used as it is in our environment, is safe. It is dangerous to think so. It should be remembered that increases in plasma creatinine only become appreciable in established renal insufficiency, whereas they may be inconsistent in early renal insufficiency. This explains the fact that creatinine is dosed at the same time as

plasma urea, because the latter increases early in renal disease, although not always specific to renal disorders(13)(14).

CONCLUSION

In this study, we evaluated the risk run by "automobile" painters by measuring plasma creatinine in a few individuals belonging to this professional category.

Indeed, the organic solvents contained in the paint, heavy metals, constituents of metallic pigments and other additives present a real risk of intoxication of the kidney tissue.

Unfortunately, in our city, "automobile" painters work without adequate protection. This is what aroused our fear and prompted us to carry out the present investigations.

In view of the results of the comparison by the Student test, we would be tempted to conclude that the renal function of car painters was not subject to any poisoning related to paint, because there is no significant difference between the plasma level observed in painters and those found in witnesses.

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