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Short communication

Polymorphism of delta-aminolevulinic acid dehydratase in lead-exposed workers

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Delta-aminolevulinic acid dehydratase (ALA-D) is an enzyme involved in the haem biosynthetic pathway. The inhibition of its activity by lead is well known. A recent study of Battistuzzi et al. (1981) documented a genetic polymorphism for ALA-D. This prompted us to conduct a survey to study lead exposed workers with regard to the response of lead relevant parameters in relation to their ALA-D phenotypes.

The study group comprised 202 male lead workers exposed to different amounts of lead and 36 male subjects of the same sociological and geographical region for reference data. Results are given in Tables 1 and 2.

As summarised in Table 1 the lead-exposed workers showed a wide range of blood-lead levels, a 50% decrease of ALA-D activity in comparison to controls and a subsequent increase of aminolevulinic acid values. Table 2 gives the distribution of ALA-D phenotypes in lead-exposed workers, evaluated by performing starch gel electrophoresis according to a modified method from Battistuzzi et al. (1981). The distrubution of the phenotypes correlated well with those estimated from Benkmann et al. (1983) for the German population. This is of some importance because 56% of the lead exposed workers were not Germans, most of them being Turks.

Forming groups of the lead relevant data according to the ALA-D phenotypes revealed increasing values for blood-lead concentrations, a corresponding decrease of ALA-D activity and increases in aminolevulinic acid values on the order of 1-1, 2-1 and 2-2 (Table 2).

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Table 1. Blood-lead concentrations (PbB), blood ALA-D activity, and urinary aminolevulinic acid values (ALA-U) in lead-exposed workers and in a reference cohort

	Lead-exposed workers $n = 202$ ($\bar{x} \pm SD$)	Reference group $n = 34$ ($\bar{x} \pm SD$)
PbB (µg/100 ml)	40 ± 17	14 ± 5
ALA-D (U/l)	18 ± 9	37 ± 13
ALA-U (mg/l)	8 ± 11	5 ± 2

Table 2. Distribution of ALA-D phenotypes in lead-exposed workers and corresponding lead relevant data

	ALA-D phenotypes		
	1-1 $n = 160$ ($\bar{x} \pm SD$)	2-1 $n = 32$ ($\bar{x} \pm SD$)	2-2 $n = 10$ ($\bar{x} \pm SD$)
PbB (µg/100 ml)	38 ± 17	44 ± 17	56 ± 18
ALA-D (U/l)	19 ± 9	16 ± 9	12 ± 5
ALA-U (mg/l)	7 ± 10	13 ± 16	11 ± 6

Table 3. Average values for ALA-D activity and ALA for ALA-D phenotype 2-2 and matched 1-1 phenotypes

	ALA-D phenotypes				Wilcoxon-test, *t*-test
	n	2-2 ($\bar{x} \pm SD$)	n	1-1 ($\bar{x} \pm SD$)	
PbB (µg/100 ml)	10	56.35 ± 18.18	20	55.53 ± 17.99	
ALA-D (U/l)	9	12.20 ± 5.11	18	11.76 ± 7.10	ns
ALA-U (mg/l)	10	10.60 ± 6.01	20	15.98 ± 16.05	ns

ns: not significant

At first glance an effect of the ALA-D phenotype on blood-lead concentration and ALA-D activity seemed to exist. The differences in the results for 1-1 and 2-1 phenotypes were not so striking. Thus we focused our attention on the values of the 1-1 and 2-2 phenotypes.

Because it is unlikely that the ALA-D phenotype affects the blood-lead level, we selected matched pairs out of the group of the exposed workers of the 1-1 phenotype by taking the blood-lead level as a basis. Four each blood lead concentration of the 2-2 phenotype, we took two matched 1-1 phenotypes. The corresponding means of ALA-D and ALA are given in Table 3. As shown the differences between the 1-1 and 2-2 phenotypes with respect to the ALA-D ac-

tivity and the ALA never reached significant values (Student's *t*-test, Wilcoxon-test for matched pairs).

From our results we can therefore conclude that the response of ALA-D activity to a certain blood-lead level is independent of the ALA-D phenotype.

References

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