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PLASMA CHROMIUM LEVELS IN SMALL-FOR-GESTATIONAL-AGE NEWBORN INFANTS*

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SUMMARY: Yurdakök M, Özer D, Erdem G, Temizer A. (Department of Pediatrics, Hacettepe University Faculty of Medicine, and the Department of Analytical Chemistry, Hacettepe University Faculty of Pharmacy, Ankara, Turkey). Plasma chromium levels in small-for-gestational-age newborn infants. Turk J Pediatr 35: 37-40, 1993.

Plasma chromium (Cr) levels were determined in 24 preterms and 18 full-term newborn infants. There was no statistically significant differences in plasma Cr levels between the preterm and full-term infants. Plasma Cr levels were similar in small-for-gestational-age infants and in infants with hypoglycemia compared with healthy infants. *Key words: chromium, hypoglycemia, newborn, plasma, small-for-gestational-age.*

Hypoglycemia in small-for-gestational-age (SGA) infants is a multifactorial phenomenon. The factors which are implicated in this condition include depletion of liver glycogen, decreased fat and protein reserves, functional hyperinsulinism, decreased fat oxidation, a low gluconeogenic rate and increased glucose demand as a result of asphyxia at birth, and a relatively large brain mass. In most SGA infants there is little evidence of a primary endocrine abnormality responsible for the hypoglycemia. However, further research, particularly related to hormone receptor activities, is needed to clarify this point[1].

Chromium (Cr), a trace element essential for humans and animals, is involved in normal carbohydrate metabolism which potentiates the action of insulin[2-4]. Improvement of glucose intolerance has been demonstrated in malnourished children, especially in those with Cr deficiency, by the supplementation of inorganic Cr[5-7]. Since information pertaining to Cr deficiency is very limited, we decided to measure the plasma levels in SGA newborn infants with the aim of

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contributing to an explanation regarding the pathogenesis of hypoglycemia in these infants.

Material and Methods

Twenty-four preterm and 18 full-term infants who were selected at random were enrolled in this study. Eight of the preterm infants and eight of the full-term infants were SGA, while the rest were appropriate-for-gestational age (AGA) infants (Table I). None of the infants had severe congenital malformations. Infants with infectious diseases[3] and inborn errors of metabolism were excluded from the study on the basis of their clinical and laboratory findings including blood cultures and plasma and urine amino acid analyses.

Table I: Some Features of the Newborn Infants

		SGA Infants	AGA Infants
Preterm	M/F	5/3	13/3
	GA (w)	35.4 ± 1.7	35.3 ± 0.9
	BW (g)	1641 ± 383	2199 ± 183
Full-term	M/F	5/3	6/4
	GA (w)	39.2 ± 1.1	39.7 ± 1.1
	BW (g)	2443 ± 220	34.15 ± 440

M/F: male/female, GA: gestational age, w: week, BW: birth weight.

Hypoglycemia was defined as a blood sugar level under 20 mg/dl in the preterm infants and 30 mg/dl in the full-term infants on the first day of life. To determine the serum Cr levels, blood was drawn from the infants on the first day of life prior to the administration of intravenous fluids. These samples were collected in plastic syringes having siliconized needles attached to heparinized polyvinylchloride tubing. After the blood was centrifuged for 15 min at X 2000 g, the plasma was carefully removed with a sterile polystyrene pipette, and 1 ml aliquots were stored in sterile polypropylene tubes at −20 °C. The plasma Cr level was assayed by atomic absorption spectrophotometry (Perkin Elmer 3030) as previously described[8]. All results were expressed as the mean ± SD and statistical analysis was performed by using the Student's *t* test.

Results

We found no difference in plasma Cr levels between the preterm and full-term infants and in SGA and AGA infants (Table II). Plasma Cr levels were also similar in the newborn infants with hypoglycemia and in those with normal blood glucose levels (Table III).

Table II: Plasma Cr Concentrations (μg/l) in the Newborn Infants

	SGA Infants	AGA Infants	Total
Preterm	34.61 ± 27.02 (n : 8)	36.62 ± 23.43 (n : 16)	35.61 ± 25.68 (n : 24)
Full-term	43.14 ± 20.28 (n : 8)	26.99 ± 24.63 (n : 10)	35.33 ± 22.46 (n : 18)
Total	31.80 ± 24.02 (n : 16)	38.87 ± 24.09 (n : 26)	

$p > 0.05$ in all comparisons.

Table III: Plasma Cr Concentrations (μg/l) in Hypoglycemic and Normoglycemic Newborn Infants

	Infants With Hypoglycemia	Infants With Normoglycemia	Total
Preterm	41.22 ± 24.26 (n : 8)	30.25 ± 23.40 (n : 9)	35.76 ± 23.53 (n : 17)
Full-term	56.92 ± 12.92 (n : 5)	29.61 ± 22.84 (n : 15)	43.26 ± 18.09 (n : 20)
Total	45.50 ± 22.34 (n : 13)	29.84 ± 22.54 (n : 24)	

$p > 0.05$ in all comparisons.

Discussion

Although it is known that Cr potentiates the action of insulin, the mechanism of this effect has not been clearly understood. Cr is essential for the peripheral action of insulin at the cellular level. The increase in insulin efficiency with supplementary Cr may be related to improvements in receptor number or affinity or both. This may cause the rapid disappearance of Cr from the circulation following an intravenous glucose load in healthy individuals. It has been reported that Cr supplementation improves impaired glucose tolerance in man[3].

Cr deficiency does not appear to be related to low fasting blood glucose levels. Impaired glucose utilization in patients with protein and calorie malnutrition is not solely due to a deficiency of a single trace element, i.e. Cr. But a positive effect

may be seen on glucose tolerance after the administration of Cr when the plasma Cr level is lower than normal[5,6]. In this study we could not find a difference in plasma Cr levels between the SGA newborn infants or the hypoglycemic infants when compared with the controls.

Pregnancy is characterized not only by significantly lowered serum Cr levels, but also by a decrease in the Cr content of hair on the scalp[9]. The role of Cr in fetal metabolism has not been clearly defined. Our results show that plasma Cr concentrations are not affected by the duration of gestation or intrauterine nutrition.

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