The Hamilton Health Sciences Corp. (Henderson and McMaster Campuses) for Cl values determined on the Vitros, CX3 Delta, and Hitachi 717 instruments.

References


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To the editor:

During internal evaluation of the COBAS Integra Cl electrode, we observed an interference of acetylsalicylic acid, 2.77 mmol/L, on Cl determinations by +8% in the direct mode and +1.5% in the indirect mode. When assessing drug interferences, it is our procedure to mention in our Method Manual only interferences exceeding 10%—as stated in the Introduction of the COBAS Integra Method Manual, pp. 10–11, along with a list of all drugs tested and their concentrations.

Since receiving the data from Mori and Waldhuber’s experiments, we have repeated the therapeutic drug interference checks and initially confirmed our previous findings. Further investigations, however, particularly with electrodes returned from customers and with electrodes that had been in heavy use for some time, have shown that not all electrodes behave the same with respect to salicylate interference. Nonetheless, we could confirm that, in initial testing, no electrodes that had been installed and in use on-board an analyzer for ≤1 month showed >10% interference from salicylate at the tested concentration of 2.77 mmol/L.

On the basis of these findings, we have informed our customers that, as an interim measure, the Cl electrode on the COBAS Integra must be replaced after every 4 weeks of use.

Our investigation has so far determined that the surface area of the membrane of some electrodes is larger than in others. We are working with the manufacturer of our Cl ISE to correct this problem and to ensure the long-term reliability of the electrode.

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higher than their extraction assays for some female sera [1]. Similarly, the water-soluble interference with a direct assay also was reported by Fitzgerald and Herold, who found that female serum testosterone results determined by a direct assay (ACS-180) compared poorly with their GC/MS measurements performed after solvent extraction [2].

We conclude that results by non-extraction assays may need confirmation by an extraction assay to avoid unnecessary workups for patients. We encourage laboratorians to avoid unnecessary workups for patients. We encourage laboratorians to avoid unnecessary workups for patients. We encourage laboratorians to avoid unnecessary workups for patients.

References

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Decline in Blood Lead in Ontario Children Correlated to Decreasing Consumption of Lead Gasoline, 1983–1992

To the Editor:
Since 1980, the Ontario government has conducted several blood lead screening surveys in children in several cities and regions of Ontario, Canada [1,2]. The blood lead concentrations in Ontario children has declined in both rural and urban areas over the past decade, this decline coinciding with the complete phasing out of lead in gasoline in 1990. Here, we evaluate this decline in terms of the geometric mean of the blood lead concentrations of 6014 children.

The collection procedure of capillary finger-prick blood samples and the method for blood lead analysis by Zeeman graphite furnace atomic absorption spectrophotometry were described previously [3] and were used identically in all our blood lead analyses. The blood lead screening and surveys evaluated were done in the Toronto Western Health Unit in 1984, 1985, and 1988; the Peel Region, 1987; the Niagara Region, 1987; Southern Ontario, covering more-urban areas (Toronto, Windsor, etc.), 1984 [1]; and Northern Ontario, covering the less-settled areas (e.g., Thunder Bay and Moosonee), 1987 and 1992 [2, 4].

Gasoline sales data in Ontario were taken from Statistics Canada (Ottawa, ON), and the annual estimates of the lead content (g/L) of leaded gasoline consumed in Ontario were provided by Ethyl Canada (Mississauga, ON). Thus the total lead in leaded gasoline consumption in Ontario was calculated as the product of grams of lead per liter of gasoline times the liters of leaded gasoline sold. In 1982, 2.6 \times 10^9 g of lead was consumed in Ontario; by 1990, this had declined to 1.9 \times 10^7 g.

The geometric mean of the blood lead concentrations and the total amount of lead in leaded gasoline consumed in each year from 1983 to 1992 are shown in Fig. 1. The linear regression analysis of the 22 blood lead summaries determined during that time shows a decline of 0.05 \( \mu \)mol/L per year \( (r = 0.8505, n = 22; 1 \text{ mol} = 207.2 \text{ g}). \) In a high-risk area of the Toronto Eastern Health Unit, the rate of decrease was 0.07 \( \mu \)mol/L per year \( (r = 0.9264, n = 10). \) It declined at 0.04 \( \mu \)mol/L per year \( (r = 0.8287, n = 3) \) for the Toronto Western Health Unit from 1984 to 1988, and at 0.06 \( \mu \)mol/L per year \( (r = 0.9441, n = 4) \) for the City of Toronto from 1984 to 1992. The lead in leaded gasoline declined by an average of 3.6 \( \times 10^8 \) g per year between 1982 and 1990 \( (r = 0.9604, n = 8). \) Moreover, the regression analysis showed that the decline in blood lead concentrations overall was closely.

Fig. 1. Decline in the geometric mean of the blood lead concentrations related to a decline in the lead consumed in leaded gasoline in Ontario for 1983–1992.

Total lead consumed per year, \( \square \); blood lead concentrations in the Toronto Eastern Health Unit (\( \bullet \)), in the Toronto Western Health Unit (\( * \)), in the City of Toronto (\( \blacksquare \)), and in other regions of Ontario (\( \bullet \)).