Radium in drinking water and the risk of death from bone cancer among Ontario youths

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Objective: To determine whether residents of Ontario who are exposed to radium 226 naturally occurring in drinking water are at increased risk of bone cancer.
Design: A population-based case–control study of records from death and birth registries. Water samples were obtained from residences at the time of birth and of death.
Setting: Ontario.
Participants: All Ontario-born people under the age of 26 years who died of bone cancer between 1950 and 1983. Control subjects were those who died of any other disease matched by age, sex and year of death.
Outcome measures: Radium exposure distributions and estimation of risk.
Results: An association was found between death from bone cancer and exposure to radium at the birthplace residence in concentrations of 7.0 mBq/L or more (odds ratio 1.58, 90% confidence interval [CI] 1.01 to 2.50; $p = 0.047$). There was a statistically significant exposure–response relation ($p = 0.045$). The increase in risk was similar for the main types of childhood bone cancer: osteosarcoma, Ewing's sarcoma and chondrosarcoma.
Conclusions: The estimated risk at these exposure levels is much higher than would be predicted. The association may be spurious, the point estimates of risk may be too high, or risk factors derived from other exposure circumstances may not be valid for exposure to radium beginning in the prenatal period. Should the findings be confirmed, consideration might be given to removing radium from drinking-water sources.

Objectif : Déterminer si les résidents de l'Ontario exposés au radium 226 naturel présent dans l'eau potable risquent davantage d'être victimes de cancer des os.
Conception : Étude cas–témoin, en fonction de la population, d'enregistrements tirés des registres des décès et des naissances. On a obtenu des spécimens d'eau des résidences au moment de la naissance et du décès.
Contexte : Ontario.
Participants : Tous les Ontariens de moins de 26 ans décédés des suites d'un cancer des os entre 1950 et 1983. Les sujets témoins sont ceux du même âge, du même sexe et décédés la même année des suites de toute autre maladie.
Mesures des résultats : Répartitions de l'exposition au radium et estimation du risque.
Résultats : On a constaté un lien entre les décès des suites d'un cancer des os et l'exposition, au lieu de résidence à la naissance, à des concentrations de radium de 7,0 mBq/L ou plus (risque relatif de 1,58, intervalle de confiance (IC) à 90 % de 1,01 à 2,50; $p = 0,047$). Il y avait un rapport important sur le plan statistique entre l'exposition et les réactions ($p = 0,045$). L'augmentation du risque était semblable pour les principaux types de cancer des os chez l'enfant : ostéosarcome, sarcome d'Ewing et chondrosarcome.
Conclusions : Le risque estimé à ces niveaux d'exposition est beaucoup plus élevé qu'on ne l'aurait prédit. Le lien peut être attribuable au hasard, les estimations ponctuelles du risque peuvent être trop élevées, ou les facteurs de risque tirés d'autres circonstances liées à l'exposition peuvent ne pas être valables dans le cas de l'exposition au radium qui commence au cours de la période prénatale. Si les constatations sont confirmées, on pourrait envisager d'éliminer le radium des sources d'eau potable.

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People are exposed to environmental radiation from many sources. With the possible exception of the association between lung cancer and exposure to radon,[1] it has not been possible to demonstrate an increased risk of cancer in populations exposed to elevated levels of environmental radioactivity. Radium is present in soil, foods and groundwater. Isotopes of radium occur naturally, arising as decay products of heavier elements present in soil and rock. The main isotopes are radium 224 (half-life 3.6 days), radium 226 (half-life 1620 years) and radium 228 (half-life 5.8 years). In communities where wells are used, drinking water can be an important source of ingested radium.

Radium is absorbed from food and water in the gastrointestinal tract. Although most is excreted, bone becomes the principal repository for retained radium because of the chemical similarity between radium and calcium. Radium can cross the placenta, and the ratio of radium to calcium in the newborn reflects the ratio in the mother's blood. The limited evidence shows that under conditions of long-term intake the concentration of radium in the body is nearly invariant throughout life.[2]

Information on the health effects of radium comes from extensive studies of ^{224}Ra, ^{226}Ra and ^{228}Ra in humans and in laboratory animals. The most important effects are bone and sinus cancers. A recent estimate of the risk of these two types of cancer from the lifelong ingestion of 200 mBq of ^{226}Ra per day was 9 and 12 cases respectively per million population during a lifespan of 75 years on average.[3] (The becquerel [Bq] is a measure of the activity of a radioactive nuclide. It is equal to 1 disintegration per second. A millibecquerel thus corresponds to one disintegration per 1000 seconds. The older system of units measured activity in picocuries [pCi]; 1 pCi = 37 mBq.)

There have been three attempts in the United States to determine whether populations that drink water containing elevated levels of radium have an increased risk of cancer. In the first study the US Public Health Service and the Argonne National Laboratory[4] studied rates of death from bone cancer in communities in Iowa and Illinois in which public water supplies contained at least 3 pCi/L (110 mBq/L) of radium. In control cities the radium content was less than 1 pCi/L (37 mBq/L). Among residents under the age of 30, there were 25 deaths from bone cancer in the exposed towns and 15 in the unexposed towns (one-tailed test, $p = 0.08$). There was no information about individual exposures, and an association was uncertain because of population mobility.

In the second study, of Iowa towns, no cases of bone cancer were reported.[5] The incidence rates of lung and bladder cancers among men and of breast and lung cancers among women were higher in towns with a ^{226}Ra level of more than 5 pCi/L (185 mBq/L) in the drinking water.

A significant association between leukemia and groundwater contamination with radium was found in Florida.[6] All exposure inferences were based on area assessments, and interpretation of the results was confounded by substantial population mobility.

In 1981 the Ontario Ministry of the Environment conducted a survey of municipal wells to assess the natural levels of radioactivity in deep groundwaters in parts of southwestern Ontario. Radium concentrations in several towns exceeded the Canadian target concentration of 100 mBq/L. A subsequent ecologic analysis indicated that rates of death from bone cancer might be elevated in those counties overlying an aquifer known to contain radium. This article describes an epidemiologic study undertaken to determine whether there was an association between measured levels of radium in home drinking water supplies and an increased risk of death from bone cancer.

Methods

A case–control study was conducted. A computer tape of Ontario death registrations was used to identify people 25 years of age or less who died of bone cancer between 1950 and 1983. This period was chosen because bone cancer is sufficiently rare that a wide time span was needed to increase the sample size. The start point was 1950 because identification data were available in machine-readable form from that point; 1983 was the last year for which such data were available at the time subjects were being identified for the study.

Birth and death certificates of Ontario-born people were abstracted. Each subject was matched, with the use of random numbers, with a control subject who had died of any disease other than bone cancer. Matching variables were birth in Ontario, sex, age and year of death.

A total of 335 matched pairs were identified. Because of the 34-year span over which the deaths occurred, no attempt was made to collect and review pathologic records. Data from the Ontario Cancer Treatment and Research Foundation (OCTRF) concerning people with bone cancer since 1964 indicated that seven of the case subjects did not have primary bone tumours; they and the matched control subjects were excluded from the study. Of the remaining 328 case subjects 270 (82.3%) had an OCTRF file or an indication of surgery or autopsy on the death certificate. This was taken as confirmatory evidence for the diagnosis listed on the death certificate.

Because of confidentiality restrictions pertaining to the use of death certificates, families could not be contacted for exposure information, and drinking water sources had to be determined from documents. The patient's address at the time of death and the mother's address at the time of birth were obtained from the death and birth certificates. For 24 case and 23 control subjects it was not possible to obtain information detailed enough to identify a birthplace sampling site. An additional 21 case and 20 control subjects were born in remote north-

ern locations, which were not sampled for financial reasons. The study population thus consisted of 283 case subjects and 285 control subjects for whom a measurement of the concentration of radium in birthplace water supplies was obtained. Table 1 provides demographic information about the study population.

Water was sampled by the Ontario Ministry of the Environment from 1987 to 1992. The people collecting the samples indicated that they were from the ministry and were performing a survey of water quality; they did not ask about the health status of current or former residents. Individual water samples were collected from residences supplied by wells. Samples from municipal supplies were obtained for residences served by communal sources. When a water softener was present an attempt was made to bypass the softener if it had been installed since the birth of the subject. It was sometimes not possible to obtain samples from the source used by the subject because the well could not be accessed. Frequently a sample was available from a newer well on the property or a neighbour's well, and information was obtained on the depth and location of the target and substitute wells. If the substitute source drew water from the same geologic stratum it was taken to be representative of the target well. If a substitute source was not representative, the sampling result was not used in the analysis and the subject was excluded. The sampling distribution among the study subjects was as follows: target well (263 cases, 263 controls), substitute well sampled on the property (4 cases, 2 controls) and substitute well sampled off site (16 cases, 20 controls).

Water was collected in acidified jugs and analysed by the Radiation Protection Laboratory of the Ontario Ministry of Labour. Neither the people collecting the samples nor the laboratory staff knew the case–control status of the subjects. The samples were filtered, and the radon emanation method was used to measure the ^{226}Ra concentration. The radium isotopes were coprecipitated with barium sulfate, and the barium–radium sulfate was taken up into solution with a complexing agent, basic diethylenetriamine penta-acetic acid. The solution was transferred to a bubbler and aerated to remove all radon 222. After aging for about 3 weeks, the radon was deemanated and transferred to a counting chamber, in which ^{222}Rn and its daughters were counted using scintillation counting techniques. In addition to its internal quality-control procedures and its participation in international laboratory standardization programs, the Radiation Protection Laboratory analysed duplicate samples collected in the field, distilled water samples ("blanks") and samples spiked with a known concentration of radium.

Table 1: Demographic characteristics of people in Ontario less than 26 years of age who died of bone cancer (case subjects) or other types of disease (control subjects) from 1950 to 1983

Characteristic	Group; no. of subjects: Case (n = 283)	Control (n = 285)
Sex		
Male	179	171
Female	104	114
Age, yr		
< 12	59	60
12–18	138	142
19–25	86	83
Year of death		
1950–1964	86	87
1965–1974	94	97
1975–1983	103	101

A radium-exposure value was assigned to each subject based on the sampling result at the birthplace residence. Subjects were assigned to the "reference" category if the radium concentration was less than 7.0 mBq/L; others were classified as "exposed." (Information on how the cutpoint between the reference level and the exposure level was selected is available from the author upon request.) Exposed subjects were further classified into two categories: those whose birthplace water sample had a radium level of 7.0 to 29.9 mBq/L and those whose sample had a radium level of 30.0 mBq/L (approximately 1 pCi/L) or more.

Odds ratios and confidence intervals were calculated and tests for trends and common odds ratios performed from exact analysis of unstratified and stratified $2 \times k$ tables with the use of the EGRET computer program (EGRET Statistical Software, Statistics and Epidemiology Research Corp., Seattle, 1988).

Logistic regression analysis, performed with the EGRET program as well, was used to determine the exposure–response relation with the use of the actual exposure value for each subject. In the first model, only the logarithm of radium concentration was considered as an explanatory variable. Subsequent models allowed for the possibility that the odds ratios would be affected by the matching variables sex, age and year of death.

The null hypothesis was that there is no association between bone cancer and exposure to radium-containing drinking water. Since radium causes bone cancer the alternative hypothesis was that exposure increases the risk of bone cancer. One-tailed p values and 90% confidence intervals (CIs) are thus presented.

Results

Grouped analysis

Overall, 87% of the subjects fell into the reference category. In the exposed category radium concentrations ranged from 7.0 to 160.0 mBq/L (Table 2). The exposed category was subdivided into radium levels ranging from 7.0 to 29.9 mBq/L and those of 30.0 mBq/L or greater.

The mean (and geometric mean) radium levels for the two subcategories were 12.2 (11.4) and 75.5 (66.3) mBq/L respectively.

Radium was present at a level of 7.0 mBq/L or more in the birthplace drinking water of 15.2% of the case subjects, as compared with 10.2% of the control subjects (odds ratio [OR] 1.58, 90% CI 1.01 to 2.50; $p = 0.047$). There was a significant trend in the odds ratio with increasing level of exposure ($p = 0.045$) (Table 2). When the results were adjusted individually for sex, age and year of diagnosis of bone cancer, there was essentially no effect on the odds ratio.

Logistic regression analysis

The risk of death from bone cancer was significantly associated with the level of radium exposure ($p = 0.04$). The functional form of the exposure–response relation was markedly sublinear, with an estimated exponent of 0.28. Sex, age and year of death were not significant factors. A sensitivity analysis, in which subjects (both case and control) in the exposed category were sequentially excluded from the data set, indicated that the results were insensitive to individual exposure values.

Risk by type of bone cancer

Table 3 presents an analysis of risk by type of bone cancer. The odds ratio was elevated for each type. Within the limitations imposed by the small number of cases of each type (particularly of chondrosarcoma) and the correspondingly wide CIs, the increase in risk could be said to be the same for each of the subtypes of bone cancer. There was no increased risk of osteosarcoma for females in the exposed category, but the number of subjects was so small that the difference in risk between the males and the females was compatible with chance fluctuation.

Table 4 shows the association between radium exposure and risk of death from bone cancer when only the control subjects who died of other types of cancer were used as the comparison group. In this case only 5.3% of

Table 2: Exposure status of subjects, by radium level in drinking-water samples from birthplace residence

Exposure status; radium level, mBq/L	Group; no. (and %) of subjects		Odds ratio (and 90% CI*)
	Case	Control	
Reference category			
< 7.0	240 (84.8)	256 (89.8)	1.00
Exposed category			
7.0–29.9	35 (12.4)	26 (9.1)	1.44 (0.88–2.35)†
≥ 30.0	8 (2.8)	3 (1.1)	2.84 (0.81–12.60)†
Total	43 (15.2)	29 (10.2)	1.58 (1.01–2.50)

*CI = confidence interval.
†$p = 0.045$, test for trend.

Table 3: Estimated risk of bone cancer, by type of cancer

Type of bone cancer	Exposure category; no. of case subjects		Odds ratio* (and 90% CI)	Common odds
	Exposed	Reference		
Osteosarcoma				
Males	15	72	1.88 (0.93–3.78)	
Females	5	53	0.80 (0.26–2.22)	
Total	20	125	1.41 (0.80–2.45)	$p = 0.33$
Ewing's sarcoma				
Males	9	64	1.27 (0.55–2.81)	
Females	6	29	1.75 (0.60–4.76)	
Total	15	93	1.44 (0.77–2.64)	$p = 0.73$
Chondrosarcoma				
Males	3	7	3.84 (0.79–15.30)	
Females	0	2	0.00 (0.00–30.90)	
Total	3	9	2.90 (0.64–10.50)	$p = 1.00$
Other (both sexes)	5	13	3.40 (1.15–9.58)	
All	43	240	1.58 (1.01–2.50)	

*For calculating the odds ratios the number of control subjects in the exposed category was 29 (17 males, 12 females), and the number in the reference category was 256 (154 males, 102 females).

the control subjects were in the exposed category, as compared with 15.2% of the case subjects.

Relation between radium exposure at place of birth and at place of death

The focus in this analysis was on exposure to radium at the birthplace residence, because this is deemed to reflect prenatal and early childhood exposure. The address at death was available from the death certificate, but there was no information about the length of time spent at any residence. A water sample from the place of death was available for 550 (96.8%) of the subjects. For 252 (89.0%) of the case subjects and 255 (89.5%) of the control subjects the exposure status (exposed or not exposed to water containing 7.0 mBq/L or more of radium) was the same at both addresses. Eleven case and five control subjects had been exposed at the birthplace residence but not at the deathplace residence. Conversely, 13 case and 14 control subjects were not exposed at the birthplace residence but were exposed at the deathplace residence. These findings suggest that the subjects in the two groups were similar in their mobility patterns.

Risk of cancer other than bone cancer

Since 95 (33.3%) of the control subjects died of cancer other than bone cancer it was possible to examine whether the risk of cancer other than bone cancer was associated with radium exposure. According to the data in Table 4, there was no evidence of an association except between radium exposure and bone cancer.

Discussion

The aim of this study was to determine whether residents of Ontario who are exposed to naturally occurring ^{226}Ra in their drinking water are at increased risk of bone cancer. The case–control design permitted a sampling program of manageable size. The study population was restricted to subjects 25 years of age or less in order to lessen the problem of population mobility. Particular attention was paid to the radium content of the water supply at the birthplace residence, on the presumption that this measurement reflects prenatal and early childhood exposures. If consumption of the radium-containing water continues after birth, the level of radium in the child's bones will remain in equilibrium with that in the water. If the child consumes water with a lower level of radium, irradiation of bone will continue while deposited radium is gradually removed by remodelling. If, in contrast, a child moves from a supply with a low radium level to one with a higher level it may take years for the radium level in the bones to come into equilibrium with the new water source.

The risk of death from bone cancer was elevated among the children whose residences at birth were supplied with water containing a radium level of 7.0 mBq/L or more. There was a trend of increasing risk with increasing exposure. There was no statistical evidence in this study that radium-associated risk was modified by age, sex or year of death.

Osteogenic sarcoma is a well-accepted result of exposure to radium. It has been observed among dial painters[7] exposed to ^{226}Ra and ^{228}Ra and among medical subjects injected with ^{224}Ra.[8] Ewing's sarcoma has not previously been associated with exposure to radium, al-

Table 4: Estimated risk of bone cancer versus other types of cancer and estimated risk of other types of cancer versus nonmalignant diseases

Variable	Exposure category; no. of subjects				Odds ratio (and 90% CI)	Common odds
	Case subjects*		Control subjects†			
	Exposed	Reference	Exposed	Reference		
Bone cancer v. other types of cancer*						
Males	30	149	4	52	2.61 (0.99–8.45)	
Females	13	91	1	38	5.39 (0.93–117)	
Total	43	240	5	90	3.17 (1.36–8.66)	$p = 1.00$
Leukemia v. nonmalignant disease†	3	34	26	222	0.75 (0.18–2.29)	
Cancer other than bone cancer v. nonmalignant disease‡	5	90	24	166	0.39 (0.14–0.94)	

*Case subjects are people in original case group; control subjects are those in original control group who died of cancer.
†Case subjects are people in original control group who died of leukemia; control subjects are all other subjects in original control group.
‡Case subjects are people in original control group who died of cancer; control subjects are all other subjects in original control group.

though there have been cases reported following radiotherapy for other types of cancer.[9] Among 218 children treated with ^{224}Ra for tuberculosis of the bone osteosarcoma or chondrosarcoma developed in 35, but there were no reported cases of Ewing's sarcoma.[9] Since Ewing's sarcoma is a disease that affects young people it is not surprising that it was not found among the dial painters, who were first exposed to radium as teenagers or adults.

Characteristic chromosomal abnormalities are present in both osteogenic sarcoma and Ewing's sarcoma. Osteosarcoma occurs with high frequency in people who carry a mutant tumour-predisposing copy at the retinoblastoma locus on chromosome 13.[10] Experimental evidence supports the theory that radiation destroys the remaining normal copy of this gene. The gene has apparently been inactivated and grossly altered in a number of cases of osteosarcomas that have no connection to radiation treatment.[10] In Ewing's sarcoma, there is a specific cytogenetic abnormality, a reciprocal translocation between chromosomes 11 and 22: (11;22) (q24;q12). Because of its ability to disrupt strands of DNA the production of translocations is one of the well-known genetic effects of ionizing radiation.

The main strength of this study was its population-based design. Study subjects were all Ontario-born people who died of bone cancer between 1950 and 1983. Cases were excluded only if a sampling location could not be identified or, for economic reasons, the people were living in the remote north. The control subjects were randomly selected from among Ontario-born youths who died of nonaccidental causes. Use of deceased control subjects does not introduce bias if one assumes that (a) exposure to radium in drinking water neither causes nor prevents death from any disease other than bone cancer and (b) there is no association between social factors that are linked to higher risk of childhood death and exposure to radium. These assumptions were supported empirically by the findings that the association persisted when the cancer victims from the control series were used as the comparison subjects and that there was no association between exposure to radium-containing water and any other type of cancer.

Because lifetime residence histories were not available, inferences were based on the results of the sampling at the birthplace residences. Given the metabolism and retention of radium it is plausible that prenatal and early childhood exposures are of prime importance in determining the dose during the first decades of life.

A source of uncertainty is whether the correct water supply was sampled. Because relatives of the subjects could not be contacted, there is no confirmation that the supply sampled was actually the source of the family's drinking water. However, it is expected that the misclassification due to sampling an incorrect water source is nondifferential. Studies of the temporal variability of the activity of radium isotopes in groundwater systems have revealed minimal variation.[11,12]

The association observed in this study was surprising because all of the radium concentrations measured were well within Canadian water-quality guidelines. In addition, the point estimate of risk was much higher than would be predicted from risk factors derived from populations occupationally or medically exposed to radium. There are three plausible explanations for the disparity. First, the observed association may be spurious and not causal. Second, the association may be causal and the point estimates of risk too high, the true risk lying toward the lower end of the confidence interval. Even though we included all cases of bone cancer occurring in Ontario, the power of the study was such that an observed odds ratio of 1.58 was of only borderline statistical significance, and the 90% CI ranged from 1.01 to 2.50. Third, the association may be causal and the risk factors derived from other exposure circumstances not valid for continuous exposure to the alpha radiation of radium beginning in the prenatal period. The results of studies of prenatal obstetric exposure to x-rays and of those involving atomic-bomb survivors suggest that fetuses and young children are more sensitive than older people to the carcinogenic effects of ionizing radiation. Nevertheless, both bone cancer and radium exposure are sufficiently rare that there are still substantial statistical uncertainties about the existence of an association between radium-containing drinking water and bone cancer and the size of the odds ratio.

If the observed association between environmental exposure to radium and an increased risk of bone cancer is real, the implications for public health depend on both the size of the risk factor and the proportion of the population exposed to radium. If 10% is the estimated proportion of the Ontario population exposed to radium and 1.58 is the estimated odds ratio, the population attributable risk[13] is 0.055. In this study 328 people died of bone cancer. Thus, the number of deaths possibly attributable to radium exposure is 18 (5.5%), one death occurring every 2 years on average.

A follow-up study of the association between nonfatal cases of bone cancer and exposure to radium-containing water has been funded. In that study, lifetime-residence histories will be obtained by questionnaire, and a more complete analysis of lifetime exposure will be possible. It is hoped that this next study will confirm or refute the findings reported here.

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Conferences
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Jan. 26–29, 1995: 21st Annual Predoctoral Education Conference — Partnerships in Education
Charleston, SC
Program Department, Society of Teachers of Family Medicine, PO Box 8729, Kansas City, MO 64114; tel (800) 274-2237, (816) 333-9700, ext. 4510

Mar. 1–5, 1995: 15th Annual Family in Family Medicine Conference — Taking Care: Healthy Healers, Families and Communities
Amelia Island, Fla.
Program Department, Society of Teachers of Family Medicine, PO Box 8729, Kansas City, MO 64114; tel (800) 274-2237, (816) 333-9700, ext. 4510

Mar. 20–25, 1995: Youth Health Assembly (includes 6th International Congress on Adolescent Health, Youth for Youth Health Conference and 27th Society for Adolescent Medicine Annual Meeting; cosponsored by the Society for Adolescent Medicine and the International Association for Adolescent Health)
Vancouver
Abstract deadline: Sept. 14, 1994
Venue West Conference Services Ltd., 645–375 Water St., Vancouver, BC V6B 5C6; tel (604) 681-5226, fax (604) 681-2503

Apr. 26–29, 1995: Canadian Association of Speech-Language Pathologists and Audiologists
Ottawa
Abstract deadline: Oct. 28, 1994
Linda J. Garcia, CASLPA Conference '95, Programme d'audiologie et d'orthophonie, University of Ottawa, 545 King Edward Ave., Ottawa, ON K1N 6N5; tel (613) 564-9918, fax (613) 564-9919

May 6–10, 1995: 28th Annual Spring Conference
New Orleans, La.
Program Department, Society of Teachers of Family Medicine, PO Box 8729, Kansas City, MO 64114; tel (800) 274-2237, (816) 333-9700, ext. 4510

May 11–14, 1995: American Association for the History of Medicine 68th Annual Meeting (in conjunction with meetings of other history of medicine and health care societies)
Pittsburgh, Penn.
Dr. Jonathon Erlen, 123 Northview Dr., Pittsburgh, PA 15209

Aug. 7–11, 1995: 4th International Congress on Amino Acids
Vienna, Austria
Abstract deadline: Apr. 30, 1995
Dr. Gert Lubec, Department of Paediatrics, University of Vienna, Währinger Gürtel 18, A-1090 Vienna, Austria; fax 011-431-40400-3238