**Electrolyte Loss in Sweat and Iodine Deficiency in a Hot Environment**

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**ABSTRACT.** The authors studied electrolyte loss from profuse sweating in soccer-team players and evaluated the relationship between this source of iodine loss and iodine deficiency. Thirteen male soccer-team players and 100 sedentary students from the same high school were evaluated for 8 d, during which the players were training. The authors analyzed 208 sweat samples to determine losses of iodine, sodium, potassium, and calcium in sweat. Excretion of urinary electrolytes by the subjects was also measured. The mean losses of iodine, sodium, potassium, and calcium in sweat following a 1-hr game were 52 pg, 1,896 mg, 248 mg, and 20 mg, respectively; the ratios of sweat loss to urinary daily loss of the four electrolytes were 0.75, 0.2, 1.88, and 0.92, respectively. Urinary iodine was significantly (p < .02) lower than the normal level of 50 pg/gm creatinine in 38.5% of the soccer players, compared with 2% of the sedentary students. Forty-six percent of the players had Grade I goiter, compared with a mere 1% of the sedentary students (p < .01). The results of the study suggest that loss of iodine through profuse sweating may lead to iodine deficiency, and loss of electrolytes through sweating may have a dietary significance for heat-stressed individuals or for individuals who perform heavy workloads.

<Key words: electrolyte, hot environment, iodine deficiency, sweat>

WHEN HUMANS are exposed to a hot environment or to warm surroundings, a balance can be established between heat production and heat loss. Once the set-point temperature is exceeded, heat dissipation is increased by sweating, and heat conservation is decreased by peripheral vasodilatation.

Sweat contains relatively large amounts of minerals and electrolytes, and profuse sweating leads to significant losses of these elements. The main substance in sweat is sodium chloride, at a concentration between 0.2 gm/100 ml and 0.4 gm/100 ml. Sodium, potassium, and calcium also abound in sweat. The electrolytes sodium, potassium, and calcium are essential to neuromuscular excitability, secretory activity, and membrane permeability, among many other cellular functions. In addition, electrolytes are dominant factors in the control of fluid movement. The consequences of deficiencies in these electrolytes include dehydration, cardiac arrhythmia, tremors, and muscular weakness, among others.

Iodine deficiency is the main cause of endemic goiter, and the relationship between iodine intake and development of goiter has been studied widely. Iodine deficiency still exists in some areas of the world; a total of 117 countries reportedly have this problem. After 1960, when iodized salt was introduced as a prophylactic treatment in many countries, the proportion of the population affected by goiter dropped to below 5%. Goiter is endemic when it affects more than 10% of a population. Until now, however, the relationship between excretion of iodine through sweat and the occurrence of iodine deficiency or goiter has received little attention.

Sung and Chen found thyroid enlargement in sever-
al workers who were exposed in particularly hot areas of the plant. Detailed explorations of climatic conditions in the factory were conducted and documented. The investigators reported a relationship between increasingly elevated workplace radiant temperatures and the presence of thyroid enlargement in workers who occupied those stations. That investigation led us to speculate about the possible relationship between thyroid enlargement or goiter and profuse sweating.

We, therefore, sought to analyze the relationship between loss of iodine caused by profuse sweating and the presence of iodine deficiency or goiter. In addition, we examined the relationship between profuse sweating and loss of sodium, potassium, calcium, and iodine.

Materials and Method

Subjects. The study occurred between 1989 and 1990. Potassium, sodium, calcium, and iodine concentrations in sweat and urine from 13 members of a well-trained high school senior soccer team and 100 sedentary students from the same school were studied. The sedentary students were selected randomly from a total of 15 classes.

All subjects were between 16 y and 18 y of age. The soccer players and sedentary students consumed normal diets during the study period. Both groups were given questionnaires, and they supplied written confirmation about (a) whether they exercised every day and for how long, (b) whether they had taken any medication(s) during the previous 6 mo, (c) how often they consumed seafood and meat, and (d) amounts of cabbage and other goitrogenic vegetables they had eaten during the previous 4 mo. None of the subjects had ever been hospitalized for a thyroid problem. None of the sedentary students participated in organized exercise activities during the preceding year. No significantly different answers for the two groups were noted, except that reported exercise differed between the two groups.

Concentrations of sodium, potassium, and calcium were measured every day during an 8-d period. The concentration of iodine was also measured for 8 d; however, given that high-iodine food supplements were provided for the players on the 5th–8th d of training (i.e., for another related study14), we used only the iodine data obtained from days 1–4 for analysis in this study.

Experimental conditions. This study was approved by the Institute Review Board of the School of Public Health, National Taiwan University; in addition, we obtained parental and all subjects' consent prior to the experiment. The 13 players and 100 sedentary students were fully informed of the objectives, procedures, and requirements of study. During the summer months prior to the study, players trained between 2:30 P.M. and 5:30 P.M. for 5 afternoons each week for 3 mo (except on rainy days). The air temperatures during the day were between 30 °C and 35 °C.

Measurements of body weight and sweat collection were performed in a single room in a concrete building that was adjacent to an athletic ground. The windows and doors were closed, thus creating a climate chamber in which the temperatures ranged between 32 °C and 37 °C. We kept wind velocity below 0.1 m/s to minimize heat exchange, and the relative humidity was 30–50% in the room.

Body weight measurement. We determined each player's weight loss by calculating the variation between body weight before and after each training day. The 3-hr training session included pre-game training during a 1-hr informal game during a 1-d period. The players had no fluid refreshments during the pregame exercise and during the 1-hr game. They did not urinate during the training courses; therefore, we were able to measure weight loss strictly relative to sweating. The accuracy of the platform balance was ± 20 gm. This acute weight loss represented pulmonary water vapor loss and metabolic loss, as well as loss from sweating.

Sweat and urine collection. Sweat samples were collected from the players on Monday through Friday of one week and on Monday through Wednesday of the following week. The samples were collected following 30 min of pregame exercise and again following the 1-hr game. The chests and backs of the players were washed thoroughly with deionized water, and they were dried with sterilized towels for which iodine was not detectable. The iodine concentration of the deionized water was determined with the catalytic reduction method.15 To facilitate rapid collection of newly appearing sweat, we used a plastic collector that measured 5 cm × 9 cm × 2 cm and held it next to the skin of the back and chest; this was used instead of filter paper16 or a macroduct.17 Between 5 ml and 10 ml of sweat was collected from each player in a 5-min period. Each sweat sample was transferred to a 20-ml plastic tube, and 1 drop of xylene was added as a preservative. A 25-ml urine sample was collected from each player between 2:00 P.M. and 2:30 P.M. (i.e., before pregame exercise) on each of the 8 d. Twenty-five-ml urine samples were collected from each of the sedentary students in accordance with the same schedule (i.e., at 2:30 P.M. – 3:00 P.M.), but collections occurred on separate days. The urine samples were preserved with 2–3 drops of xylene.

Electrolyte concentrations. To calculate sweat electrolyte loss, we corrected the weight of sweat loss, derived from players' weight loss after the game, by the weight loss of breathing, which we estimated to be 22 gm/hr · person.18 Estimated loss of electrolytes through sweating for each 1-hr game = (mean of electrolyte concentration in sweat) × (body weight loss in a game)/(specific gravity of sweat [1.002]). The mean electrolyte concentration in sweat was based on the average of two sweat samples—one obtained after the pre-game exercise, and the other following a 1-hr game.

We used a heat-system sonicator equipped with a 1.6-mm tapered microtip probe to homogenize the sweat samples, and homogenization of the urine samples was achieved with a vortex.

We used the Folin-Wu method19 to determine urine creatinine. We used a modified version of the Joseph
Benotti method to determine the concentration of iodine in sweat and urine samples. An Hitachi 180/70 atomic absorption spectrophotometer measured the concentration of calcium, potassium, and sodium in the sweat and urine samples provided by the players; recovery was determined in spiked samples at three concentrations. The percentage mean recoveries (mean ± standard deviation) in sweat for the three elements were as follows: calcium, 110 ± 6 (coefficient of variance [CV] = 6); potassium, 94 ± 4 (CV = 4); and sodium, 92 ± 5 (CV = 5). The mean percentage recoveries in urine were as follows: calcium, 105 ± 6 (CV = 6); potassium, 92 ± 5 (CV = 5); and sodium, 88 ± 4 (CV = 5). No measurements of these three elements were made for the sedentary students.

Urinary iodine is expressed as micrograms per gram creatinine (µg/gm Cr). Samples with a creatinine concentration of less than 0.5 gm/l were discarded. We used two equations to estimate electrolyte losses in urine and sweat. In the first equation, we estimated loss of electrolytes through sweating per 1-hr game: (mean of electrolyte concentration in sweat) × (body weight loss in a game)/specific gravity of sweat [i.e., 1.002]). The mean electrolyte concentration in sweat was based on two average sweat samples: one obtained after 30 min of pregame exercise and the other following a 1-hr game. In the second equation, we estimated loss of electrolytes through urine per day: (mean of electrolyte concentration [in µg/gm Cr]) × (0.023 gm Cr/kg) (body weight).

Classification of iodine deficiency and endemic goiter. Endemic goiter was measured with the method developed by Pere et al. and recommended by the World Health Organization in 1960. The method is based on a "grading" of the size of goiter into five categories. The results were designated as follows: cases with no goiter, grade 0; cases with palpable goiter with the head in a normal position and readily visible with the head thrown back, grade 1; cases with a visible goiter, grade II; and cases with a very large goiter, grade III. The examination was conducted while the subject and examiner remained standing; the subject's head was initially in the normal position, followed by an extension of the neck. The subject's neck was well exposed to light. The examiner then palpated the thyroid area with his right forefinger.

Folli et al. classified severity of iodine deficiency into five groups, by increasing severity. The concentration of iodine in urine in the majority of subjects exceeded 50 µg iodine/gm Cr. Folli et al. described the five groups as follows: Group 1: no individuals excrete less than 25 µg l/gm Cr; Group 2: no individuals excrete less than 25 µg l/gm Cr, although some (~25%) are in the 26-50 µg l/gm Cr range; Group 3: no more than 15% fall into the 0-25 µg l/gm Cr range, and a larger proportion fall into the 26-50 µg l/gm Cr range; Group 4: more than 15% of the excretion values fall into the 0-25 µg l/gm Cr range (but this percentage is not greater than in any of the other 25-µg increments); and Group 5: the largest proportion of individuals excrete in the 0-25 µg l/gm Cr range. This classification has the advantage that its grading is based on a judgment of the whole distribution of values, rather than on the basis of a single mean.

Statistical analysis. We used Fisher's exact test to compare urinary iodine concentrations and prevalence of goiter in the soccer group with those in the sedentary group.

Results

The mean concentrations of the four electrolytes found in sweat of the players are shown in Table 1. The mean (± standard error) concentrations (in descending order) are as follows: sodium, 1,270 ± 624 mg/l; potassium, 155 ± 70 mg/l; calcium, 12.6 ± 4.7 mg/l; and iodine, 37.0 ± 6.6 µg/l.

The mean concentrations of the four electrolytes in the urine of the soccer players during the 8-d observation period are shown in Table 2. Mean concentrations (in descending order) are as follows: sodium, 4,340 ± 930 mg/l; potassium, 2,460 ± 410 mg/l; calcium, 29.7 ± 16.8 mg/l; and iodine, 59 ± 28.0 µg/l.

The concentration of sodium in sweat was lower than the concentration in urine for all of the players. The same was true for potassium concentrations; the mean concentration of potassium in urine was 16 times higher than that in sweat. The mean calcium concentration in sweat was also much lower than in urine in all 13 players; however, calcium concentrations ranged between 6.6 mg/l and 23.9 mg/l. Iodine concentrations were slightly lower in the players' sweat than in their urine, but only minor variations existed in iodine concentrations between individual players or between samples provided by the same player.

The mean original body weight and mean body weight loss per day in the soccer players, before and after the first 4 d of training, are shown in Table 3. At the start of the 4-
d period, the mean body weight was 62.45 ± 6.79 kg; at the end of the training, the mean body weight loss was 1.54 ± 0.57 kg. Subject Y, the goalkeeper, experienced the least average weight loss (0.254 ± 0.04 kg) during the 4-d period.

The players' estimated daily losses of sodium, potassium, calcium, and iodine in sweat, and the estimated urinary losses of these electrolytes during 1 hr of training, are shown in Table 4. These calculations were based on (a) loss of body weight (weight of respiratory water loss excluded) during exercise and (b) the relationship between urinary Cr concentration and body weight. Subject Y, the goalkeeper, again scored the lowest; he lost just 0.4 g sodium during 1 hr of play—one-sixth the average loss of other players. If the goalkeeper is excluded, the ratio of sweat loss to urinary loss of sodium ranged from 36.7% to 155.0%. The greatest amount of sodium in sweat was 5.9 gm for a 1-hr game. The average hourly sodium loss from sweat among the players was 78.7% of their daily urinary sodium excretion.

The mean sodium excretion of potassium for the 13 players was 248 mg for a 1-hr game. The mean daily urine potassium loss was 1,188 mg. Potassium loss from sweat during a 1-hr game was 20% of the daily urinary potassium loss. The sweat loss of calcium ranged from 2.6 mg to 57.4 mg per game (mean = 20.0 mg). The mean daily urinary calcium loss was 15 mg, and the ratio of sweat loss to urinary excretion of calcium ranged from 67–884%, except in subject Y, for whom the mean ratio was 188.1.

The iodine lost in sweat per game ranged from 11.6 µg to 99.8 µg (mean ± standard error = 51.9 ± 23.8 µg). The mean urinary iodine excretion per day was 86.2 ± 41.5 µg. Thirty-eight percent of the players had a mean iodine loss in sweat for a 1-hr game that exceeded the mean urinary iodine excretion; subject T scored the highest ratio of 367% (overall mean ratio = 92.4%).

We used the classification system proposed by Follis et al.23 to group the urinary iodine concentrations in athletes and sedentary students (Table 5). We found that 38.5% of the players had urinary iodine values that were less than 50 pg/g Cr, but only 2% of sedentary students evidenced so low a volume. The difference was significant (p < .001). The iodine deficiency among players was a Group 423 deficiency (i.e., not more than 15% of the values fall into the 0–25-µg range, but this percentage is not greater than in any of the other >5-µg increments), whereas the urinary iodine concentrations in the sedentary students were normal (i.e., >50 µg iodine/gm Cr [Group 121]). We used the classification system of Pere et al.,22 and 46.1% of the athletes had grade I goiter, compared with only 1% of the sedentary students (p < .001 [Table 6]).

### Discussion

The highest electrolyte concentration in sweat and urine was sodium, followed (in descending order) by potassium, calcium, and iodine. In all 13 players, concentrations of the four electrolytes were lower in sweat than in urine—especially potassium, which had a urinary concentration 16 times greater than that found in sweat. Although excretion of urinary electrolytes fluctuated after food or drink was consumed (i.e., excretion functioned as a safety valve in the body's regulatory system14), the order of magnitude of electrolyte excretion in both urine and sweat was the same: sodium was excreted in thousands of ppm, potassium in hundreds of ppm, calcium in tens of ppm, and iodine in tens of ppb. The preceding amounts and order of magnitude have not been reported previously.
The mean losses of sodium, potassium, calcium, and iodine via sweating during a 1-hr game were 2.3 (1,896 mg vs. 3.0 g), 1/4 (248 mg vs. 1.2 gm), and 1/2 (52 µg vs. 150 µg), respectively, of the required daily amount. Higher ratios were found for sodium, potassium, and iodine, but sodium and potassium abound in the daily diet. In addition, sport drinks (i.e., typically contain 400 mg/l sodium and 200 mg/l potassium) are usually consumed by soccer players after exercise. We did not, therefore, find symptoms of sodium and potassium deficiency in the soccer players. In contrast, iodine content of food is uneven and, in some cases, scarce. In soccer players, such a loss is particularly significant.

The 3–8 µg/l (data not shown) of iodine in tap water and in commercial health drinks in Taipei City does not make up for the iodine loss that occurs via sweat of players, because their sweat contains an average of 37 µg/I of iodine.

Furthermore, in our study, soccer players did not supplement iodized salt, except with their regular diets. The subjects' favorite food was pork, not seafood, although Taiwan is an island supplied with plenty of seafood. According to the research questionnaire, 50% of the subjects disliked seafood. According to the Taiwan Health Administration (1972 and 1993 Recommended Daily Nutrient Allowance, Health Administration, Taiwan), the electrolytes lost from sweat among the players (compared with sedentary students) in a 1-hr game were 2.3 (1,896 mg vs. 3.0 g), 1/4 (248 mg vs. 1.2 gm), and 1/2 (52 µg vs. 150 µg), respectively, of the required daily amount. Higher ratios were found for sodium, potassium, and iodine, but sodium and potassium abound in the daily diet. In addition, sport drinks (i.e., typically contain 400 mg/l sodium and 200 mg/l potassium) are usually consumed by soccer players after exercise. We did not, therefore, find symptoms of sodium and potassium deficiency in the soccer players. In contrast, iodine content of food is uneven and, in some cases, scarce. In soccer players, such a loss is particularly significant.
thyroids were larger during the summer. The loss of
from 55% in summer to 39% in winter. There was a sea-
feces, urine, and sweat. Furthermore, the soccer players
who sweats up to 3-5 l daily, iodine excretion would
excretion of the entire trunk. The maximum sweat
ducts.

by Pere et al., found that the prevalence of goiter
among schoolchildren in New South Wales in 1965 fell
from 55% in summer to 39% in winter. There was a sea-
crease in urinary iodine output, from 52 µg/gm Cr in summer to 80 µg/gm Cr in winter. Ko27 noted that
thyroids were larger during the summer. The loss of
iodine via sweating in the summer might explain the
findings by both Hales et al,26 and Ko,27 although sea-
changes in diet cannot be ignored.

In accordance with the diagnosis methodology posited
by Pere et al.,22 46% of the soccer players had grade I goi-
ter, and 38.5% of the players had urinary iodine concen-
trations below 50 µg/gm Cr—a critical level for the diag-
nosis according to Follis’ classification. Therefore, two
distinct methodologies diagnosed goiter among the soc-
cer players. The Follis classification had the advantage
that its grading is based on a judgment of the whole dis-
bution of values, rather than on a single mean. Sonog-
raphy is a good instrument for determination of goiter,
but this method is inconvenient in field studies.

The iodine/creatinine ratio was used by Follis et al.23
and by Vought et al.25 as a single-urine specimen
indicative of iodine intake of schoolchildren. These
investigators suggested that estimates of urinary iodine
excretion derived in this manner are satisfactory for
field study, except in the presence of serious renal dis-
ease. Collection of urine for 24 hr from an in-hospital
patient and pooling the urine is practicable and accu-
rate. Controlling the quality of urine collection during
24 hr in the field study is less practical. In a medical set-
ing, it is not good enough that we use electrolytes
(mg/gm Cr) and body weight to estimate daily urinary
electrolyte excretion; in this study, however, it was suf-
cient for the measurement of urinary levels of iodine
in players and sedentary students.

Our method of sweat collection allowed us to collect
5–10 ml of sweat within a 5-min period, which was
faster than filter paper disks, gauze pads,16 and macro-
ducts.17 Moreover, use of the plastic collector reduces
contamination of the sweat specimen, maintains the
original concentration of sweat, and represents the
sweat excretion of the entire trunk. The maximum sweat
excretion was 2.5 l/hr, which comports with the find-
ings of a previous report.4

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Submitted for publication October 18, 1999; revised; accepted for
publication May 17, 2000.

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