Thirst Satiation and the Temperature of Ingested Water

Abstract. *Ingestion in rats given limited daily access to water of 12°, 24°, and 37°C is a positive function of water temperature, even though ingestion of warm water decreases blood osmotic concentration faster than cold water. The paradox suggests that temperature-dependent gastric factors and water-transport factors determine stomach distention cues of thirst satiation.*

It is commonly held that a cold drink is more thirst quenching than a warm one, although no satisfactory explanation for this has yet been reported. In animal work, this qualitative relationship is usually translated into quantities consumed—the more a water-deprived animal drinks of a particular liquid in a given time, the less satiating it is taken to be. Kapatos and Gold (*1*) found that when rats were maintained on a water-restriction schedule in which they received a daily 30-minute exposure to water of one temperature in the absence of food, they ingested less cold water than water at body temperature. Their conclusion was that cold water satiates thirst more effectively than body-temperature water, a restatement of the commonly held conception. Behavioral and physiological data which lead to a relatively simple explanation of the relationship between the temperature of ingested water and its thirst quenching ability (*2*) are presented below.

When animals are maintained on a water-rationing schedule in the continuous presence of food, the amounts of water and food ingested are related to the water's temperature. In experiment 1, ten male Holtzman albino rats (average weight, 408 g) were housed in individual cages with food tunnels containing powdered Purina Lab Chow. For 30 minutes daily each rat was allowed access to water by means of a thermally insulated bottle and drinking tube affixed to the front of its cage, and water and food intake were measured. For 6 days, the rats received room-temperature (24°C) distilled water during the access period and reached a stable baseline of water intake. Every other day for the next 11 days, the rats were given a 30-minute exposure to distilled water at one of three temperatures, 12°, 24° and 37°C (approximately body temperature), with each presented twice in counterbalanced order. On the five control days intervening between experimental days, all ten rats received 24°C water. Temperatures were determined by a thermistor probe in the drinking tubes (*3*).

The results revealed a systematic, positive relation between water ingestion and water temperature; the differences were small in magnitude but regular and significant. The mean water intake was 21.07, 22.67, and 23.55 g, for 12°, 24°, and 37°C water, respectively ($P < .05$) (*4*). The linear trend accounted for 97.3 percent of the variability between conditions and was also significant ($P < .01$). As the water intake increased, so did the food consumption during the exposure period, which averaged 4.74, 5.11, and 5.41 g across the three temperatures ($P < .05$), with the linear trend accounting for 99.6 percent of the between-condition variability ($P < .01$). The relation between food and water intake remained relatively constant across the temperature conditions. The means of the water to food ratios were 4.83, 4.58, and 4.78 for 12°, 24°, and 37°C, respectively, and an analysis of variance yielded no reliable differences. Hence rats maintained on a water-rationing schedule drank and ate less when cold water was available than they did in the presence of warm water. These results are essentially an expansion of those reported by Kapatos and Gold (*1*).

One hypothesis that explains this finding involves body temperature regulation; it is possible that rats decrease their intake of cold water to prevent excessive hypothermia. Recent evidence has shown that when a water-deprived rat drinks cold water on a schedule like that used in experiment 1, its body temperature drops by as much as 1.2°C and remains depressed for at least 20 minutes (*5*). To test this hypothesis, 12 male Holtzman albino rats (average weight, 450 g) were maintained on the same 30-minute water, 24-hour food schedule used in experiment 1, until their water intake stabilized. On day 1, the distilled water presented to half the rats was 12°C, and the other half received 37°C water for 30 minutes. Water and food intake were measured. On day 2, all animals received 24°C water; on day 3, the 12° and 37°C exposure was repeated with the subgroups unchanged. However, 3 hours before the exposure period began the rats received an intraperitoneal injection of 2.1 ml of sodium salicylate in isotonic saline at a dosage of 240 mg per kilogram of body weight, previously shown to lower the body temperature of rats (*6*). Body temperatures were measured by a rectal thermistor probe 30 minutes before water presentation, during which the groups received the same water temperature as on day 1, and water and food intake were measured. After 3 days, with 30-minute access to 24°C water, the experiment was repeated (on days 7 and 9) with the groups reversed. In addition, body temperatures were taken 30 minutes before the water exposure on day 7. All 12 rats were, therefore, exposed to both water temperatures with and without the sodium salicylate injection.

The results of experiment 2 fail to support the temperature-regulation hypothesis: lowering the body temperature before exposure decreased overall water intake but did not significantly suppress the ingestion of cold water relative to warm water, a change expected if the prevention of excessive hypothermia were the determining factor in the relationship. The mean intake was 23.7 and 29.0 g of 12° and 37°C water, respectively, on the noninjection test days, and it was 18.2 and 25.7 g under the influence of sodium salicylate. Whereas the difference in water ingestion due to temperature was significant ($P < .001$), as was the overall lowering effect of sodium salicylate ($P < .01$), there was no interaction of the two variables ($F < 1$). But the effect of salicylate on the animals' temperature was profound. Mean body temperature when no injections were given was 38.6°C, and after the injections of salicylate it was 36.2°C, a significant difference ($P < .0001$). Thus, the injections effectively lowered body temperature by an average of 2.4°C and total water intake was decreased, but no change was found in the relation between water temperature and ingestion.

Although body temperature regulation does not appear to be the mediator in the relation between water temperature and its ingestion, it is possible that systemic changes accompanying shifts in body temperature are responsible in that most chemical and biological actions are related to temperature. Water ingestion decreases plasma osmolality, the decrease leads to eating (*7*), and the latency between the start of drinking and the initiation of eating is a behavioral measurement of the speed and degree of decrease in osmolality (*8*).

If there is a difference in the passage of water of different temperatures, there should be a corresponding difference in the latencies to the onset of eating after water ingestion. Experiment 3 tested this possibility.

Eighteen Holtzman albino male rats (average weight, 205 g) were maintained on a water-rationing schedule in which they received four 4-ml portions of room-temperature water daily, presented in a small cup mounted in each animal's cage, with Purina Lab Chow available continuously. On day 1 of this experiment, half of the rats were given 12° and the other half 37°C distilled water for their first 4-ml portion of water. After the animals were observed for 20 minutes, three more rations of room-temperature water were given. Two days later the conditions were reversed. On both test days, the time between water presentation and the initiation of eating after the first water ration was recorded for each rat by an observer with a stopwatch. The results show a clear difference between temperature conditions. The mean latency to initiation of eating was 8.96 and 5.89 minutes after the 12° and 37°C water ration, respectively ($P < .02$). The fact that eating followed the ingestion of body-temperature water more rapidly than it did cool water suggests that the warmer water caused a more rapid decrease in plasma osmotic concentration. To determine whether this was the case, another experiment was conducted in which serum osmolality was measured.

In experiment 4, 36 male Holtzman albino rats (average weight, 336 g) were assigned to three groups of 12 animals each. All animals were habituated to the four-portion water-rationing schedule prior to the test day. One group (control) was killed by decapitation at the time it normally received the first water ration. The rats in the second group received two 4-ml portions of 12°C distilled water, presented sequentially so that drinking was uninterrupted, and were killed 3 minutes after they completed the second portion. The third group received two 4-ml portions of 37°C distilled water, and were likewise killed 3 minutes after they completed the second portion. Blood was taken at the time of decapitation and centrifuged, and the osmolality of each rat's serum was determined by an Advanced Instruments osmometer. The results show a marked positive relation between the temperature of ingested water and the degree of its absorption into the blood. The mean serum osmolality for the control group was 318.9 milliosmole/kg, the animals drinking 12°C water averaged 315.7 milliosmole/kg, and the group receiving 37°C water, 309.0 milliosmole/kg. The overall between-group difference was significant ($P < .01$) (*9*). Ingestion of 12°C water thus led to a considerably smaller decrease in osmolality than did the ingestion of 37°C water, even though the time for absorption was approximately the same (*10*).

These last results, taken in comparison with the differences in water intake found in experiment 1, present an apparent paradox. Hatton and Bennett (*11*) have suggested that satiation can be attributed to lowered osmolality, rather than to an anticipatory (that is, preabsorptive) satiety mechanism. However, if this were true, rats would be expected to drink more cold water, rather than less, in a 30-minute period because cold water decreases osmolality more slowly than warm water. A way out of the paradox can be found in the rate of movement of liquid from the stomach, where practically no absorption takes place, into the small intestine, where it then passes across the gut into the blood. There is evidence that stomach emptying is slower for cold drinks than for warm (*12*), and the lowered body temperature that accompanies cold-water ingestion may further slow the movement of water across the gut. Both factors explain the difference in serum osmolality found in experiment 4, which, in turn, explains the difference in latencies to the initiation of eating found in experiment 3.

That rats drink less cold water during a 30-minute exposure period also is explained by these gastrointestinal events. Fitzsimons has concluded that the inhibition of thirst in preabsorptive satiety "appears to be brought about by the distention of the stomach" (*13*); and the works of Adolph (*14*), Towbin (*15*), and others support this conclusion. The interaction of stomach-distention cues of thirst satiation and temperature-dependent stomach emptying account for the ingestion differences found in experiment 1 and by others (*1, 5*). Since gastric emptying of cold water is slow, distention occurs quickly and satiation is signaled after less is consumed; body-temperature water passes through the stomach more rapidly, thereby allowing a greater volume to be ingested before stomach distention signals the preabsorptive satiation of thirst. Thus, the difference in intake between warm and cold water is due to the corresponding difference in the rate of gastric emptying and concomitant temperature changes at the gut, and lowering overall body temperature (as with salicylate) should not affect the difference between conditions. In experiment 2 the relation between water temperature and ingestion held while overall water intake was suppressed, possibly because of slowed passage across the gut or because of a direct pharmacological effect.

The explanation that gastrointestinal events cause the common (and experimentally established) phenomenon that cold water is more thirst quenching than warm water can be extended to other aspects of drinking behavior. A hyperosmotic cold drink (such as a soda) should elicit relatively fast signals of thirst reduction, due to the slowness of its passage out of the stomach, whereas drinking the same amount of a hypoosmotic warm liquid (such as black coffee) should produce relatively insufficient feelings of satiation, even though it causes more complete rehydration at the cellular level. Subjective observations, at least, document this paradox.

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References and Notes

1. G. Kapatos and R. M. Gold, *Science* **176**, 685 (1972).
2. One should not confuse preabsorptive satiety, which is the topic of this report, with "true" (or "permanent") satiety, which apparently requires cellular rehydration. The difference is both temporal and causal, and the hypothesis presented here emphasizes that difference.
3. Yellow Springs Instrument model 402 probe and 45 telethermometer, accurate to ± 0.10°C.
4. All statistical tests, unless otherwise noted, were analyses of variance, which will be supplied on request.
5. E. Deaux and R. Engstrom, *Physiol. Psychol.* **1**, 152 (1973).
6. E. Satinoff, *Science* **176**, 532 (1972).
7. J. W. Kakolewski and E. Deaux, *Amer. J. Physiol.* **218**, 590 (1970).
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9. A separate statistical test found the difference between the two groups receiving water to be significant ($t = 2.76$; d.f. $= 22$; $P < .02$).
10. The mean duration of drinking the 12°C water slightly exceeded that for 37°C; thus, the time for absorption was a bit longer for the cold water, even though the osmotic decrease was less.
11. G. I. Hatton and C. T. Bennett, *Physiol. Behav.* **5**, 479 (1970).
12. W. C. Alvarez, *The Mechanics of the Digestive Tract* (Hoeber, New York, 1928); W. R. Hess, *Helv. Physiol. Pharmacol. Acta* (Suppl. 4) (1947).
13. J. T. Fitzsimons, *Physiol. Rev.* **52**, 468 (1972); see especially *ibid.*, p. 498.
14. E. F. Adolph, J. P. Barker, P. A. Hoy, *Amer. J. Physiol.* **178**, 538 (1954).
15. E. J. Towbin, *ibid.* **159**, 533 (1949).

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