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Factors Influencing Dicarboxylic Amino Acid Content of Human Milk

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Human milk is a major nutrient for infants throughout the world. In the United States, 50% of women breast feed their newborn infants at the time of hospital discharge. In developing countries, essentially all infants are breast fed. Glutamate is a major constituent of human milk, occurring in both the free and protein-bound forms. As such, it is a major source of nitrogen and energy for the growing infant. Free glutamate and aspartate are present in particularly large quantities in human milk.

Animal studies have raised questions about the safety of dicarboxylic amino acid ingestion by the lactating woman. Although human free plasma glutamate levels are low, milk and tissue levels are high (18). Thus, it has been questioned whether increased dietary ingestion of monosodium glutamate would elevate plasma glutamate levels, resulting in the movement of glutamate from plasma to human milk. To study this question, we gave lactating women loads of monosodium glutamate (MSG) or aspartate (as Aspartame—a dipeptide sweetener) and measured plasma and milk amino acid levels. In this chapter, we will review data previously reported on this subject (3,19) and present new information on the effects of dicarboxylic amino acid ingestion on plasma and human milk levels of glutamate and aspartate.

In 1972, we evaluated the effect of 6-g loads of MSG on plasma and human milk levels in lactating women (19). This load approximated a dose of 100 mg/kg body weight in these subjects. As shown in Table 1, the subjects studied had well-established lactation periods of 30- to 90-days duration. These subjects received the MSG load, contained in twelve 0.5-g capsules, at 0800 hr after an overnight fast. In four tests the MSG was given in conjunction with water; in nine tests the MSG was given with Slender (Carnation Products Co.), a liquid, ready-to-feed meal product; and in six tests a placebo (6 g lactose in the capsules) was given in water.

Milk samples were obtained at 0, 1, 2, 3, 4, 6, and 12 hr after administration of MSG or lactose. Blood samples were drawn at 0, 30, 60, 120, and 180 min after administration of MSG with water and at 0, 60, 90, 150, and 210 min after administration with Slender.

Mean plasma glutamate and aspartate levels in these subjects are shown in Fig. 1. Plasma glutamate and aspartate levels increased with time following administration

TABLE 1. *Schedule of participation in loading tests*

Subject	Days of lactation[a]		
	30	60	90
1	B	A	B
2	A	B	B
3	B	—	B
4	B	—	—
5	—	—	A
6	B	A	B
7	C	—	—
8	—	C	—
9	—	C	—
10	—	—	C

[a] A, MSG in water; B, MSG in Slender; C, lactose in water.

of MSG with both water and Slender. Peak plasma glutamate levels occurred earlier in subjects receiving MSG with water than in subjects receiving MSG with Slender. No change in plasma glutamate or aspartate levels was noted in subjects receiving the lactose placebo.

Figure 2 shows human milk glutamate and aspartate levels in these subjects. No significant differences between milk aspartate and glutamate levels were noted between subjects receiving MSG with water, MSG with Slender, or the lactose placebo. Concentrations of both amino acids in milk increased with time in all cases. It should be noted that the subjects were permitted to eat normally following the 4-hr milk sample.

Recently, we reviewed these older data in light of the more extensive data

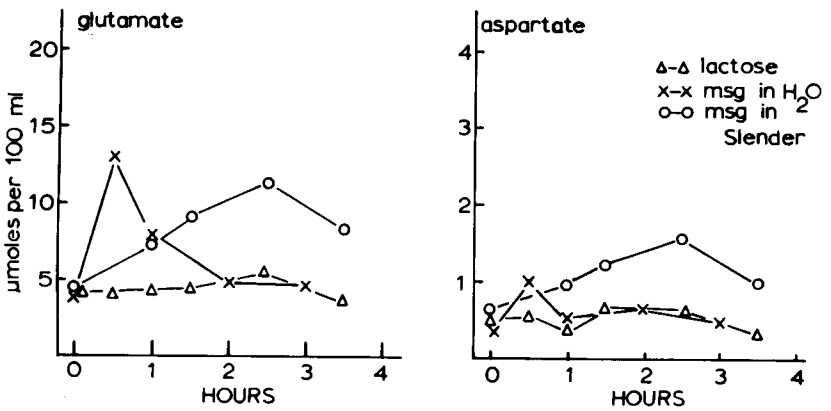

FIG. 1. Mean plasma glutamate and aspartate levels in lactating women given 6 g glutamate (MSG) in either water (×) or Slender (○), or given a lactose placebo (Δ). (From Stegink, ref. 18, with permission.)

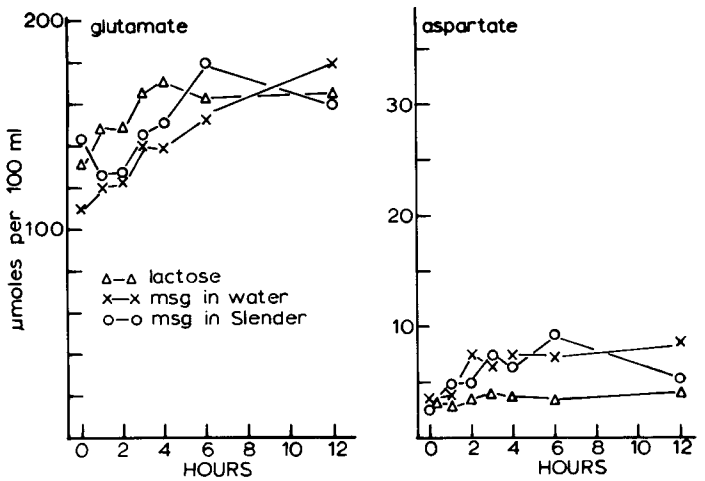

FIG. 2. Mean human milk free glutamate and aspartate levels in lactating women given 6 g glutamate (MSG) in either water (×) or Slender (○), or given a lactose placebo (Δ). (From Steginк, ref. 18, with permission.)

accumulated on the effects of glutamate loading on plasma amino acid levels (20). Two differences were apparent. First, plasma glutamate levels in subjects receiving MSG in capsules were lower than values obtained more recently (20), where an equivalent dose of MSG was dissolved in water. Second, in contrast to our earlier study (19), our recent data indicate that the addition of glutamate to a meal results in a much lower plasma glutamate level than that resulting from an equivalent dose of glutamate given in water (20).

Figure 3 shows the individual plasma glutamate levels in our original subjects receiving MSG in capsules with water. Considerable individual variation in plasma glutamate levels was noted. Plasma levels in some subjects hardly changed, whereas other subjects showed significant elevations. This variation raised a number of questions: (a) Do these differences arise because of individual variation in glutamate metabolism? (b) Do these differences reflect differing rates of capsule dissolution in the gut? (c) Were all subjects totally fasted? Some nursing notes from the metabolism unit evaluated retrospectively suggest that one or more subjects might have had toast and coffee before coming to the unit for study.

An important question to be resolved is whether our original conclusion that 6-g loads of MSG did not affect human milk glutamate levels was valid. Since two subjects given MSG in capsules with water did not show significantly increased plasma glutamate levels, we were concerned that these two subjects unduly influenced the statistical comparison of milk glutamate levels in subjects ingesting MSG with water with those receiving the lactose placebo. To evaluate this possibility, we studied two additional women with well-established lactation patterns. These two subjects received MSG dissolved in water (4.2 ml/kg body weight of a 2.4%

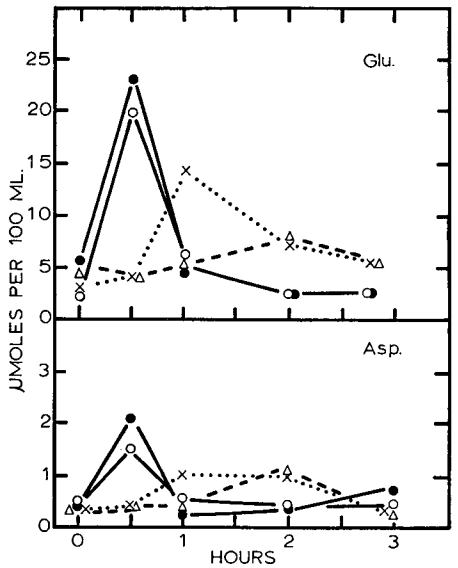

FIG. 3. Plasma glutamate (Glu) and aspartate (Asp) levels in individual subjects after administration of MSG with water. The symbols ○, ●, ×, and △ show values in individual subjects. (From Stegink et al., ref. 19, with permission.)

solution of MSG). This level provides a dose of 100 mg/kg body weight. Plasma and milk levels of free amino acids were determined as described in the original study (19).

The data in Table 2 compare plasma glutamate levels in these two subjects to values obtained in our previous study (19). Plasma glutamate values in these new subjects are significantly higher than values obtained previously (19), but are

TABLE 2. *Plasma glutamate levels in lactating women administered MSG in water at approximately 100 mg/kg body weight*

Time (min)	Current subjects[a]		Previous subjects[a,b]
	M.B.	M.M.	(mean ± SD)
0	6.5	3.2	3.9 ± 1.7
15	31.7	4.4	—
30	49.4	30.6	13.0 ± 10
45	28.6	33.1	—
60	22.6	19.7	7.5 ± 4.8
90	10.2	10.8	—
120	8.5	5.4	5.1 ± 2.7
150	6.2	3.9	—
180	7.7	5.0	4.8 ± 2.3
240	6.9	2.54	—

[a] In μmoles/dl.
[b] $N = 4$.
From Stegink et al., ref. 19.

TABLE 3. *Plasma glutamate levels in the four lactating women showing the highest plasma response to MSG ingestion in water at 100 mg/kg body weight*

Time (min)	Lactating subjects[a]				Mean ± SD for lactating subjects[a]	Mean ± SD for nonlactating subjects[a,b]
	M.S.	M.W.	M.B.	M.M.		
0	2.5	5.6	6.5	3.2	4.45 ± 1.90	2.70 ± 0.88
15	—	—	31.7	4.4	18.1 ± 19.3	14.7 ± 15.0
30	20.1	23.4	49.4	30.6	30.8 ± 13.1	47.1 ± 25.4
45	—	—	28.6	33.1	30.8 ± 3.11	50.1 ± 23.9
60	6.5	6.0	22.6	19.7	13.7 ± 8.68	24.9 ± 11.3
90	—	—	10.2	10.8	10.5 ± 0.42	8.19 ± 5.31
120	3.5	4.0	8.5	5.41	5.35 ± 2.24	4.28 ± 2.60
150	—	—	6.2	3.93	5.07 ± 1.61	3.72 ± 2.24
180	3.0	3.5	7.7	5.00	4.79 ± 2.10	4.19 ± 2.82
240	—	0	6.9	2.54	4.74 ± 3.11	3.05 ± 1.61

[a] In μmoles/dl.
[b] From Stegink et al., ref. 20.

comparable to those we have obtained in nonlactating subjects given an equivalent dose of MSG dissolved in water (20).

To determine the effect of elevated plasma levels on human milk levels, we evaluated the data obtained from the four subjects who had significant elevation of plasma glutamate levels and eliminated those two subjects showing little or no increase in plasma glutamate after loading. Table 3 lists the individual and mean plasma glutamate levels for these four subjects, along with mean values for nonlactating subjects administered an equivalent dose. Table 4 gives human milk glutamate levels in these four subjects compared with values in 10 subjects receiving the lactose placebo.

These data indicate that, although there was a significant increase in plasma glutamate levels, milk glutamate levels were not affected. The milk glutamate levels for these subjects are similar to those noted in subjects receiving the lactose placebo.

TABLE 4. *Milk glutamate levels in the four lactating subjects showing the highest plasma response to MSG ingestion at 100 mg/kg body weight*

Time after loading (hr)	Subjects[a]				Subjects given MSG[a] (mean ± SD)	Subjects given lactose (mean ± SD)[a,b]
	M.S.	M.W.	M.B.	M.M.		
0	69	158	65	117	102 ± 44	116 ± 29
1	82	145	62	108	99 ± 36	114 ± 39
2	140	150	82	78	113 ± 38	121 ± 27
3	117	155	112	129	128 ± 19	116 ± 27
4	126	165	103	112	126 ± 27	118 ± 30
12	121	166	128	82	122 ± 30	130 ± 37

[a] In μmoles/dl.
[b] From Baker et al., ref. 3; and Stegink et al., ref. 19.

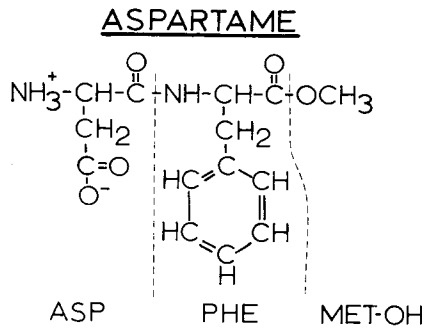

FIG. 4. Structure of Aspartame, L-aspartyl-L-phenylalanine methyl ester.

Thus, a load of MSG equivalent to two-thirds of the Acceptable Daily Intake (ADI) of glutamate in one dose produced no effect on milk glutamate levels.

These data suggest that a significant amount of glutamate in human milk is synthesized by the mammary gland. The data of Egan and Black (5) in the lactating cow indicate that most of administered ^{14}C-glutamate is converted to citrate by the mammary gland. Little lable was incorporated into milk free glutamate pools or milk protein.

Aspartate, like glutamate, is reported to exert neurotoxic effects in the neonatal rodent (13). However, in even the most sensitive animal species (the neonatal mouse), plasma aspartate and glutamate levels must be grossly elevated before neuronal necrosis is noted (23). To evaluate the possible effects of aspartate loading on human milk levels, we studied Aspartame (Searle Laboratories, Skokie, Ill.), a dipeptide containing aspartate. Aspartame is L-aspartyl-L-phenylalanyl-methyl ester (Fig. 4) and is 180 to 200 times sweeter than sucrose. It is hydrolyzed in the intestinal mucosa to its component amino acids and methanol. The latter are absorbed, metabolized, and cleared in a manner similar to that of aspartate, phenylalanine, and methanol arising from dietary protein and methylated polysaccarides.

Six healthy women with well-established lactation patterns were studied (3). The subjects were administered either Aspartame or a lactose load at a level of 50 mg/kg body weight. The order of administration was randomized in a crossover design. Aspartame or lactose was dissolved in 300 ml cold orange juice and administered to the subjects at 0800 hr after an overnight fast. Subjects were fasted for an additional 4 hr after administration of the test materials, but were allowed a normal diet after this time. Plasma and milk samples were collected and analyzed as described previously (19).

Table 5 shows plasma aspartate levels after Aspartame and lactose loading. No significant effect of either Aspartame or lactose loading was noted on plasma aspartate levels. Plasma phenylalanine levels increased significantly after Aspartame loading, reaching a mean peak value of 16.2 $\pm$ 4.9 μmoles/dl, but were not affected by lactose. The peak level of phenylalanine noted was only slightly higher than that observed postprandially in formula-fed infants (22).

TABLE 5. *Plasma aspartate and phenylalanine levels in lactating women given Aspartame or lactose at 50 mg/kg body weight*

Time (min)	Aspartate[a]		Phenylalanine[a]	
	Aspartame	Lactose	Aspartame	Lactose
0	0.42 ± 0.33	0.32 ± 0.20	4.61 ± 1.72	5.04 ± 1.13
15	0.42 ± 0.24	0.28 ± 0.07	8.34 ± 2.72	4.47 ± 0.81
30	0.54 ± 0.55	0.24 ± 0.09	14.5 ± 4.47	4.99 ± 0.98
45	0.31 ± 0.15	0.23 ± 0.05	16.2 ± 4.86	4.23 ± 0.95
60	0.34 ± 0.19	0.40 ± 0.40	14.2 ± 4.08	4.45 ± 0.55
90	0.30 ± 0.17	0.24 ± 0.14	15.7 ± 6.19	4.33 ± 0.83
120	0.30 ± 0.15	0.17 ± 0.05	12.8 ± 3.75	4.38 ± 0.93
180	0.17 ± 0.05	0.22 ± 0.03	8.07 ± 2.25	4.62 ± 0.86
240	0.23 ± 0.12	0.21 ± 0.03	6.42 ± 2.02	4.98 ± 1.18

[a] In μmoles/dl (mean ± SD).

Human milk levels in these subjects are shown in Table 6. Small, but statistically insignificant, increases in phenylalanine, aspartate, and glutamate levels were noted after Aspartame administration when compared to the same subjects after lactose loading. During the 4-hr fasting period after Aspartame loading, milk phenylalanine levels increased from 0.5 μmoles/dl to 2.3 μmoles/dl, whereas human milk aspartate levels increased from 2.3 μmoles/dl to about 4.8 μmoles/dl. The levels of these amino acids in milk samples collected, after the evening meal, 12 hr after the loading dose, were similar to those noted in milk samples collected 3 to 4 hr after loading.

EFFECT OF LENGTH OF LACTATION

Human milk glutamate levels vary considerably from individual to individual. We have evaluated the possibility that the duration of lactation might effect free amino acid levels. Free amino acid levels were measured in 225 milk samples obtained from 45 healthy women who had been lactating from 2 to 163 days. The effect of the duration of lactation on milk glutamate, aspartate, and taurine levels is shown in Table 7. Glutamate and aspartate levels were lower in colostrum (2 to 10 days lactation) than in mature milk. Glutamate levels were 47 μmoles/dl in colostrum and rose to a mean level of 128 μmoles/dl later in lactation. Similarly, aspartate levels increased from 2.7 to 5.2 μmoles/dl. In contrast, taurine levels decreased from 50 μmoles/dl in colostrum to 34 μmoles/dl in mature milk.

QUANTITY OF GLUTAMATE INGESTED BY THE TERM INFANT

These figures allow us to estimate the quantity of free and protein-bound glutamate and aspartate ingested by the normal 3.5-kg breast-fed term infant. As shown in Table 8, infants fed *ad libitum* with breast milk ingest a mean of 171 ml

TABLE 6. *Milk glutamate, aspartate, and phenylalanine levels in six lactating women given either Aspartame or lactose at 50 mg/kg body weight*

Time (hr)	Aspartate[a]		Glutamate[a]		Phenylalanine[a]	
	Aspartame	Lactose	Aspartame	Lactose	Aspartame	Lactose
0	2.25 ± 1.16	2.62 ± 0.82	109 ± 14	104 ± 24	0.48 ± 0.27	0.80 ± 0.35
1	2.82 ± 0.94	3.10 ± 0.54	106 ± 19	93 ± 31	2.06 ± 1.11	0.89 ± 0.39
2	4.67 ± 1.49	3.61 ± 0.85	122 ± 20	105 ± 22	2.29 ± 1.07	0.87 ± 0.32
3	4.53 ± 2.23	3.90 ± 1.46	128 ± 23	107 ± 32	2.08 ± 0.94	1.40 ± 1.85
4	4.82 ± 1.68	3.76 ± 1.84	120 ± 18	104 ± 29	1.99 ± 0.88	0.90 ± 0.53
12	5.59 ± 3.22	4.11 ± 2.50	155 ± 25	111 ± 23	1.19 ± 0.59	0.93 ± 0.59

[a] In μmoles/dl (mean ± SD).

TABLE 7. *Variation in human milk glutamate, aspartate, and taurine levels with duration of lactation*

Samples (N)	Days lactation		Subjects (N)	Taurine[a]	Glutamate[a]	Aspartate[a]
	Range	Mean				
16	2–10	6	13	50 ± 14	47 ± 34	2.7 ± 2
64	12–48	30	12	35 ± 11	138 ± 47	5.1 ± 3
36	49–69	56	5	35 ± 14	112 ± 36	4.4 ± 3
47	70–90	80	7	35 ± 16	144 ± 45	7.8 ± 13
62	> 90	110	8	29 ± 8	116 ± 28	3.7 ± 2

[a] In μmoles/dl (mean ± SD).

milk/kg/day (7). This level provides approximately 115 kcal/kg body weight. The free glutamate intake of this breast-fed infant is about 36 mg/kg/day, whereas the intake of protein-bound glutamate approximates 357 mg/kg/day. The breast-fed infant thus ingests a quantity of free glutamate equivalent to 46 mg/kg of MSG per day (36 mg × 1.28 to correct for the sodium ion and water of hydration in MSG).

The data in Table 9 show the expected daily per capita intake of MSG in the United States as estimated by the Committee on GRAS List Survey—Phase III (4). These data indicate that infants 12 to 23 months of age have the highest MSG intake of all groups, with a mean expected intake of 6.8 mg/kg/day. Since our calculations indicate that the breast-fed infant ingests 46 mg MSG/kg/day, such infants are at a level equivalent to the 99th percentile of expected daily intake.

TABLE 8. *Estimated glutamate and aspartate intake in the 3.5-kg breast-fed infant*

Variable	Mean	Range
Mean milk intake[a]	171 ml/kg/day	114–228
Free amino acid content[b]		
Glutamate	138 μmoles/dl (21 mg%)	
Aspartate	5 μmoles/dl (0.67 mg%)	
Total glutamate + aspartate[c]		
Glutamate (free and protein bound)	230 mg%	
Aspartate	116 mg%	
Total daily intake		
Free glutamate	36 mg/kg	24–48 mg/kg
Protein-bound glutamate	357	237–476
Total glutamate	393	262–524
Free aspartate	1.2 mg/kg	0.8–1.5 mg/kg
Protein-bound aspartate	197	131–262
Total aspartate	198	132–264

[a] From Fomon, ref. 7.
[b] See Table 7.
[c] Data from Macey et al., ref. 10 and Svanberg et al., ref. 24.

TABLE 9. *Expected daily per capita intake of MSG*[a]

Age	Mean	Total sample Intakes (mg/kg/day) Percentile 90th	99th	99.9th
0–5 months	0.3	0	11	25
6–11 months	1.9	1.9	36	46
12–23 months	6.8	30	43	61
2–5 years	5.5	23	37	56
6–17 years	2.7	10	25	40
18+ years	1.5	7	12	19

[a] From Committee on GRAS List Survey—Phase III, ref. 4.

SPECIES VARIATION

It has been noted that the neonatal rodent is markedly sensitive to dicarboxylic amino acid-induced neuronal necrosis (9,12,18). The neonatal nonhuman primate does not appear to be sensitive to these amino acids, even at high ingestion levels (1,2,11,17,21,25), although this is a controversial finding (14,15). Table 10 compares milk glutamate and aspartate levels in a variety of animal species (8,16,19). These data indicate considerably higher glutamate levels in milk from human and nonhuman primates than in rodent milk. However, even the neonatal rodent ingests glutamate from its mother's milk. This glutamate must be rapidly metabolized, since it has no known effect on their growth and development. Similarly, data to be presented later in this symposium (6) indicate that the human infant rapidly metabolized the glutamate present in human milk.

SUMMARY

1. We have shown that the amino acid content of human milk varies with the duration of lactation. Levels of amino acids, such as glutamate and aspartate, increase with time, whereas others, such as taurine, decrease with time.

2. The breast-fed human in the U.S. ingests more glutamate on a kg per body weight basis, than at any other time during life.

3. Ingestion of MSG at 100 mg/kg increases the plasma levels of the lactating human but not milk levels.

4. Ingestion of MSG at 100 mg/kg body weight by lactating women has little or no effect on the glutamate intake of their infants.

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TABLE 10. *Milk glutamate and aspartate levels in various species*[a]

Species	Rassin et al. (16)		Iowa		Ghadimi and Pecora (8)	
	Aspartate	Glutamate	Aspartate	Glutamate	Aspartate	Glutamate
Mouse	0.9	5.7				
Rat						
Colostrum	7.3 ± 1.2	15.1 ± 3.5	—	—	—	—
Mature	11.1 ± 1.2	11.1 ± 0.9	—	—	—	—
Guinea pig	1.6 ± 1.5	15.1 ± 4.1	—	—	—	—
Dog	0.2 ± 0.1	6.6 ± 0.7	—	—	—	—
Cat	trace	17.7 ± 9.4	—	—	—	—
Cow						
Fresh	1.4 ± 0.2	6.8 ± 1.2	—	—	0.3	4.8
Middle	1.8 ± 0.1	12.8 ± 2.4			0.5	3.8
Rhesus monkey	5.8 ± 2.2	31.4 ± 7.0	—	—	—	—
Baboon	8.1 ± 1.3	43.9 ± 5.6	—	—	—	—
Chimpanzee	18.5 ± 0.5	264 ± 7.5	—	—	—	—
Human						
Colostrum	5.1 ± 3.0	68.4 ± 12.9	2.7 ± 2.0	47 ± 34	22	76
Mature	4.2 ± 0.5	127 ± 8	5.2 ± 2.1	128 ± 20	7	42

[a] In μmoles/dl (mean ± SD), unless only one value available.

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