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Responses of Urine pH, Blood pH, Blood Lactate Concentration and Anaerobic Endurance to Sodium Bicarbonate, Sodium Citrate and Glutamate Administration

Sung-Gye Cho, Young-Su Kim, Tae-Hyung Kim, Dong-Ju Yoon
(Korea Sport Science Institute)
Jun Kim, Sang-Chul Park (Medical College, Seoul National University)

Six healthy male subjects performed a treadmill running test to exhaustion at 95% $\dot{V}O_{2max}$. The study was carried out after oral administration of a current sports beverage (P: placebo), glutamate (G=5g), $NaHCO_3$(S: sodium bicarbonate=0.3g/kg body weight) and Na_3citrate (C: sodium citrate=0.3g/kg body weight).

Arterialized-venous blood samples were drawn at specific times(pre-treatment, pre-exercise=90min before treadmill running, recovery 3min, recovery 15 min) and analyzed for pH and HLa (blood lactate concentration). Also, urine samples were taken three times(pre-treatment, pre-exercise, recovery 15 min) for pH.

The results were as follows:

(1) Urine pH at pre-exercise was highest in C and lowest in P (C>S>G>P; $p<.001$).
(2) In blood pH, the highest was S and the lowest C at the pre-exercise (S>C>G>P; $p<.001$). The rates of recovery at recovery 15 min tended to be higher in S and C than in G and P.
(3) Accumulation of blood lactate at recovery 3 min was highest in S and lowest in G, even though there was no statistically significant difference.
(4) There was no statistically significant difference in anaerobic endurance under the 4 conditions. However, the exercise times to exhaustion in S, C and G were delayed 14.5%, 18.7%, and 6.7% compared to P respectively.

From these results, we could suggest that ingestion of $NaHCO_3$ and Na_3citrate prior to exercise induces alkalosis and may affect improvement of anaerobic endurance, and may increase the rate of recovery from acidosis after exhaustion following high intensity exercise. Also, ingestion of glutamate may enhance anaerobic endurance, but may cause acidosis during recovery after exercise.

INTRODUCTION

The fatigue(inhibition of performance) is directly attributable to increased intra-and extracellular lactate and hydrogen ion concentration $[H^+]$(acidosis) which occur during short term high intensity exercise in

which energy is supplied mainly by the process of anaerobic glycolysis and oxidative phosphorylation (Hermansen, 1981: Shalin, 1983, 1986).

Lactate concentration facilitates the decrease of muscle pH (Wasserman et al, 1973), isotonic muscle contraction (Fitts and Holloszy, 1976), myosin APT_{ase} activation (Shadler, 1967), creatine phosphate (Shalin et al, 1981) and Ca^{++} in sarcoplasmic reticulum (Nakamura and Schwartz, 1972). Increased $[H^+]$(or decreased pH) inhibits muscle contractibility by decreasing the activation of PFK (phosphofructokinase), which is the chief enzyme of anaerobic glycolysis (Danforth, 1965: Sutton et al, 1981) and also by inhibiting the combination of $Ca^{++}$and troponin and its efflux from the sarcoplasmic reticulum (Donaldson and Hermansen, 1978; Fabiato and Fabiato, 1978; Vøllestad and Séjersted, 1988).

In 400mH-1,500m track events, 100m-400m swim events, most of the track events in cycling, short and middle distance races in rowing and canoe that require 30sec to 6−7min, various ball games and long distance endurance events that require more than 10min, winning is determined when the athlete overcomes fatigue factors while in tense energy generating during the later half and toward the end of the competition.

Recently, sport scientists have been studying how to overcome fatigue factors with deep interest and have reported a considerable number of results. The study of the ergogenic effect of $NaHCO_3$ and Na_3citrate on performance is a good example.

According to Sung-Gye Cho et al(1990), Bouissou et al(1988), Costill et al(1980), Kowalchuk et al(1984), Mckenzie et al(1980), Rupp et al(1984), Sutton et al(1981), and Wilkes et al(1983), an administration of $NaHCO_3$ prior to exercise could induce alkalosis and increase athletic performance at high intensity beyond 95%$\dot{V}O_{2max}$ in a state of alkalosis and improve records in events which require 40sec to 7min. However, some researchers reported they observed no improvement in athletic performance and records(Brien and Mckenzie, 1989; Horswill et al, 1988; Katz et al, 1984; Kinderman et al, 1977; Linderman et al, 1988; MaCartney et al, 1983; Magaria et al, 1971; Parry-Billings and MacLaren, 1986; Poulous et al, 1974; Simmons and Hardt, 1973; Wijnen et al, 1984), thus failing to reach a conclusion. However, the ergogenic effect of $NaHCO_3$ is gradually being received affirmatively based on alkalosis mechanism(Linderman and Fahey, 1991).

In connection with the control of acid-base balance during exercise, studies of sodium citrate have been performed. They start on the principle that the increase of intracellular citrate, particularly mitochondria, inhibits PFK, which is a control enzyme of anaerobic glycolysis, and decreases lactate

production by slowing the metabolism(Leninger, 1981).

While Hewitt and Calloway (1936) and Denning (1937) reported that citrate ingestion improved records in 100m-400m swimming, long distance running and cycling, Johnson and Black(1953), Kowlchuk et al(1989), Parry-Billings and MacLaren (1986) reported that citrate ingestion showed no significant effects on high and maximal intensity performance.

Another area of study attempted to inhibit lactate production and acidosis aims at controlling the production and removal of lactate by admino acid ingestion, particularly glutamate, which is connected to anaerobic glycolysis, in order to elevate performance.

Sang-Chul Park et al(1991) reported that they could inhibit lactate concentration in tissues and improve performance in middle and long distance runners and cyclists by administrating glutamate to them.

Though efforts have been continuing to control acid-base balance artificially during exercise and to enhance performance by delaying the onset of fatigue temporarily , those efforts have reached no conclusion and the question still remains controversial.

This study concentrated on the observation of changes in urine and blood pH, blood lactate concentration, and anaerobic endurance following sodium bicarbanate, sodium citrate and glutamate ingestion by the subjects in order to ascertain the ergogenic effects(acid-base balance, performance and the recovery from fatigue)of alkalinizers and amino acid.

METHODS

1. Subjects

The subjects were 6 male students from the Physical Education Department, S and H university. On the average, they were 20.8 years old, 177cm tall, weighed 67.3kg, and $\dot{V}O_{2max}$ was 63.8ml/kg·min^{-1} as shown in Table 1.

Table 1. Physical characteristics of subjects

(unit=mean±SD)

Subject(n)	Age(yr)	Height(cm)	Weight(kg)	$\dot{V}O_{2max}$(ml/kg·min^{-1})
6	20.8±1.2	177±6.9	67.3±6.2	63.8±10.3

2. Protocol

The measurement variables in this study were urine pH, blood pH, blood lactate concentration at pre-, middle-, and post-treatment (administration of test beverage) and the treadmill running time at 95%$\dot{V}O_{2max}$ The orders of the treatment and experiment were determined by a randomized double blind fashion.

3. Methods and Procedure

(1) Treatment

The materials used in the treatment were sodium bicarbonate($NaHCO_3$: S), sodium citrate (Na_3citrate: C), glutamate(G), and ordinary sports beverage (Gatorade: P).

Sodium bicarbonate and sodium citrate were administered in a solution mixed with 500ml of Gatorade and 500ml of pure Gatorade was used as a placebo, Glutamate was given in a gelatine capsule with 500ml of Gatorade.

One dose of sodium bicarbonate and sodium citrate was in a ratio of 300mg/kg bodyweight and that of glutamate was 5g, the same amount as Sang-Chul Park et al (1991) administered; thses were administered 90min before the treadmill running at 95%$\dot{V}O_{2max}$ and 5min was required for the treatment.

(2) Measurement

In the preliminary test performed prior to the main experiment, physiological condition and individual exercise intensity for anaerobic endurance test were examined. Maximal oxygen uptake of the subjects was also measured using the gradual exercise test of the Korea Sport Science Institute(KSSI).

The study's protocol was as shown in Fig. 1.

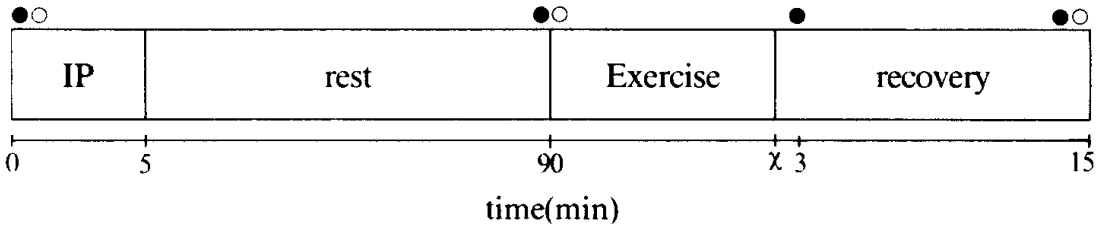

Figure. 1. Schematic illustration of study's protocol.
IP: ingesting period
Exercise: continuous treadmill running for anaerobic endurance
χ: all-out(exhaustion)
●: blood sampling for blood pH & HLa
○: urine sampling for urine pH

For the speed adjustment of the treadmill and the subjects' adaptation in the treadmill running test given 90min after the treatment, the subjects walked fast or jogged at 90-110m/min for 30 sec, then jogged or ran slow at 130-150m/min for 30sec, then finally ran to exhaustion at 95%$\dot{V}O_{2max}$ and the running time to exhaustion was set as the anaerobic endurance variable. Urine was sampled 3 times for urine pH analysis: at pre-treatment (90min before anaerobic endurance test), 90min after the treatment (right before anaerobic endurance test), recovery 15 min, and the pH level was analyzed by using the pH-meter (Metrohm 691: Swiss).

Blood pH and Blood lactate(HLa) were analyzed with a automated blood gas analyzer(AVL 945: USA) and an automated lactate analyzer (YSI L23: USA). Throughout the experiment blood samples were taken 4 times: pre-treatment, 90min after treatment, recovery 3min and 15min; 60ml was taken by using fingertip method.

4. Data Analysis

A one-way ANOVA with repeated mesures was used to test the results obtained from processing the data related to the variables in measurement. When the differences were found to be significant, Newman-Keul's multiple range test was applied to the post-hoc analysis to examine the differences among the variables and the level of statistical significance(α) was set at 5%.

RESULTS

1. Urine pH

Table 2 shows the mean urine pH and the results examined by testing time: pre-, middle-, and post-treatment by placebo (P), glutamate (G), sodium bicarbonate (S), and sodium citrate (C). The difference in urine pH at the pre-treatment was not significant, but the difference which occurred before the anaerobic endurance test (90min after the treatment) was statistically significant at 0.1%. The difference by C and S were greater than that by P. Particularly, the increases by C and S were 20% (pH 1.33) and 15%(pH 1.02) respectively while the increases by P and G were almost the same as those before the treatment.

At recovery 15min following the anaerobic endurance test, the increase in urine pH by P, G, S, and C was at 0.1%, statistically significant ($p<.001$).

The increase by G was higher than that by P; the increases by S and C were still higher than that by G; the decreases by P, G, S, and C were 13%(pH 0.89), 14% (pH 1.04), 7%(pH 0.56), and 9%(pH 0.70) respectively.

Table 2. Urine pH at pre-treatment, pre-exercise and recovery 15 min following treadmill running to exhaustion at 95%$\dot{V}O_{2max}$ in the 4 experimental conditions.

Item	P[a]	G[b]	S[c]	C[d]
pre-treatment	6.65(±0.70)	7.04(±1.20)	7.68(±0.65)	6.53(±1.90)
pre-exercise	6.67(±0.44)	7.33(±0.56)***	7.80(±0.16)***	7.86(±0.20)***
recovery 15 min	5.78(±0.54)	6.29(±0.57)	7.24(±0.18)***	7.16(±0.21)***

· P[a]: placebo · G[b]: glutamate · S[c]: sodium bicarbonate · C[d]: sodium citrate
***denotes significant difference ($p<.001$) among 4 conditions in each test
(——) underline denotes results of post hoc analysis
· Values are mean(±SD)

2. Blood pH

Table 3 shows the blood pH at pre-, middle-, and post-treatment and the differences by measurement time among P, G, S, and C. Blood pH was at almost the same level at rest after the treatment as the urine pH was. At rest 90min after the treatment, the blood pH was at 0.1%, statistically significant difference ($p<.001$). S showed a greater difference than P, G, and C; C was greater than P; G and C were at the same level. Compared with the level at rest before the treatment, P and G showed little change and pH by S and C increased 0.11 and 0.05 respectively.

Table 3. Blood pH at pre-treatment, pre-exercise and recovery following treadmill running to exhaustion at 95%$\dot{V}O_{2max}$ in the 4 experimental conditions.

Item	P[a]	G[b]	S[c]	C[d]
pre-treatment	7.41(±0.02)	7.43(±0.04)	7.38(±0.03)	7.40(±0.02)
pre-exercise	7.41(±0.03)	7.42(±0.02)***	7.49(±0.04)***	7.45(±0.20)***
recovery 3 min	7.15(±0.05)	7.19(±0.10)	7.20(±0.08)	7.19(±0.05)
recovery 15 min	7.28(±0.06)	7.28(±0.02)	7.32(±0.07)	7.34(±0.04)

***denotes significant difference ($p<.001$) among 4 conditions in each test
(——) underline denotes results of post hoc analysis
· Values are mean(±SD)

Blood pH at recovery 3min after the anaerobic endurance test was almost equal among the test conditions. The pH levels by P, G, S, and C were drastically lowered to pH 0.23-pH 0.29, compared with the level right before the all-out exercise.

3. HLa

It was shown that the differences in HLa examined by testing time under the test conditions were not statistically significant(Table 4).

HLa increases by P, G, S, and C at recovery 3min after the all-out exercise were 11.3mM, 10.6mM, 13.3mM, and 11.7mM respectively. HLa decreases (recovery rates) by P, G, S, and C were 5.62mM(45.4%), 4.24mM(35.9%), 5.58mM(38.4%), 5.6mM(42.8%) respectively at recovery 15min against recovery 3min.

Table 4. HLa at pre-treatment, pre-exercise and recovery following treadmill running to exhaustion at 95%$\dot{V}O_{2max}$ in the 4 experimental conditions.

(unit=mM)

Item	P[a]	G[b]	S[c]	C[d]
pre-treatment	1.48(±0.31)	1.30(±0.32)	1.45(±0.30)	1.47(±0.70)
pre-exercise	1.60(±0.44)	1.20(±0.24)	1.23(±0.45)	1.42(±0.43)
recovery 3 min	12.37(±3.60)	12.82(±3.15)	14.53(±4.15)	13.08(±2.25)
recovery 15 min	6.75(±1.90)	7.58(±2.16)	8.95(±3.37)	7.48(±1.90)

· Values are mean(±SD)

4. Anaerobic Endurance

Anaerobic endurance was measured by the treadmill running time to an all-out at 95%$\dot{V}O_{2max}$ at 90min after the treatment as shown in Table 5.

Table 5. Treadmill running time to exhaustion at 95%$\dot{V}O_{2max}$ following ingestion of placebo(P), glutamate(G), sodium bicarbonate(S) and sodium citrate(C)

(unit=second)

Subject	P	G	S	C
K1	420(7:00)*	440(7:20)	420(7:00)	413(6:53)
K2	438(7:18)	484(8:04)	450(7:30)	593(9:53)
K3	480(8:00)	484(8:04)	600(10:04)	545(9:05)
K4	372(6:12)	379(6:19)	509(8:29)	496(8:16)
K5	398(6:38)	468(7:48)	479(7:59)	512(8:32)
K6	300(5:00)	314(5:14)	300(5:00)	300(5:00)
mean	401.3(6:41.3)	428.2(7:08.2)	459.7(7:39.7)	476.5(7:56.5)
SD	61.7(1:01.7)	68.4(1:08.4)	99.7(1:39.7)	104.9(1:44.9)

( )* denotes (min: sec)

Though the mean difference under 4 conditions were not statistically different($p>.05$), in comparison with P(6min 41.3sec), G, S, C were found improved 26.9sec (6.7%), 58.4sec(14.5%), and 1 min 15.2sec(18.7%) respectively. The changes in each subject's exercise duration were observed based on P. G showed a slight increase (4 sec-1 min 10sec) in all of the 6 subjects, S increased 4sec-2 min in 4 out of 6 subjects and 2 showed no change, and C increased 0.5sec-2 min 35sec in 4 subjects; one showed no change; another decreased a 1.7%(7sec).

DISCUSSION

Changes in urine pH were observed by administering sodium bicarbonate, sodium citrate, glutamate, and placebo; significant differences were found under different conditions 90min after the treatment (immediately before all-out exercise). This showed acid-base balance has different effects. 15% increase by $NaHCO_3$ and Na_3citrate indicated that these two substances could induce alkalosis with a dose of 0.3g/kg bodyweight. The kidney's control of acid-base balance was also observed.

Significant changes resulted from different test conditions at recovery 15min after all-out exercise. The four test conditions caused a considerable decrease in urine pH. The decrease of urine pH was 4-6% less than the 13% decrease by placebo, from which it is considered that these basic substances were continually absorbed by the kidney.

In the responses of blood pH to the four test conditions, 90 min after the treatment revealed that $NaHCO_3$ and Na_3citrate clearly increased blood pH by 0.11 and 0.05 respectively while placebo and glutamate caused no change. This is generally in agreement with Parry-Billings and MacLaren (1986). Blood pH changed little following the treatments at 3 min after all-out exercise, but considerable amount of acidosis might have been caused by increase of $[H^+]$ which is usually accompanied with [lactate] and [pyruvate] when anaerobic glycolysis increases in the process of energy required by heavy exercise.

No difference was observed under the test conditions at recovery 15min, but blood pH, when administered with $NaHCO_3$ and Na_3citrate, was closer to the level at rest than placebo and glutamate. This indicated fast recovery of blood pH from acidosis and a continuous influx of alkali-agents of $NaHCO_3$ and Na_3citrate into the blood.

The mechanism of effects of $NaHCO_3$ and Na_3citrate on urine and blood pH, which has been discussed so far, can be explained as follows. After the ingestion of a dose of $NaCHO_3$ splits into $[Na^+]$ and $[HCO_3^-]$ ion in the body as time passes, and, as the blood and urine $[HCO_3^-]$concentration is increased by the continuing influx of $[HCO_3^-]$, alkalosis ($H^+ + HCO_3^- \rightarrow H_2Co_3 \rightarrow H_2O + CO_2$) is induced by the neutralized hydrogen ion $[H^+]$ which results from anaerobic glycolysis. Therefore it is considered that the blood pH can recover its level at rest before the recovery 15min after all-out exercise.

The ingested Na_3citrate is no longer a compound in the body, but separate forms of ion $[Na^{3+}]$ and $[citrate^{3-}]$, citrate, which is transported in negative ion, can be easily transmitted across cell membranes.

If the blood loses its acid-base balance from the influx of $[citrate^{3-}]$into the cells, the ions also lose balance and positive ions increase. In order to restore the balance negative ions increase and positive ions decreases. The positive ion, which is a hydrogen ion$[H^+]$, decreases when combined with protein or hemoglobin. Thus the bicarbonate ion $[HCO^{3-}]$ balanced with the hydrogen ion enhances relatively. Consequently, the decrease of $[H^+]$ and the increase of $[HCO_3^-]$induce alkalosis (Stewart, 1983). That this process continues even after the all-out exercise may indicate the blood pH can quickly recover its level at rest.

The changes in the blood pH induced by glutamate showed that 90min after the administration of glutamate was nearly equal to the level at rest before the treatment. This may indicate that glutamate and the abovementioned alkali-agents affects blood pH differently. The decrease of blood pH at 3min after the all-out exercise was the smallest, which may suggest that acidosis was decreased by the high intensity exercise. The decrease might have resulted from the increased activation of transamination reaction of pyruvate to alanine which was induced by glutamate ingestion (Sang-Chul Park et al, 1991). The recovery rate of the blood pH to resting level was lower that the levels by $NaHCO_3$ and Na_3citrate and even lower than the level by placebo. This may indicate that the results might have been caused by acidosis induced by the metabolism of amino-radicle in glutamate, which is an amino acid.

The blood lactate concentration [HLa] was analyzed at 90min after the treatment, recovery 3min and recovery 15min after the all-out exercise. The HLa level at recovery 3min, which is usually highest, showed no significant difference. This agreed with Parry-Billings and MacLaren (1986). However, it was higher in 4 out of 6 subjects when $NaHCO_3$ was administered than

the placebo was. The level was lower in 2 subjects, but was generally higher by 2mM than ordinary, which showed a similar trend to the report by Inbar et al (1983), Katz et al (1984) and Sutton et al (1981). Na_3citrate and placebo showed a similar level, which agreed with the findings by Kowalchuk et al (1989). The HLa increase induced by glutamate was a little lower than that induced by placebo.

The differences in HLa at recovery 15min under the four test conditions were not statistically significant, and the HLa removals (recovery rates) at recovery 15min against the HLa levels at recovery 3min were in the following order(high to low): placebo, Na_3citrate, $NaHCO_3$, and glutamate. Particularly, the HLa removal by glutamate was 10% lower than that by placebo.

The results reveal that $NaHCO_3$, as Kowalchuk et al(1989) reported, stimulates lactate production in high intensity exercise by buffering lactate in the blood. Na_3citrate seems to have nothing to do with the stimulation of lactate production. It rather seems to be induced by citrate which decreases PFK activation in the anaerobic glycolysis in the cells. It is considered that these alkalinizers do not affect HLa removal at recovery 15min. Though it is partially recognizable that glutamate, which inhibits lactate production during exercise, facilitate transamination reaction, it is not predictable that HLa is removed by the activation of transamination reaction at recovery 15min.

The anaerobic endurance was analyzed by treadmill running time at 95%$\dot{V}O_{2max}$ 90min after the ingestion of the test beverages (Table 5). The exercise times after the ingestion of $NaHCO_3$, Na_3citrate, and glutamate improved 14.5%, 18.7%, 6.7% respectively. The mean exercise times by individual subject improved in 14 out of 18 cases with no change in 3 and a decline in 1. This shows a general improvement, but the differences among the four test conditions were not statistically significant, which may indicate that the number of the subject was too limited and standard deviation in subjects' performance was too great.

The results of the analysis of the anaerobic endurance are similar to the findings by the researchers who do not recognize the ergogenic effect of $NaHCO_3$ and Na_3citrate(Brien and Mckenzie, 1989: Horswill et al, 1988: Kinderman et al, 1977: Linderman et al, 1988: MaCartney et al, 1983: and Kowalchuk et al, 1989). However, the changes in individual subject's performance agree with those who recognize the effects of alkalosis(Sang-Chul Park et al, 1989: Sung-Gye Cho et al, 1990: Bouissou et al, 1988; Costill et al, 1984, Mckenzie et al, 1986; Rupp et al, 1984: and Sutton et al, 1981). As far as the improvement of performance and its actual cases are concerned,

this study has done the most.

It is a known fact that 1-2% difference in the athlete's performance can bring significant results in athletic events which require anaerobic endurance. Therefore it should not be disregarded that judgements solely based on statistical figures could lead to serious errors. As a result, it is considered 300mg $NaHCO_3$ ingestion per 1kg bodyweight or Na_3citrate at 90min before the exercise can result in a positive imrovement of anaerobic endurance and 5g glutamate ingestion can bring a limited improvement of anaerobic endurance. This can be explained by physiological mechanism.

$NaHCO_3$ loading enhances extracellular $[HCO_3^-]$ reservoir by which the greater pH gradient inside and outside the cell membranes stimulate the muscle cells to induce a efflux of $[H^+]$and $[La^-]$. This delays acidosis, that is, the time is delayed during which the intracellular pH can lower to the critical inhibitory level (Hirche et al, 1972; Jones et al, 1977; Mainwood and Worsley-Brown, 1975; Sutton et al, 1981). Delaying the onset of fatigue can improve anaerobic exercise endurance (Goldfinch et al, 1988: Hermansen, 1981; Sahlin, 1983, 1986; Wilkes et al, 1983).

Though Na_3citrate loading slows anaerobic glycolysis by inhibiting PFK activation, the increased blood $[citrate^{3-}]$ is transmitted across the cell membranes, reduces $[H^+]$ by combining $[H^+]$ to protein and hemoglobin, induces alkalosis by enhancing $[HCO_3^-]$ concentration, and prevents metabolic acidosis induced by lactate concentration. Therefore the onset of fatigue can be delayed and highly intense short term performance is generated (Kawalchuk et al, 1989; Leninger, 1981; Parry-Billings and McLaren, 1986; Stewart, 1981).

Glutamate loading enhances the transamination reaction substrate in the tissues and accelerates the change of pyruvate to alanine, inhibiting lactate concentration in the tissues. Thus fatigue can be reduced and performance is enhanced (Sang-Chul Park et al, 1991).

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