Brillouet, J.-M.; Treche, S.; Sealy, L. *J. Food Sci.* **1981**, *46*, 1964–1967.
Collinge, S. K.; Randall, S. G; Mahoney, A. W. *J. Food Biochem.* **1980**, *4*, 111–117.
Dovell, C. J.; Harris, N. D. *J. Sci. Food Agric.* **1982**, *33*, 185–193.
Jeltema, M. A.; Zabik, M. E. *J. Sci. Food Agric.* **1980**, *31*, 820–829.
Kawabata, A. *Mem. Tokyo Univ. Agric.* **1977**, *19*, 155–200.
Kayisu, K.; Hood, L. F.; Van Soest, P. J. *J. Food Sci.* **1981**, *46*, 1885–1890.
Lund, E. D.; Smoot, J. M. *J. Agric. Food Chem.* **1982**, *30*, 1123–1127.
Marlett, J. A.; Lee, S. C. *J. Food Sci.* **1980**, *45*, 1688–1693.
Martin, F. W. In "Tropical Foods: Chemistry and Nutrition"; Inglett, G. E.; Charalambous, G., Eds.; Academic Press: New York, 1979; Vol. 1, p 249.
Morrison, I. M. *J. Sci. Food Agric.* **1980**, *31*, 639–645.
Robertson, G. L.; Swinburne, D. *J. Food Sci.* **1981**, *46*, 1557–1562.
Roe, B.; Bruemmer, J. H. *Proc. Fla. State Hortic. Soc.* **1977**, *90*, 180–182.
Selvendran, R. R.; DuPont, M. S. *J. Sci. Food Agric.* **1980**, *31*, 1178–1182.
Sharma, K. K.; Pattabiraman, T. N. *J. Sci. Food Agric.* **1982**, *33*, 255–262.
Sinclair, W. B. "The Grapefruit, Its Composition, Physiology and Products"; University of California, Division of Agricultural Science: Riverside, CA, 1972; p 223.

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A Study of the Mutagenicity of Irradiated Sugar Solutions: Implications for the Radiation Preservation of Subtropical Fruits

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A comprehensive investigation of the γ-radiolysis of sugars present in subtropical fruit was carried out and mutagenicity against *Salmonella* tester strain TA 100 demonstrated. An attempt was made to identify the products responsible for the mutagenic activity of irradiated sugar solutions and glucosone was implicated as a mutagenic agent. Irradiated mango fruit was found *not* to be mutagenic. The effect of fruit components on the mutagen glucosone was investigated and a decrease in activity demonstrated. The implications of these findings for the safety of radurized foods are discussed.

During the past 3 decades, proof of the wholesomeness of irradiated foods has been a major obstacle to the acceptance of food irradiation as a viable process. Recent animal feeding studies (International Food Irradiation Project, 1979) have explicitly proved the safety of irradiated subtropical fruits such as mango and papaya, while all foods irradiated to a dose of 10 kGy were recently declared wholesome for human consumption by a Joint Expert Committee of the International Atomic Energy Agency, the Food and Agriculture Organization, and the World Health Organization. Nevertheless, results indicating mutagenicity or cytotoxicity of irradiated, one-component sugar solutions in short-term mutagenic tests need further clarification.

For this reason a comprehensive investigation of the γ-radiolysis of the major sugars present in subtropical fruits, viz., fructose, glucose, maltose, and sucrose, together with ribose, which were shown (Aiyar and Subbo Rao, 1977) to become mutagenic upon irradiation, was carried out. At the same time an attempt was made to identify the specific products responsible for the observed effects. The investigation was further extended to the mutagenicity testing of an irradiated aqueous model mango solution (Basson et al., 1979) as well as irradiated mango fruit.

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EXPERIMENTAL PROCEDURES

Mutagenicity Testing. The *Salmonella* mutagenicity assay of Ames et al. (1975) was employed in the testing of synthesized compounds by using five tester strains, viz., TA 1535, 1537, 1538, 98, and 100. A modification of the preincubation mutagenesis assay described by Aiyar and Subba Rao (1977) was carried out for the testing of irradiated sugar solutions. Two milliliters of an 18-h nutrient broth culture of the tester strains was centrifuged (2000*g*, 15 min) and resuspended in 2 mL of minimal medium broth, supplemented by a trace of histidine (0.5 mM) and Biotin (0.5 mM). This facilitates the growth of the bacteria for a few generations, which is necessary for mutagenesis to occur. To this broth, 2 mL of the irradiated sugar solution to be tested was added and incubated for 3.5 h at 37 °C with constant shaking. After incubation the cells were centrifuged, washed with saline, and then resuspended in 2 mL of saline. Of the latter suspension, 0.1 mL was incorporated into 2 mL of molten (45 °C) supplemented top agar and, after being mixed, poured onto a minimal medium plate and incubated for 48 h at 37 °C, after which it was scored. Plates showing maximum mutagenesis were replicated onto minimal plates without histidine in order to check the true revertant nature of colonies. Viable counts were always made from the saline suspension to determine the toxicity of the solution being tested.

All experiments were carried out in duplicate and repeated at least twice.

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Table I. Mutagenic Response[a] of Irradiated Sugar Solutions

sugar solutions	dose yielding maximum response, kGy	no. of revertants[b] (strain TA 100)
control[c]	0	100
glucose oxygenated	10	252
glucose deoxygenated	30	174
fructose oxygenated	25	385
fructose deoxygenated	–[d]	–
sucrose oxygenated	20	262
sucrose deoxygenated	30	184
maltose oxygenated	11	298
maltose deoxygenated	19	192
ribose oxygenated	20	401
ribose deoxygenated	50	351

[a] A mutagenic response was regarded as positive when the number of revertants was increased at least 2-fold (Ames et al., 1975). [b] Viable counts varied between 10^8 and 10^9 organisms/mL. [c] Unirradiated sugar solutions. [d] No mutagenic activity.

Sugar solutions were made up with distilled water to a concentration of 1% (w/v) (glucose and fructose 5.5×10^{-2} M; sucrose 2.9×10^{-2} M; maltose 2.7×10^{-2} M; ribose 6.6×10^{-2} M).

The sterility of all solutions, amino acid supplements, top layer, and plates was also routinely checked in every test. Solutions were sterilized by filter (pore size 0.2 μm) with Sartorius filters.

Mango pulp supernatant was prepared by peeling ripened Kent mangoes and homogenizing the flesh in a Stomacher 400 for 2 min, followed by centrifugation at 18000*g* for 10 min at 4 °C. The supernatant was decanted and stored at 4 °C in screw-capped vials until used (within 24 h). The supernatant was too viscous to be sterilized by filtration, and in order to eliminate bacterial contaminants, Ampicillin was added in concentrations of 8 mg/mL. This did not affect the tester strain TA 100 as it contained the resistance transfer factor pKM101.

Irradiation Procedure. All solutions were irradiated in an AECL Gammabeam-650 facility of a dose rate of 16.64 kGy/h. Irradiations in the absence of oxygen were carried out in ampules sealed after degassing with the aid of an ultrasonic water bath. For the oxygen-saturated samples, the sugar solutions were irradiated in conical flasks while oxygen was being bubbled through. The temperature during irradiation was 25 °C.

RESULTS AND DISCUSSION

Mutagenicity of Irradiated Sugar Solutions. With the exception of deoxygenated fructose solutions, all irradiated sugar solutions were found to be mutagenic toward strain TA 100. The presence of oxygen during irradiation increased the mutagenic potential of all the sugars tested (Table I). Ribose showed the highest activity, followed by fructose, maltose, sucrose, and glucose. Deoxygenated fructose solutions showed no mutagenicity toward any of the tester strains, even when irradiated to doses of 40 kGy. At doses higher than the maximum one, the sugar solutions became toxic and caused a decrease in revertant numbers as well as the inhibition of the background lawn (Figure 1). No mutagenic response was found against any of the other tester strains. Our results on deoxygenated sucrose and ribose are qualitatively similar to those of Aiyar and Subba Rao (1977), obtained in the presence of either air or nitrogen. However, it should be pointed out that oxygen is depleted at a dose of approximately 0.5–0.75 kGy. Their results therefore reflect a deoxygenated state during irradiation rather than a true oxygenated state as in our experiments.

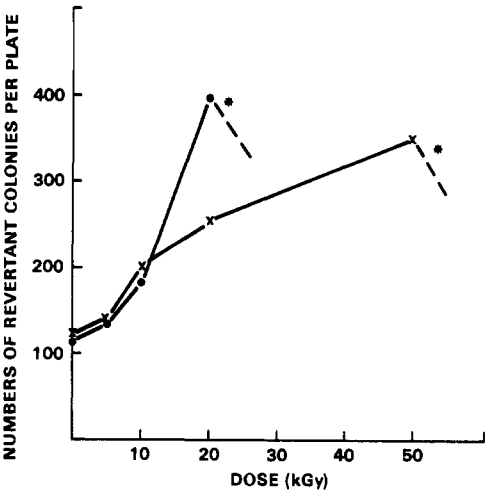


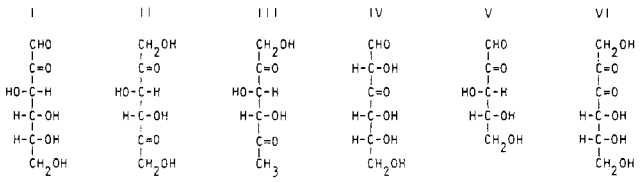
Figure 1. Mutagenic response of irradiated 1% aqueous ribose solutions to strain TA 100: (×) deoxygenated; (●) oxygen saturated; (*) toxicity to the tester strain was observed at doses higher than this optimum.

Effect of Peroxides. In order to test the possible effect of peroxides on the observed mutagenic response, catalase, an enzyme with peroxide-splitting capacity (final concentration 0.1%) was added to irradiated solutions of ribose immediately after irradiation. No differences in the mutagenicity could be detected due to the presence of catalase. Distilled water, irradiated to a dose of 50 kGy under oxygenated as well as deoxygenated conditions, was also tested, with no effect on the strains. We conclude, therefore, that the formation of peroxides in irradiated sugar solutions had no effect on the tester strains.

Mutagenicity of Individual Compounds. Our finding that the mutagenicity of irradiated sugar solutions is increased at least 2-fold by the presence of oxygen points to the formation of radiolysis products (or a series of products) that are oxygen dependent. The yield of products such as glucosone and glyoxal is indeed very low in deoxygenated solutions, as pointed out by Schubert and Sanders (1971). This is also true of the total carbonyl yield, which has been determined by Dizdaroglu et al. (1975) and Schuchmann and Von Sonntag (1977) as $G = 0.51$ for oxygen-free glucose solutions. However, in the presence of oxygen the yields increase considerably and the total yield for uloses was determined by these workers as $G = 2.6$ in an 80:20% N_2O–O_2 solution. In the case of ribose, the increase is even more pronounced from $G = 1.1$ to $G = 3.6$.

We decided to synthesize some of the radiolytic products of the sugars in subtropical fruit representative of various dicarbonyl structures and to test these compounds individually for mutagenicity. Six compounds were synthesized (den Drijver, 1979) and tested (Figure 2), together with 2-deoxyribose and 2-deoxyglucose as well as methyl vinyl ketone, crotonaldehyde and glyoxal.

2-Deoxyribose and 2-deoxyglucose were chosen as examples of deoxy sugars that have previously been suggested by Scherz (1968) as mutagenic agents. Methyl vinyl ketone and crotonaldehyde, found to be cytotoxic by Schubert and Sanders (1971), were also tested as examples of α,β-unsaturated carbonyl compounds. Glyoxal was shown to be mutagenic by Sasaki and Endo (1978) and by Bjeldanes and Chew (1979), although no dose–response data were given by the first authors, while the conclusions



I: D-arabino-Hexos-2-ulose (glucosone)
II: D-threo-Hexo-2,5-diulose
III: 6-deoxy-D-threo-Hexo-2,5-diulose
IV: D-ribo-Hexos-3-ulose
V: D-threo-Pentos-2-ulose (xylosone)
IV: D-erythro-Hexo-2,3-diulose

Figure 2. Dicarbonyl compounds synthesized.

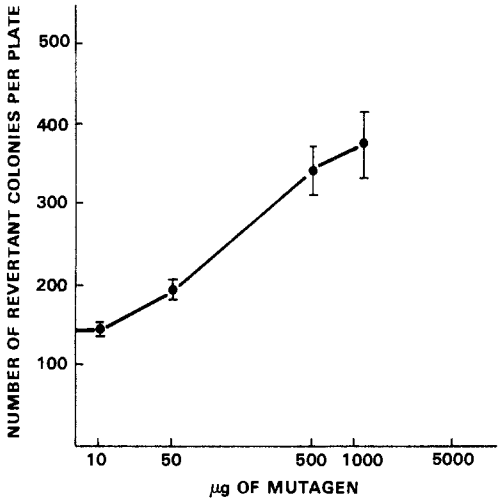


Figure 3. Mutagenic response of glyoxal to strain TA 100.

of the latter authors were based on a two-point mutagenicity response curve. The compounds were first tested by a screening procedure through the use of a simple spot test. Any indication of mutagenic activity was then followed up by a dose–response analysis over a wide concentration range of the compound using the standard plate incorporation assay. Due to the volatile nature of methyl vinyl ketone and crotonaldehyde, these compounds could not be tested quantitatively.

The following results were obtained. (i) Methyl vinyl ketone and crotonaldehyde were found to be highly cytotoxic, but no mutagenic activity could be observed by application of the spot test. (ii) Glyoxal was found to be mutagenic toward strain TA 100 (Figure 3). The dose–response curve shows increasing mutagenic activity from 10 to 1000 μg/plate, after which toxicity becomes pronounced. The highest concentration used (1000 μg) gives a 2.5-fold increase of the mutation frequency. (iii) No mutagenic activity could be ascribed to D-*threo*-hexo-2,5-diulose, D-*ribo*-hexos-3-ulose, or 6-deoxy-D-*threo*-hexo-2,5-diulose, either in the spot test or in the plate-incorporation assay. Similarly, no mutagenicity was observed for the 2-deoxy compounds. (iv) Preliminary experiments showed a very high mutagenic response for D-*erythro*-hexo-2,3-diulose. However, it was found that this compound was rather unstable at room temperature and would most probably decompose in aqueous solution. This instability rendered the compound unsuitable for further investigation. (v) D-*threo*-Pentos-2-ulose (xylosone) exhibited a mutagenic response at 1000 μg/plate with a maximum at 5000 μg/plate (Figure 4). (vi) D-*arabino*-Hexos-2-ulose (glucosone) was mutagenic at relatively high concentrations toward strain TA 100 (Figure 4). The mutagenic response only becomes meaningful at concentrations higher than 1000 μg/plate. The maximum response was obtained at 10000 μg/plate.

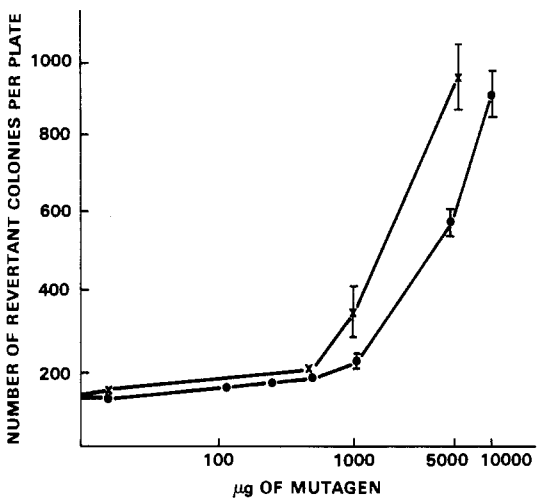


Figure 4. Mutagenic dose–response of D-*arabino*-hexos-2-ulose (glucosone) and D-*threo*-pentos-2-ulose (xylosone): (●) glucosone; (×) xylosone.

The presence of glucosone as a radiolytic product of irradiated glucose and fructose solutions has been demonstrated indirectly by Dizdaroglu et al. (1975), Von Sonntag (1980), and also Kawakishi et al. (1973). The formation of glucosone as a radiolytic product in oxygenated irradiated fructose solutions has recently been confirmed (den Drijver, 1979) in a study that also demonstrated for the first time the formation of D-*erythro*-hexo-2,3-diulose by utilizing capillary GC and also HPLC.

The optimum concentration of glucosone to use in the plate incorporation to demonstrate mutagenicity was found to be 10000 μg/plate, although mutagenicity was observed from 3000 μg upward. Kotchetkov et al. (1979) reported glucosone to be formed with G values of 0.9 for glucose, 0.6 for sucrose, and 2.2 for fructose when solutions of these sugars were irradiated in the presence of oxygen. This means that the glucosone used in the incubation procedure was approximately 165 μg/mL for glucose, 219 μg/mL for sucrose, and 1000 μg/mL for fructose.

Effect of S-9 Microsomal Enzymes on Glucosone Mutagenicity. No difference in the mutagenic response could be detected over the entire dose range in the presence of 100 μL of S-9 liver fraction (Ames et al., 1975) per plate. Sasaki and Endo (1978), however, described a weakening in the mutagenicity of glyoxal in the presence of microsome enzymes.

Study of Mutagenicity in Model Mango. The model mango, consisting of an aqueous solution of the various chemical components present in the real mango fruit (Basson et al., 1979), is made up of a number of classes, namely, class A, organic acids, class B, phenol and lipids, class C, cellulose and starch, and class D, sugars and carotene. The mutagenic behavior of this model mango was investigated. It was found that no mutagenicity could be ascribed to the solution in both its nonirradiated and irradiated (0.1–20 kGy) states.

Subsequently, the influence of the constituents of model mango on the mutagenicity of glucosone was studied. Two concentrations of glucosone were used, viz., the concentration that produced the maximum mutagenic response (10000 μg/plate $\approx 2.8 \times 10^{-2}$ M) and the concentration calculated from the initial G value for the formation of glucosone in glucose solutions [$G \approx 1$ according to Kotchetkov et al. (1979)] at a dose of 20 kGy (2×10^{-3} M). It should be stressed that both these concentrations are greatly in excess of that which would be found (if formed

Table II. Interaction of Glucosone with Model Mango

combinations with glucosone	no. of revertants[a] (strain TA 100)
control: glucosone in H_2O (2×10^{-3} M)	240
glucosone in model mango (2×10^{-3} M)	45
control: glucosone in H_2O (2.8×10^{-2} M)	342
glucosone in model mango (2.8×10^{-2} M)	67

[a] Viable counts varied between 10^8 and 10^9 organisms/mL.

Table III. Effect of Different Classes of Fruit Components on the Mutagenicity of Glucosone (2×10^{-3} M)

medium	no. of revertant colonies[a] (strain TA 100)	expression of mutagenicity, %
complete model mango	42	0
control (glucosone in H_2O)	241	100
model mango less class A (organic acids)	164	62
model mango less class B (phenol and lipids)	70	15
model mango less class C (cellulose and starch)	38	0
model mango less class D (sugars and carotene)	13	0
model mango less classes A and B	211	85

[a] Viable counts varied between 10^7 and 10^9 organisms/mL.

at all) in a fruit radurized for commercial purposes (0.75 kGy).

A considerable decrease in the mutagenicity of glucosone was observed in the presence of model mango (Table II). At both concentrations of glucosone tested, the mutagenic activity in model mango decreased when compared to that of glucosone in water only, viz., from 240 to 45 revertants at 2×10^{-3} M and from 342 to 67 revertants at 2.8×10^{-2} M. An attempt was made to identify the compounds responsible for the decrease. The effect of the various classes as described above on the mutagenicity of glucosone was monitored by a process of elimination. This approach was adopted in preference to mixing the single compounds on a one-by-one basis with glucosone, in order to be able to determine the total effect of all the other fruit components. The results obtained are shown in Table III.

From the table it is clear that the group consisting of classes A and B had the greatest effect on the mutagenicity of glucosone, since the omission of these compounds from the model mango allowed 85% of the mutagenicity to be expressed. The inclusion of these compounds (yielding a complete model mango) caused a decrease in mutagenicity of approximately 100%. As a single class of compounds, the greatest effect was found with the organic acids (class A). However, this class does not only have an individual effect, but an additive effect was observed when class B was added. It should perhaps be mentioned that this effect is not due to a change in pH caused by the absence of acids, since their deletion has virtually no effect on the pH of the mixture.

Study of the Mutagenicity of Kent Mangoes. In order to evaluate the meaning of the preceding results with respect to the radurization of real fruit, we investigated the mutagenicity of irradiated Kent mango and the interaction of the unirradiated fruit with glucosone.

Table IV. Interaction of Glucosone with Kent Mango

combinations with glucosone	no. of revertants[a] (strain TA 100)
control: glucosone in H_2O (2×10^{-4} M)	105
glucosone in mango supernatant (2×10^{-4} M)	51
control: glucosone in H_2O (2.8×10^{-2} M)	587
glucosone in mango supernatant (2.8×10^{-2} M)	188

[a] Viable counts varied between 10^7 and 10^9 organisms/mL.

Neither irradiated Kent mango juice nor the supernatant of the pulp of irradiated whole fruit exhibited any mutagenicity. This effect was observed at both the commercial radurization dose (0.75 kGy) and even at an extreme dose of 20 kGy.

The effect of Kent mango juice on the mutagenicity of glucosone was subsequently investigated by adding glucosone at two concentrations, 2×10^{-4} and 2.8×10^{-2} M, to the supernatant of unirradiated mango pulp. The mixture was tested for mutagenicity with the incubation procedure. Results are shown in Table IV, which clearly shows decreases in excess of 2- and 3-fold respectively.

CONCLUSIONS

The major conclusions drawn from this study can be summarized as follows. (1) One-component sugar solutions irradiated in the presence of oxygen were mutagenic toward tester strain TA 100 but had no effect on tester strains TA 1535, TA 1537, TA 1538, or TA 98. (2) Our findings on the mutagenicity of glucosone and xylosone, both 1,2-diuloses, is in accordance with that of Bjeldanes and Chew (1979), who found glyoxal and diacetyl to be mutagenic. The mutagenic activity of irradiated sugar solutions, therefore, could be ascribed to the presence of compounds with a 1,2-diketo structure. The possible role of α,β-unsaturated carbonyls as suggested by Schubert and Sanders (1971) cannot be totally excluded at this stage. However, several investigations by Dizdaroglu et al. (1975), Von Sonntag and Dizdaroglu (1977), and Kawakishi et al. (1973) failed to reveal the presence of these compounds in various irradiated sugar solutions. Furthermore, our own experience with the synthesis and purification of uloses and diuloses of compounds has demonstrated their extreme sensitivity in alkaline solutions, conditions under which Schubert and Sanders isolated α,β-unsaturated compounds from irradiated sugars. (3) No mutagenicity was found in either irradiated model mango or in irradiated real fruit. At this stage it is difficult to decide whether this is the result of mutagenic compounds such as glucosone not forming or of the reaction of such compounds with the constituents of the fruit. (4) When fruit components were added to the mutagen glucosone, its mutagenic activity disappeared. This effect was caused by a combination of various compounds and could not be linked to a single compound. (5) From the foregoing it should be clear that even if mutagenic compounds similar to glucosone were formed during the irradiation of fruit, they would be inactivated in the presence of fruit components. The fact should also be stressed that, at the doses used for food preservation, the yields of these products (if formed at all) would be 200–2000 times less than the concentrations used in our tests. All these findings correlate with the results of comprehensive animal feeding

studies (International Food Irradiation Project, 1979, 1981) that demonstrated the safety of irradiated subtropical fruits such as the mango.

Registry No. I, 26345-59-5; II, 1684-29-3; III, 57538-80-4; IV, 2092-61-7; V, 26188-06-7; VI, 28057-58-1; glucose, 50-99-7; fructose, 57-48-7; sucrose, 57-50-1; maltose, 69-79-4; ribose, 50-69-1; glyoxal, 107-22-2; crotonaldehyde, 4170-30-3; 2-deoxyribose, 1724-14-7; 2-deoxyglucose, 154-17-6; methyl vinyl ketone, 78-94-4.

LITERATURE CITED

Aiyar, A. A.; Subba Rao, V. *Mutat. Res.* **1977**, *48*, 17.
Ames, B. N.; McCann, J.; Yamakasi, E. *Mutat. Res.* **1975**, *31*, 347.
Basson, R. A.; Beyers, M.; Thomas, A. C. *Food Chem.* **1979**, *4*, 131.
Bjeldanes, L. F.; Chew, H. *Mutat. Res.* **1979**, *67*, 367.
den Drijver, L. M.Sc. Thesis, Rand Afrikaans University, 1979.
Dizdaroglu, M.; Henneberg, D.; Schomburg, G.; Von Sonntag, C. *Z. Naturforsch., B: Anorg. Chem., Org. Chem.* **1975**, *30B*, 416.
International Food Irradiation Project 1979, Report R51.
International Food Irradiation Project 1981, Reports R57 and R58.
Kawakishi, S.; Kito, Y.; Namiki, M. *Carbohydr. Res.* **1973**, *30*, 220.
Kotchetkov, N. K.; Kudrjashov, L.; Chlenov, M. A. "Radiation Chemistry of Carbohydrates", 1st ed.; Pergamon Press: New York, 1979; Chapter 5 and Appendix.
Sasaki, Y.; Endo, R. *Mutat. Res.* **1978**, *54*, 251.
Scherz, H. *Nature (London)* **1968**, *219*, 611.
Schubert, J.; Sanders, E. B. *Nature (London), New Biol.* **1971**, *233* (41), 199.
Schuchmann, M. N.; Von Sonntag, C. *J. Chem. Soc., Perkin Trans. 2* **1977**, *2*, 1958.
Von Sonntag, C. *Adv. Carbohydr. Chem. Biochem.*, **1980**, *37*, 7.
Von Sonntag, C.; Dizdaroglu, M. *Carbohydr. Res.* **1977**, *58*, 21.

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Mutagenicity of Extracts of Some Vegetables Commonly Consumed in the Netherlands

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An assessment was made of the mutagenic properties of six vegetables commonly consumed in the Netherlands. Extracts were screened in the *Salmonella typhimurium* strains TA98, TA100, and TA1537 by using the standard protocol. In addition, extracts of the gut flora (GFE) of rats were used as a metabolizing system. In total 27 cultivars grown under known and identical conditions were tested. Cultivars of lettuce, paprika, and rhubarb were mutagenic in TA98 in the presence of GFE. String beans were mutagenic in TA98 and TA100 with GFE. Rhubarb was also mutagenic in TA1537 when tested with liver homogenate. Spinach and Brussels sprouts were found negative. The mutagenic vegetables showed marked intercultivar variations with respect to their mutagenic properties. Evidence is obtained that quercetin glycosides are mainly responsible for the mutagenicity of lettuce and string beans. The mutagenicity of rhubarb in TA1537 is caused by emodin.

Identification of mutagenic factors in the environment is of concern because they may represent an important health hazard to man and other organisms. It is well documented that certain mutagenic chemicals play a predominant role in the etiology of cancer (e.g., McCann et al., 1975; Sugimura et al., 1981) and perhaps of artherosclerosis (Benditt, 1977; Bond et al., 1981). Moreover, it is expected that exposure of the population to mutagenic chemicals may result in congenital disorders including an increased predisposition for cancer as well as genetic diseases brought about by enzyme deficiencies or other anomalies. A consideration of mutagens in food is of special concern as, according to the results of various epidemiological studies, nutritional factors seem to be of considerable importance with regard to the etiology of cancer (Doll and Peto, 1981). This hypothesis is further supported by experimental studies which show that dietary variations and the presence of nutritional and other chemical factors in food markedly influence the tumor incidence in experimental animals (Visek et al., 1978). The chemical factors identified so far include carcinogens and/or mutagens, like mycotoxins, *N*-nitroso compounds, and pyrolysis products, flavonoids, and various modulating factors such as promoters and anticarcinogens.

The present study deals with the occurrence of mutagens in commonly consumed vegetables. Various studies already show that food plants contain mutagens. Ever since the work of Auerbach and Robson (1944) mutagenicity has been reported for a large variety of other food products of plant origin (Miller and Miller, 1976; Sugimura and Nagao, 1979; Nagao et al., 1979; Takahashi et al., 1979; Cheng et al., 1980; Stich et al., 1981; Lu et al., 1981; Uyeta et al., 1981; Ivie et al., 1981). However, until now, no systematical screening of food plants has been reported. As it was suspected that the mutagenic properties of crops might show considerable regional variation as a consequence of the variability of growing conditions and cultivars, special attention was given to intercultivar variation in mutagenicity of the vegetables investigated.

MATERIALS AND METHODS

Chemicals. Quercetin dihydrate was obtained from Fluka AG (West Germany), emodin from Sarsyntex (France), and rutin from Riedel—de Haën AG (West Germany). Sodium azide, L-histidine, and dimethylnitrosamine were purchased from Merck Schuchardt (West Germany). Ethidium bromide was from BDH Chemicals, Ltd. (England). Dimethyl sulfoxide (Me_2SO) and all other solvents were analytical grade. The NADPH-generating

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