Annales Agriculturae Fenniae

Maatalouden
tutkimuskeskuksen
aikakauskirja

Vol. 11,6

Journal of the
Agricultural
Research
Centre

Helsinki 1972

Annales Agriculturae Fenniae

JULKAISIJA — PUBLISHER

Maatalouden tutkimuskeskus
Agricultural Research Centre

Ilmestyy 4—6 numeroa vuodessa
Issued as 4—6 numbers a year

ALASARJAT — SECTIONS

Agrogeologia et -chimica — Maa ja lannoitus
Agricultura — Peltoviljely
Horticultura — Puutarhaviljely
Phytopathologia — Kasvitaudit
Animalia nocentia — Tuhoeläimet
Animalia domestica — Kotieläimet

KOTIMAINEN JAKELU

Valtion painatuskeskus, Annankatu 44, 00100 Helsinki 10

FOREIGN DISTRIBUTION

Agricultural Research Centre, Library, SF-01300 Tikkurila, Finland

ANNALES AGRICULTURAE FENNIAE, VOL. 11: 381—385 (1972)
Seria ANIMALIA NOCENTIA N. 63 — Sarja TUHOELÄIMET n:o 63

LOSS OF LINDANE, DIMETHOATE, AND METHYL PARATHION RESIDUES FROM SEEDLINGS OF SUGAR BEET AS INFLUENCED BY PLANT GROWTH

ANNA-LIISA VARIS

VARIS, ANNA-LIISA 1972. **Loss of lindane, dimethoate, and methyl parathion residues from seedlings of sugar beet as influenced by plant growth.** Ann. Agric. Fenn. 11: 381—385.

The insecticide residues in the aerial parts of cotyledonous sugar beet plants were determined at intervals after the following treatments: 1) seed dressing with lindane and thiram, 2) spraying with dimethoate, and 3) dusting with methyl parathion. Treatment 1 was carried out on the day of sowing. Active carbon was added to the preparation employed. Treatments 2 and 3 were carried out while the plants were at the cotyledon stage 14 days after sowing.

Fifteen days after treatment and sowing the first lindane determination gave a value of 15 p.p.m. There was a c. 50 per cent reduction of lindane residue in 2—3 days and a 87 per cent reduction in 6 days. On the day after treatment the dimethoate residue was 10 p.p.m. and the methyl parathion residue 0.50 p.p.m. With dimethoate the concentration fell to half the first-day value in 2—3 days, and with methyl parathion in little more than 24 hours. The reduction of dimethoate and of methyl parathion residues was c. 90 per cent in 6 days. The plants grew fairly rapidly during this period. The weight of the plants doubled in about 4 days and quadrupled in about 7 days.

The reduction of insecticidal residues due to growth was 73 per cent in 6 days.

Control measures with several insecticides have not proved to be very effective against the European tarnished plant bug (*Lygus rugulipennis* Popp.) on sugar beet crops. These bugs appear on sugar beet crops in early spring, when the plants are small and their relative growth is fairly rapid. Plant growth is an important factor diminishing insecticide concentrations in plants (e.g. HAMILTON 1929, SLOAN et al. 1951, DE PIETRI-TONELLI et al. 1965), and the residues tend to disappear more rapidly in young plants because of more rapid growth (DECKER 1957). An attempt was made to ascertain the rate at which some insecticides disappear from young sugar beet plants and the extent to which the rate of disappearance was due to the growth of the plant.

Material and methods

The investigations were performed in 1969. The experiment was carried out in the field at Tikkurila (near Helsinki). The sugar beet was sown on May 19. The treatments were:

Seed dressing containing 75 % lindane and 10 % thiram (Lindamal), 10 g per kg of seed. To reduce any phytotoxicity, use was also made of active carbon, 10 g per kg of seed. The seed dressing and the active carbon were first mixed together and then mixed with the dry seeds. The treatment was performed on the day of sowing.

40 % dimethoate spray (Roxion) 1.6 litres per hectare, with 640 litres of water per hectare.

1.5 % methyl parathion dust (Bladan E 605) 20 kg per hectare.

Plots 9.75 m^2 in size were replicated 4 times and randomized in blocks. As a very large number of seedlings were needed for the analysis, and as the sugar beet was not to be grown beyond the seedling stage, the rows were spaced at only 15 cm. Each plot thus contained 65 m. of rows. The soil was medium coarse sand. Lindane seed dressing was done on the day of sowing, and dimethoate spraying and methyl parathion dusting were applied 14 days after sowing, when the plants were at the cotyledon stage. For the purpose of the residue analyses the plants were broken off at the root collar and the aerial parts sent for analysis in plastic bags. The determinations were made on the dates below:

Date	Days after lindane treatment	Days after dimethoate or methyl parathion treatment
June 3	15	1
June 4	16	2
June 5	17	3
June 6	18	4
June 9	21	7
June 11	23	9

The intention was to take the same weight of plants for every analysis from each replicate of each treatment, in samples of at least 50 g per treatment. On the first day of determination some of the samples weighed slightly less than this, and the number of plants per treatment collected on that occasion was 900—1000. On the last occasion, it was possible to obtain samples weighing 80—90 g containing only 200 plants. All the samples were weighed. They were analysed by gas chromatography at the State Institute of Agricultural Chemistry. The methods used are described in a publication of that Institute (ANON. 1970).

Any effect of rain was eliminated by covering the treated plots during rain with plot-sized plastic covers fixed to wooden frames. During the trial it rained only once, on June 8, three days before the last determination of residue, the precipitation on that occasion being 9.2 mm. The temperatures of the days in the periods between treatment of the stand and determination were (°C):

	Mean temperature	Maximum temperature	Minimum temperature
June 2	12.0	18.2	4.6
June 3	10.1	13.8	7.0
June 4	10.9	15.8	7.8
June 5	7.8	11.7	3.2
June 6	8.8	10.9	7.1
June 7	11.1	16.7	3.0
June 8	13.8	21.8	7.5
June 9	14.7	20.8	8.2
June 10	18.3	23.0	9.5
June 11	18.4	22.9	10.0

The average daily temperature varied between + 5.0 and + 13.4 °C in the period between sowing and treatment of the stand.

Results and discussion

Figure 1 shows the diminishing of lindane, dimethoate, and methyl parathion concentrations. The residues of each insecticide in p.p.m. are calculated as percentages of the respective values for the first day of determination. These latter were lindane 15 p.p.m., dimethoate 10 p.p.m., and methyl parathion 0.50 p.p.m.

The insecticide residues of the plants (p.p.m.) diminished quite rapidly. When analysed 15 days after treatment and sowing, the aerial parts of the young seedlings contained 15 p.p.m. of the lindane used as seed dressing and after a further 6 days a mere 2 p.p.m. The dimethoate concentration fell to 50 per cent in 2—3 days, as did the methyl parathion concentration in somewhat above 24 hours. The concentrations of both these insecticides decreased by 90 per cent in 6 days. SANTI et al. (1962) investigated dimethoate residues in the roots of sugar beet, the leaves of which had been sprayed with 0.04 % P^{32} Rogor. The plants had been treated

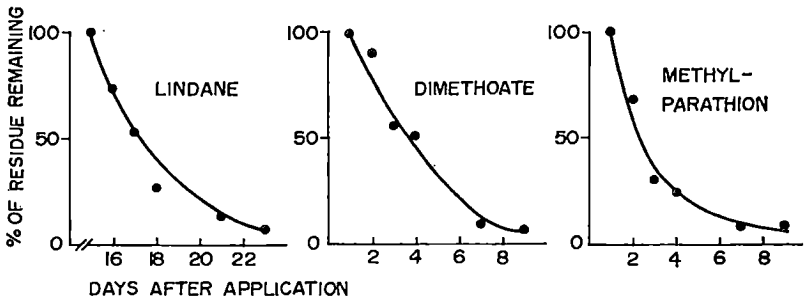


Fig. 1. Percentage disappearance of lindane, dimethoate and methyl parathion residues from sugar beet seedlings.

36 days after the emergence of the seedlings. The concentration of Rogor and its P = 0 derivative diminished at a rate corresponding to a half-life in 6—7 days. Five days after treatment, according to DECKER et al. (1950), leaves of apple and peach contained an average of 4—5 per cent of the initial parathion residue and c. 14 per cent of the initial lindane residue.

In the present study the concentration diminished rapidly. The disappearance of insecticidal residues depends on the plant's rate of growth and on other factors, particularly weather. EBELING (1963) gives the following list of factors influencing the rate of disappearance of the residues: nature of the plants surface, growth of the plant, formulation, rain, humidity, volatilization, wind, temperature, light and the inherent characteristics of the pesticide.

The effect of growth is quite considerable in rapidly growing plants and especially in young seedlings. The relative mass of these undergoes a rapid increase, and the residue, expressed in parts per million by weight, is quickly diluted even when the absolute amount of insecticide in the plant decreases at a slower rate.

At the cotyledon stage the weight of sugar beet seedlings increases quite rapidly (Fig. 2). While the weight of 1000 plants was 55.3 g on June 3, it had doubled in 4 days and quadrupled in c. 7 days. The disappearance of insecticidal residues in the sugar beet seedlings with growth (i.e. with increase in weight) is shown in Figure 3. It is the same for all the insecticides. The decrease of residue caused by growth was 50 per cent in 3—4 days and 73 per cent in 6 days.

SLOAN et al. (1951) studied the factors affecting the loss of parathion spray residues on lettuce. The combined action of growth and weathering for 2 weeks accounted for a reduction of 99 per cent of the residue. Growth alone caused a 73 per cent reduction in the two weeks before harvest. This was merely due to the increase in plant weight. HAMILTON (1929)

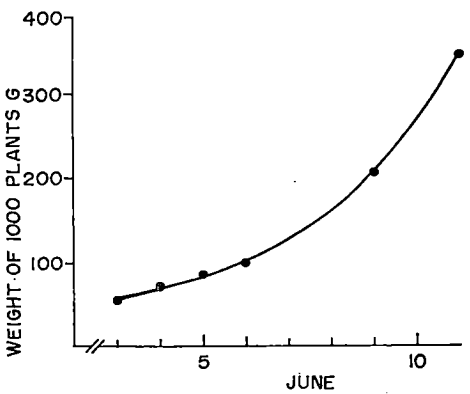


Fig. 2. The growth (weight increase) of young sugar beet seedlings. Sowing date 19.5.

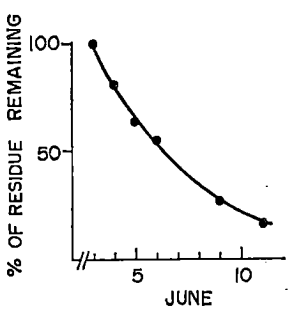


Fig. 3. Percentage disappearance of residue from sugar beet seedlings caused by growth (weight increase).

Table 1. The disappearance of residues of lindane, dimethoate, and methyl parathion in sugar beet seedlings. The lindane-thiram seed dressing was applied on the day of sowing, and dimethoate spraying and parathion dusting were done on June 2, when the plants were at the cotyledon stage.

Date of sampling	Residue mg/1000 seedlings		
	lindane	dimethoate	methyl parathion
3.6.	0.830	0.553	0.028
4.6.	0.750	0.512	0.020
5.6.	0.690	0.475	0.013
6.6.	0.498	0.398	0.012
9.6.	0.410	0.205	0.010
11.6.	0.314	0.209	0.010

investigated the decrease of arsenic residue in apples. When growth was rapid, it was the more important factor in the reduction of the residue; but when it was slow, weathering was more important.

According to DECKER (1957), the effect of growth upon residue is greatly dependent on the stage of growth of the plant at which treatment is given. When red clover was treated with DDT at three different stages of growth it was found not only that the initial deposits in p.p.m. were different because of the differences of surface to mass, but also that the residues disappeared more slowly from older plants because of their slower growth.

The effect of factors other than growth, primarily that of weathering, was assessed by calculating the amounts of insecticide contained in 1000 sugar beet plants in mg on the basis of the weight of the seedlings and the residue analysis (Table 1). It was then found that in 6 days the amount of lindane had decreased by 51 per cent, that of dimethoate by 63 per cent and that of methyl parathion by 64 per cent, on account of factors other than growth. It will be recalled that the effect of rain had been eliminated in the experiment by protecting the treated plots with plastic covers during rain.

The results show that lindane disappears more slowly, although, with the quantities of insecticide used, this fact is almost concealed by the rapid decrease due to growth, with the result that lindane disappears only slightly more slowly than dimethoate and methyl parathion.

Summary

In a field experiment at Tikkurila, the disappearance of lindane, dimethoate and methyl parathion residues from sugar beet seedlings and the effect of plant growth on the reduction of residues was studied.

The lindane-thiram seed dressing (75 % lindane and 10 % thiram), 10 g per kg of seed, was applied on the day of sowing. Active carbon was added to the preparation employed to reduce any phytotoxicity. Dimethoate as a 40 % spray, 1.6 l per hectare, and methyl parathion as a 1.5 % dust, 20 kg per hectare, were applied fourteen days after sowing, when the plants were at the cotyledon stage. The plants were broken off at the root collar and their aerial parts were analysed for insectidal residues by gas chromatography at the State Institute of Agricultural Chemistry. Dimethoate and methyl parathion were determined one, two, three, four, seven and nine days after application. The determinations of lindane were made concurrently, i.e. when 15, 16, 17, 18, 21 and 23 days had passed since treatment. During rain the plots were protected with plastic covers.

The lindane concentration on the first day of its determination fell by half in 2—3 days, and dropped by 87 per cent in 6 days. On the day after treatment, the concentration of dimethoate was 10 p.p.m. and the concentration of parathion 0.50 p.p.m. A decrease of residue to half this amount took 2—3 days for dimethoate, and slightly more than 24 hours for methyl parathion. In 6 days the loss of dimethoate and methyl parathion residues was c. 90 per cent. The plants were growing relatively rapidly at that period. The weight of 1000 seedlings doubled in about 4 days and quadrupled in about 7 days. Mere growth caused a decrease in insecticide residues of 73 per cent in 6 days.

REFERENCES

ANON. 1970. Investigations on pesticide residues 1969. Publ. State Inst. Agric. Chem. 5: 1—28.

DE PIETRI-TONELLI, P., BAZZI, B. & SANTI, R. 1965. Rogor

(dimethoate) residues in food crops. Residue Reviews 11: 60—99.

DECKER, G. C. 1957. Pesticide residue on plants. Agric. Chemicals 12: 39.

—, WEINMAN, C. J. & BANN, J. M. 1950. A preliminary report on the rate of insecticide residue loss from treated plants. J. Econ. Ent. 43: 919—927.

EBELING, W. 1963. Analysis of the basic processes involved in the deposition, degradation, persistence, and effectiveness of pesticides. Residue Reviews 3: 35—163.

HAMILTON, C. C. 1929. The growth of the foliage and fruit of the apple in relation to the maintenance of a spray coating. J. Econ. Ent. 22: 387—396.

SANTI, R., RADICE, M., GIACOMELLI, R. & BAZZI, B. 1962. Studio sul metabolismo del Rogor P^{32} nelle bietole da zucchero e da foraggio. Ist Ric. Agrar. Soc. Montecatini 1962. (Ref. De Pietri-Tonelli, P. et al. 1965.)

SLOAN, M. J., RAWLINS, W. A. & NORTON, L. B. 1951. Factors affecting the loss of DDT and parathion residues on lettuce. J. Econ. Ent. 44: 701—709.

MS received 31 August 1971

Anna-Liisa Varis
Agricultural Research Centre
Dept. of Pest Investigation
SF-01300 TIKKURILA, Finland

SELOSTUS

Lindaanin, dimetoaatin ja metyyliparationin häviäminen sokerijuurikkaan taimista ja kasvien kasvun vaikutus siihen

ANNA-LIISA VARIS

Maatalouden tutkimuskeskus, Tuhoeläintutkimuslaitos, Tikkurila

Kun sokerijuurikasmailla keväisin suoritettavissa peltoluteen torjuntakäsittelyissä torjunta-aineiden tehot jäävät melko heikoiksi, järjestettiin vuonna 1969 Tikkurilassa tutkimuksia, joissa selvitettiin peittausaineena käytetyn lindaanin, ruiskutteena käytetyn dimetoaatin ja pölytteenä käytetyn metyyliparationin häviämisnopeutta sokerijuurikkaan taimista.

Aineisto kasvatettiin kenttäkokeena. Siementen lindaanikäsittely tehtiin kylvöpäivänä. 75 % lindaania ja 10 % tiraamia sisältävää peittausainetta käytettiin 10 g siemenkiloa kohti. Mahdollisen fytotoksisuuden vähentämiseksi sekoitettiin peittausaineeseen aktiivista hiiltä samoin 10 g siemenkiloa kohti. Dimetoaattiruiskutus ja metyyliparationipölytys tehtiin kasvien ollessa pienellä sirkkataimiasteella, 14 vuorokauden kuluttua kylvöstä. 40 %:n dimetoaattiruiskutetta käytettiin 1.6 l/ha, vesimäärä oli 640 l/ha. 1.5 %:n metyyliparationipölytettä käytettiin 20 kg/ha. Insektisidipitoisuusanalyysejä varten taimet katkaistiin juurenniskasta ja niiden maanpäälliset osat analysoitiin. Analyysit tehtiin valtion maatalouskemian laitoksessa. Dimetoaatti- ja metyyliparationipitoisuus määritettiin 1, 2, 3, 4, 7 ja 9 vuorokauden kuluttua käsittelystä. Samanaikaisesti määritettiin myös lindaanipitoisuus. Siemenen lindaanikäsittelystä oli tällöin kulunut 15, 16, 17, 18, 21 ja 23 vuorokautta. Sateen ajaksi käsitellyt ruudut peitettiin muovikatoksella. Kokeen aikana satoi vain kerran, kolme vuorokautta ennen viimeistä määritystä.

Käsittelyn jälkeisenä päivänä oli taimien dimetoaattipitoisuus 10 mg/kg ja parationipitoisuus 0.50 mg/kg. Dimetoaattipitoisuus laski 2—3 vuorokaudessa ja metyyliparationipitoisuus runsaassa vuorokaudessa puoleen tästä määrästä. Kuudessa vuorokaudessa sekä dimetoaatti- että metyyliparationipitoisuus laski n. 90 %. Vastaavasti ensimmäisen määrityspäivän lindaaniarvo laski puoleen 2—3 vuorokaudessa, ja kuudessa vuorokaudessa sen lasku oli 87 % (kuva 1).

Kasvit kasvoivat tänä aikana varsin nopeasti. 1000 taimen paino kaksinkertaistui noin neljässä ja nelinkertaistui noin seitsemässä vuorokaudessa (kuva 2). Pelkästään kasvien kasvun aiheuttama insektisidipitoisuuden aleneminen oli kuudessa vuorokaudessa 73 % (kuva 3).

ANNALES AGRICULTURAE FENNIAE, VOL. 11: 386—390 (1972)

Seria AGROGEOLOGIA ET -CHIMICA N. 61
Sarja MAA JA LANNOITUS n:o 61

THE EFFECT OF AMMONIATION ON THE EFFICIENCY OF SUPERPHOSPHATE

RAILI JOKINEN

JOKINEN, R. 1972. **The effect of ammoniation on the efficiency of superphosphate.** Ann. Agric. Fenn. 11: 386—390.

A highly ammoniated granular superphosphate (8.7 % P, 8.3 % N) was tested in pot trials in comparison with an equivalent amount (based on total P) of ordinary non-granular superphosphate (8.5 %) at different levels of liming. With no liming (soil pH 5) equally large grain and total yields were obtained with both the ordinary and the ammoniated superphosphate. Increasing rates of liming (to pH 6 and pH 7) produced greater differences between the effects of the two fertilizers. This was clearest in the first year and statistically significant in three years out of four. The total uptake by oats over the entire experimental period amounted to 19 % of the P given in the ordinary superphosphate and 15 % of the P given in the ammoniated superphosphate. At the end of the experiments the soils fertilized with ammoniated superphosphate had lower contents of phosphorus and calcium soluble in acid ammoniumacetate than had the soils fertilized with ordinary superphosphate.

The phosphorus contained in superphosphate is mainly water soluble. In the ammoniation process the proportion of water-soluble P has been found to decrease steeply with the increasing degree of ammoniation (i.a. BRABSON and BURCH 1964). The effectiveness of ammoniated superphosphate as a fertilizer has been tested in both pot and field experiments (COOKE and WIDDOWSON 1953, TERMAN et al. 1956, OLSON et al. 1956). The results show higher crop yields with ordinary superphosphate than with superphosphates ammoniated to various levels. Chemical determinations of usability, however, have shown the two types of superphosphate to be of almost equal value. The aim of the present paper is to study the fertilizer effect of a Finnish-manufactured ammoniated superphosphate in comparison with an ordinary superphosphate, as tested in pot experiments at different levels of soil acidity.

Material and methods

A lot of very highly ammoniated granular superphosphate (asf), manufactured by the Finnish company Rikkihappo Oy[1] at a pilot plant in Kotka, was subjected to comparison with an ordinary non-granular superphosphate (sf). The compositions of the two fertilizers were as follows:

	Total P %	Water-soluble P %	N %
sf	8.5	7.9	
asf	8.7	1.1	8.3

[1] The present name from the 1st July 1972: Kemira Oy.

The fertilizers were applied at rates corresponding to 0, 218 and 437 mg P per pot (calculated on the basis of total P).

Mitscherlich pots were filled with 4.5 l of soil (muddy clay, organic C 2.0 %, pH 5.0, P 4.4, K 370, Ca 1400, Mg 700 mg/l). The following yearly dressing was given:

1000 mg N as NH_4NO_3 or NH_4NO_3 + asf
0, 218 or 437 mg P (total P) as sf or asf
830 mg K as KCl
10 mg H_3BO_3
50 mg $CuSO_4 \cdot 5H_2O$
50 mg $MnSO_4 \cdot 7H_2O$
50 mg $ZnSO_4 \cdot 7H_2O$
10 mg $Na_2MoO_4 \cdot 2H_2O$
2000 mg $MgSO_4 \cdot 7H_2O$ (in 1969 only)

Those pots fertilized with the ordinary superphosphate received their entire dose of nitrogen as ammonium nitrate. The amounts of nitrogen delivered in the ammoniated superphosphate, 213 and 426 mg, were taken to the required level by adding ammonium nitrate. Soil acidity was adjusted close to pH values 5, 6 and 7 by applying, in the first year, various rates of $CaCO_3$ ($Ca_0 = 0$, $Ca_1 = 12$, $Ca_2 = 24$ g/pot). The soil pH_{H_2O} was cheked yearly after harvest and the following changes were found:

	Ca_0	Ca_1	Ca_2
1967	5.1	6.0	6.8
1968	4.9	5.5	6.4
1969	4.8	5.4	6.2
1970	4.7	5.2	6.0

The test crop, Pendek oats, was in each year harvested at full maturity.

Results and discussion

The following yields were obtained without phosphorus fertilization:

	Ca_0	Ca_1	Ca_2
		Grain yield g/pot	
1967	16.9	17.2	27.8
1968	12.2	14.0	20.1
1969	28.8	22.0	29.2
1970	12.8	10.0	20.6
		Total yield g/pot	
1967	43.7	50.8	60.9
1968	30.6	31.2	41.7
1969	57.6	49.6	56.6
1970	29.2	25.7	39.6

The favourable effect of liming on the availability of soil P (SALONEN 1964, KAILA 1965) was evident in this muddy clay soil in a couple of years only. The rise in yield in 1969 can be ascribed to the magnesium applied in that year.

Figure 1 shows the yearly grain and total

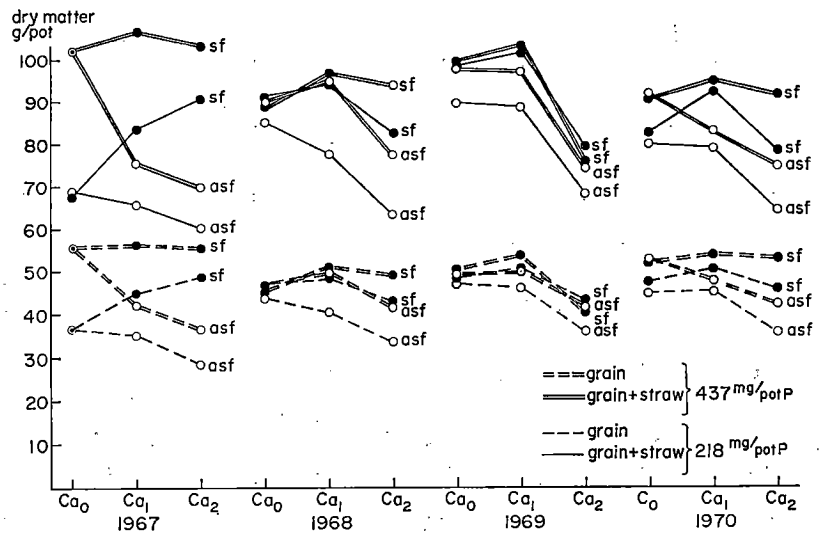


Fig. 1. The effect of liming on the yields of oats obtained with ordinary superphosphate (sf) and ammoniated superphosphate (asf) in the years 1967—70.

Table 1. Nutrient contents (mg/g dry matter) of the grain and straw yields.

	Grain			Straw		
	Ca_0	Ca_1	Ca_2	Ca_0	Ca_1	Ca_2
			Nitrogen (N)			
No phosphorus	20.0	25.1	22.7	14.7	13.6	8.6
sf Rate 1	15.9	15.7	16.4	4.1	3.9	3.8
„ „ 2	14.3	14.8	16.1	3.8	3.3	4.1
asf Rate 1	16.7	19.4	21.3	4.5	4.7	5.5
„ „ 2	15.2	16.4	17.7	4.0	3.7	5.0
			Phosphorus (P)			
No phosphorus	3.5	3.7	3.6	0.9	1.1	0.8
sf Rate 1	2.5	2.5	2.9	0.3	0.3	0.3
sf Rate 2	2.9	3.1	3.3	0.3	0.4	0.5
asf Rate 1	2.6	2.8	3.4	0.3	0.3	0.6
„ „ 2	2.8	3.1	3.2	0.2	0.3	0.4
			Potassium (K)			
No phosphorus	4.8	4.4	4.4	38.5	40.7	39.7
sf Rate 1	4.0	3.9	3.6	25.1	26.4	28.1
„ „ 2	3.9	4.1	4.2	23.0	25.7	28.0
asf Rate 1	3.6	3.9	4.3	29.8	33.3	37.8
„ „ 2	4.1	4.3	4.4	25.1	31.8	34.1
			Calcium (Ca)			
No phosphorus	0.6	0.7	0.7	2.3	3.2	3.4
sf Rate 1	0.5	0.6	0.7	2.1	2.8	3.5
„ „ 2	0.5	0.7	0.7	2.5	3.3	4.1
asf Rate 1	0.5	0.6	0.7	1.9	2.5	3.2
„ „ 2	0.5	0.6	0.7	2.3	2.5	3.0
			Magnesium (Mg)			
No phosphorus	1.4	1.3	1.3	1.8	1.8	1.8
sf Rate 1	1.1	1.1	1.2	1.5	1.4	1.4
„ „ 2	1.3	1.2	1.3	1.5	1.5	1.4
asf Rate 1	1.2	1.2	1.4	1.4	1.3	1.7
„ „ 2	1.2	1.3	1.4	1.4	1.4	1.4

yields of oats obtained with the two fertilizers at the different levels of liming. The results are averages of three replicates. The increase in yield following phosphorus fertilization was very significant in all years. With no liming approximately equal yields were obtained with both of the two P fertilizers. The difference between the effects of the fertilizers became clearer with the rising level of liming (liming rate × type of fertilizer $P > 0.01$) in all years except 1969. Irrespective of the rate of liming, ammoniated superphosphate gave lower yields on the average than the ordinary superphosphate (type of fertilizer $P > 0.01$). The main reason for the difference between the two fertilizers probably lies in their different contents of water-soluble P. The ammoniation process produces phosphates insoluble in water and citrate. Furthermore, the fact that the ammoniated superphosphate used for these experiments was in granulated form, whereas the ordinary superphosphate was not, may also have contributed to the results.

The nutrient contents of the grain and straw yields (Table 1) were clearly dependent on the rate of liming and the P fertilization. There were differences in the nitrogen contents of the grain as well as in the nitrogen, potassium and calcium contents of the straw following the use of the two types of fertilizer. The differences in Ca are probably due to differences in availability to the plants of the calcium phosphates contained in the fertilizers. The higher N and K contents of the yields obtained with ammoniated superphosphate may be ascribed to the postponement of maturing due to P deficiency. The proportion of fresh green shoots in the yield

was the larger, the higher the rate of liming. Delayed maturing is also indicated by the determinations of dry matter in the total yields:

	Dry matter in yield %					
	Ca_0		Ca_1		Ca_2	
	sf	asf	sf	asf	sf	asf
1967	58.7	54.5	64.3	52.1	67.4	42.9
1968	58.1	57.5	58.7	57.0	56.9	51.7
1969	48.7	49.6	47.3	45.6	46.4	42.2
1970	74.2	69.0	66.1	63.7	68.9	53.6

There was no difference between the two fertilizers in the four-year total uptake of nitrogen (mg/pot, Table 2) by the crops (grain + straw). The treatments that received ordinary superphosphate showed a significantly higher uptake of P, Ca and Mg but a lower uptake of K than the treatments with ammoniated superphosphate.

The plants used a significantly higher proportion of the phosphorus contained in the ordinary superphosphate (average 19 %, Table 3) than of that contained in the ammoniated superphosphate (15 %). The availability of the ammoniated superphosphate to the plants decreased with increasing rate of liming. The higher rate of P application gave the clearest difference between the fertilizers. The following changes occurred during the experimental period in the apparent recovery of the phosphorus given in the two types of fertilizer:

P applied	sf		asf	
	recovery %	increase from previous	recovery %	increase from previous
1st year	7.1		6.6	
1st + 2nd year	12.7	+ 5.6	9.1	+ 2.5
1st + 2nd + 3rd year ..	15.3	+ 2.6	10.9	+ 1.8
1st + 2nd + 3rd + 4th year	19.0	+ 3.7	14.8	+ 3.9

In the first and fourth year of the experiments the level of P recovery from both of the two fertilizer types was almost the same, whereas in the other two years P uptake was higher from the ordinary superphosphate (total cumulated increase 8.2 %) than from the ammoniated superphosphate (increase 4.3 %).

Table 2. Four-year total uptake of nutrients (mg/pot) by the oats (grain + straw).

	Ca_0	Ca_1	Ca_2
	Nitrogen (N)		
No phosphorus	2733	2807	3091
sf Rate 1	3434	3692	3552
" " 2	3605	3817	3852
asf Rate 1	3507	3855	3464
" " 2	3831	3634	3495
	Phosphorus (P)		
No phosphorus	319	333	430
sf Rate 1	485	524	569
" " 2	635	723	718
asf Rate 1	482	513	524
" " 2	614	630	563
	Potassium (K)		
No phosphorus	3821	4091	4437
sf Rate 1	4687	5474	4874
" " 2	4963	5748	5396
asf Rate 1	5143	5508	5202
" " 2	5341	5979	5307
	Calcium (Ca)		
No phosphorus	248	332	408
sf Rate 1	428	621	735
" " 2	547	744	791
asf Rate 1	368	463	489
" " 2	509	516	514
	Magnesium (Mg)		
No phosphorus	263	267	314
sf Rate 1	436	467	427
" " 2	519	545	471
asf Rate 1	400	394	387
" " 2	501	457	405

Table 3. Four-year total uptake of fertilizer phosphorus (mg/pot and %) by the oats (grain + straw).

	Ca_0	Ca_1	Ca_2	
		mg/pot		
sf Rate 1....	166	191	139	
" " 2	316	390	288	
asf Rate 1	163	180	94	
" " 2	295	297	133	
		%		average %
sf Rate 1	19.0	21.9	15.9	18.9
" " 2	18.1	22.3	16.5	19.0
asf Rate 1	18.7	20.6	10.8	16.7
" " 2	16.9	17.0	7.6	13.8

Soil samples (Table 4) taken from the various treatments after termination of the experiments showed that the phosphorus content of the soil was dependent on the level of liming (rate of liming × type of fertilizer $P > 0.1$).

Table 4. Nutrient contents of the experimental soils (mg/l of soil, soluble in acid ammoniumacetate) at the end of experiments.

	Ca_0	Ca_1	Ca_2
		pH_{H_2O}	
No phosphorus	4.6	5.1	6.0
sf Rate 1	4.7	5.2	5.9
„ „ 2	4.5	5.0	5.7
asf Rate 1	4.6	5.2	5.8
„ „ 2	4.6	5.0	5.7
		Phosphorus(P)	
No phosphorus	3.7	3.1	2.9
sf Rate 1	8.6	7.1	6.3
„ „ 2	14.2	12.0	11.8
asf Rate 1	7.5	6.3	6.8
„ „ 2	11.2	8.7	11.5
		Potassium (K)	
No phosphorus	230	225	205
sf Rate 1	170	150	185
„ „ 2	155	150	170
asf Rate 1	150	155	160
„ „ 2	160	155	160
		Calcium (Ca)	
No phosphorus	1450	2275	3200
sf Rate 1	1925	2750	3625
„ „ 2	2325	3125	4000
asf Rate 1	1750	2650	3325
„ „ 2	1800	2800	3575
		Magnesium (Mg)	
No phosphorus	600	600	590
sf Rate 1	580	575	580
„ „ 2	540	555	590
asf Rate 1	605	630	580
„ „ 2	580	605	570

REFERENCES

BRABSON, J. A. & BURCH, W. G., Jr. 1964. How available is availabler. J. Assoc. Offic. Agric. Chemists 47: 439—442.

COOKE, G. W. & WIDDOWSON, F. V. 1956. Field experiments on phosphate fertilizers. A joint investigation. J. Agric. Sci. 53: 46—63.

KAILA, A. 1965. Effect of liming on the mobilization of soil phosphorus. J. Sci. Agric. Soc. Finl. 37: 243—254.

OLSON, R. A., DREIER, A. F., LOWREY, G. W. & FLOWERDAY, A. D. 1956. Availability of phosphate carriers to small grains and subsequent clover in relation to I. Nature of soil and method of placement. Agron. J. 48: 106—111.

SALONEN, M. 1964. Kalkin ja fosforilannoitteiden samanaikaisen käytön vaikutuksesta niiden tehoon. Summary: The effect of simultaneous application of lime and phosphorus fertilizers on their efficiency. Ann. Agric. Fenn. 3: 287—295.

TERMAN, G. L., ANTHONY, J. L., MORTENSEN, W. P. & LUTZ, J. A., Jr. 1956. Crop response of NPK fertilizers varying in granule size and water solubility of the phosphorus. Proc. Soil Sci. Soc. Amer. 20: 551—556.

MS received 8 January 1972

Raili Jokinen
Agricultural Research Centre
Dept. of Agricultural Chemistry and Physics
SF-01300 TIKKURILA, Finland

SELOSTUS

Ammonoinnin vaikutus superfosfaatin tehoon

RAILI JOKINEN

Maatalouden tutkimuskeskus, Maanviljelyskemian ja -fysiikan laitos, Tikkurila.

Suomessa (Rikkihappo Oy, nyk. Kemira Oy) valmistettua hyvin pitkälle ammonoitua rakeista superfosfaattia (asf, 8.7 % P, 8.3 % N) verrattiin astiakokeessa happamalla liejusavella (org. C 2.0 %, pH 5.0, P 4.4, K 370, Ca 1400, Mg 700 mg/l) tavalliseen jauheiseen superfosfaattiin (sf, 8.5 % P). Ammonoitu superfosfaatti sisälsi 1.1 % ja superfosfaatti 7.9 % veteen liukenevaa fosforia.

Kokeessa kaikki lannoitteet (typpi, fosfori, kalium ja hivenaineet) annettiin astioihin vuosittain, kalkitus ($Ca_0 = 0$, $Ca_1 = 12$, $Ca_2 = 24$ g/ast. kalsiumkarbonaattia) kuitenkin vain ensimmäisenä vuotena. Fosforilannoitelajien vertailu suoritettiin fosforin kokonaismäärän mukaan lasketuilla 0, 218 ja 437 mg/ast. P vastaavilla määrillä lannoitteita.

Ilman kalkitusta ei tutkituilla lannoitteilla saatujen kauran jyvä- tai kokonaissatojen (kuva 1) välillä ollut eroa. Kalkitus vähensi ammonoidulla superfosfaatilla saatuja satoja kaikkina vuosina, selvimmin ensimmäisenä vuotena.

Ammonoidulla superfosfaatilla saaduissa sadoissa oli ehkä fosforin puutteen vuoksi korjuuhetkellä vihreitä sivuversoja sitä enemmän, mitä runsaampi kalkitus oli annettu. Tämä seikka vaikutti satojen ravinteiden pitoisuuksiin (taul. 1) ja otettujen ravinteiden määriin (taul. 2). Kaura käytti hyväkseen koko aikana annetusta superfosfaatin fosforista 19 % ja ammonoidun superfosfaatin fosforista 15 %. (taul. 3).

Kokeen loputtua oli ammonoidulla superfosfaatilla lannoitetuissa maissa (taul. 4) happamaan ammoniumasetaattiin liukenevaa fosforia ja kalsiumia vähemmän kuin superfosfaattia saaneissa maissa.

ANNALES AGRICULTURAE FENNIAE, VOL. 11: 391—406 (1972)

Seria AGROGEOLOGIA ET -CHIMICA N. 62
Sarja MAA JA LANNOITUS n:o 62

POSSIBLE CAUSAL RELATIONSHIP BETWEEN NUTRITIONAL IMBALANCES ESPECIALLY MANGANESE DEFICIENCY AND SUSCEPTIBILITY TO CANCER IN FINLAND

HELVI MARJANEN and SYLVI SOINI

MARJANEN, H. & SOINI, S. 1972. **Possible causal relationship between nutritional imbalances especially manganese deficiency and susceptibility to cancer in Finland.** Ann. Agric. Fenn. 11: 391—406.

An increase in the incidence of cancer seems to exist concomitantly with a decrease in the amount of easily soluble manganese in Finnish arable mineral soils. According to various research workers, manganese is one of the most important factors in the normal reactions of cells, as an activator or catalyst. Faulty reactions in the cells are possible when the internal balances are upset. Deficiencies of many elements have been found in plant foods in Finland. These include Mg, Mn, Se and the SH group, Cu, Co, Zn, B and Mo. All are important for animal or plant metabolism. Treatment of the products prior to cooking reduces the content of minerals and vitamins. A shortage of the above compounds, or divergences in their proportions from certain norms seem to be directly or indirectly connected with susceptibility to cancer. Manganese deficiency in Finland may trigger an upset in metabolism to such a degree that faulty reactions resulting in cancer may occur.

Introduction

The Finnish cancer research workers HAKAMA and SAXÉN suggested in 1967 that the high mortality rates of stomach cancer in Finland might be associated with high cereal consumption, which correlates significantly with the mortalities. At the Ninth International Cancer Congress, HIGGINSON (1967) immediately posed the question of how, in this case, it was possible to explain the high consumption of cereals with a low incidence of stomach cancer in the nations of Africa. The reply might be that, until recently, Africans have used their cereal products milled together with the husks. Bread or porridge made of whole grain cereal is a balanced food, while the wheat bread which has been rather commonly consumed by the Finns since the 1930s, is made chiefly from wheat flour or marrow wheat flour. According to KOIVISTOINEN (1971) only 10—15 % of the magnesium and manganese, 20—28 % of the iron and zinc and some 50 % of the copper of the whole grain is left in the Finnish wheat flour. It has not been determined how much selenium is left. The selenium content of whole

grain wheat in Finland is only 0.004—0.085 ppm (OKSANEN and SANDHOLM 1970) and in the United States 0.01—3.0 ppm (COWAN 1971).

ROBINET (1930, 1934), DELBET (1934), DELBET and ROBINET (1934) and FAVIER (1951) found plenty of magnesium in the soil, as did TROMP (1954) and in the water, in places where the incidence of cancer was low. TROMP and DIEHL (1955) found in Holland, as did MARJANEN (1969) in Finland, that there is an increase in the incidence of cancer with a decrease in the amount of easily soluble manganese in arable soils. TROMP (1954) reports the same about the manganese in drinking water. ROSE (1968) found in South Africa that in areas where the cancer incidence was high, cultivated plants were deficient in manganese, molybdenum, copper, boron, zinc and iron. KMET and MAHBOUBI (1972) mention that there may be deficiencies of iron, manganese, boron, copper and zinc in plants from that area of Iran where the cancer incidence is highest. The area is on alkaline, saline soil. According to FROST (1970, 1972) studies have been published in which the possible value of selenium against cancer was reported. CLEMMESEN (1965) mentions that cancer patients show deficiencies of iron and molybdenum, as well as vitamins A, B_1, B_{12} and C. Attention has also been paid to excess amounts of trace elements. HALME (1968) associates excesses of zinc with the incidence of cancer, and KEIDERLING and SCHARPF (1953) excesses of copper. According to LESHAN (1959) psychological states could frequently be revealed as factors affecting the development of malignant disease. Most attention recently, however, has been paid to the virus theory.

The actual causes of cancer are not yet regarded as conclusively established. This is true despite the many investigations concerning the essence of cancer. Some of the studies deal with disturbances in functions of the cell. That receiving most frequent mention implies injury to cell respiration (WARBURG 1956). Others deal with deficiencies in various nutrients obtained from the soil through plants. Yet others deal with various viruses.

A view of cell metabolism

Agriculture is expected to produce wholesome food products of the vegetable and animal kingdoms. It is reasonable to have a look at the significance of various minerals, and of some vitamins and their mineral demands in metabolism. In order to facilitate analysis, patterns of some of the reaction pathways of the normal cell have been combined in a diagram based on various investigations. In this diagram attention is paid chiefly to the activators. Some of the activators of vegetable cells have been marked out in the diagram, these being indispensible for the nutrition of the animal kingdom.

LIEBERMAN and BAKER (1965) mention that Ca^{++}, Mg^{++}, Mn^{++} and Sr^{++} as well as P and K^+ collect in plants on the inner surfaces of the mitochondria. CHAPPELL et al. (1963) showed that of these elements, manganese (Mn^{++}) is easily driven out of the mitochondria membrane. This occurs if the respiratory chain (Fig. 1 point. 1) does not function due either to an inhibitory agent or a shortage of phosphorus. In turn, a partial cause of the latter may be an excess of K^+, which, according to RASMUSSEN et al. (1964), causes a decrease in Mg^{++} and an accumulation of phosphates, which in turn begin to cause a reduction in the uptake of potassium.

The starting materials of the respiratory chain are energy linked through niacin derivatives in the forms of NADPH and NADH and the riboflavin derivative $FADH_2$ and succinate all of which are intermediates in the Krebs cycle (2).

As more manganese than magnesium is needed in the Krebs cycle starting from acetyl-CoA (3), it is possible that the reactions in the cycle may be made difficult on account of a deficiency of Mn^{++} and reactions from acetyl-CoA onwards may be directed to the synthesis of fatty

acids (6) instead of the Krebs cycle. In other words, in the absence of Mn^{++} food may turn into fat without giving the body the necessary energy. PLUMLEE et al. (1956) have found that a deficiency of Mn^{++} causes excessive fatness in pigs.

The chief activating metal of anaerobic oxidation, i.e. of glycolysis (5 Embden-Meyerhof pathway), in carbohydrate metabolism is Mg^{++}, which is mentioned as being the sole activator in four reaction stages and as replaceable by Mn^{++} in four other reaction stages. In the glycolysis, approximately 10 % of the hexose energy is released in the form of ATP, and the end result is pyruvate (4). During the further oxidation of pyruvate from acetyl-CoA, the reactions may divide into the Krebs cycle and the fatty acid spiral, or from pyruvate directly into amino acid synthesis (14) and lactic acid (7) or alcoholic fermentation (8). In the break-down of alcohol, Zn^{++} is the activating metal and in the break-down of lactic acid Mn^{++} in microbes (MENGER 1967). If a metabolic energy supply is available, mammalian liver and kidney can synthesize glucose from short-chain precursors like lactate and in resting skeletal muscle phosphorylated threecarbon compounds are similarly converted back to glycogen at the expense of creatine phosphate (MAHLER and CORDES 1971).

If the oxidation pathway of pyruvate to the Krebs cycle by way of acetyl-CoA is impeded, it would seem possible that the formation of fatty acids would begin to increase, as might also lactic acid and alcoholic fermentation and the further use of lactic acid and alcohol. When it is recalled that some three-quarters of the ATP energy produced by the glycogen molecule and perhaps an important part of the GTP energy are obtained from the Krebs cycle and the respiratory chain; and only a little by means of glycolysis, it is natural to think that the cell, which has to increase the glycolysis for the formation of ATP, does not use energy efficiently.

Energy is indeed also obtained from the direct oxidation of glucose (9), but only as NADPH. One of the activating elements of this cycle also is Mn^{++}, at one reaction perhaps the sole activating metal, and in another alternatively with Fe^{++} or Mg^{++}.

Generally, the production of polypeptides and enzymes takes place in the cell, programmed by DNA with the aid of RNA. In these reactions Mg^{++} and Mn^{++} are mentioned as activators, GTP as the provider of the coupling energy for the codes between the messenger RNA (mRNA) and the transfer RNA (tRNA), and ATP in the other events of coupling and release (BOUTER 1970). The cell builds ribose nucleotides and, from these, RNA and through deoxygenation DNA (10) out of amino acids and the intermediates of direct glucose oxidation (9).

The DNA is assumed chiefly to regulate its own replication as well as the production of the cell RNA and thus also that of enzymes and other cell proteins, but the functions of DNA itself involve several intracellular regulating systems. In the cell, the production, for instance, of specific enzymes may be dependent either on the abundance of various other enzymes or the presence of a substrate, such as lactose, or on the effector repressor system or plasma factors such as hormones or proteins which surround the DNA. In the vicinity of the RNA-forming DNA there are acid proteins, i.e. "chromosome puffs" or "Balbiani rings", and alkaline histones surrounding inactive gaps. The digestive enzymes of DNA, RNA and other specialized cell-building components are in the lysosomes of the cell. As reviewed by FROST (1970), selenium may participate in the final stages of protein biosynthesis, and protect the lysosomal membranes (TAPPEL and CLADWELL 1967).

Discussion I: Cancer research and the activators of cell metabolism

Injury to cell respiration in man, according to WARBURG (1956), occurs in the form of an increase in lactic acid fermentation, from its being almost unused to supplying nearly half the ATP energy. Cancer in rats develops more quickly, because some of the energy in rats is

produced by lactic acid fermentation even under normal conditions. The ATP production of the respiratory chain differs from the ATP production of glycolysis in that it is associated with the highly developed structure of the cells. The ATP of glycolysis is formed in the fluid cytoplasm and is found in the very earliest stages of embryonic development. Thus the cancer cell, which produces perhaps half of each, could be a result of cell divisions in which the development is reversed, as it were. According to WALLACH (1969) restriction of the normal oxidation path of pyruvate causes the "Warburg phenomenon", i.e. increasing aerobic glycolysis, and an increase in lactic fermentation. As the malfunction in the Krebs cycle further reduces the acquisition of structural parts, it would be understandable for this reason, too, that the activities of DNA and RNA may be turned in a degenerative direction. HUEBNER et al. (1970) actually suggest that embryonic development might be controlled by genes which later in life would act as determinants of cancer. One cause of a faulty direction of development, for instance, may be a shortage of GTP energy, the formation of which is mentioned in connection with the Krebs cycle. It is needed also in protein synthesis, in coupling tRNA to mRNA in the ribosome. If this coupling does not occur, faulty couplings or loose ribosomes and RNA may be expected in consequence. WEBB et al. (1964, 1965) found that in cancer cells the proportion of ribosomes which are membrane-bound decreases from the 60—70 % in normal adult liver to the 20—0 % in various hepatomas. According to OHE et al. (1967) the number of mitochondria had actually decreased in cancer cells from 19—33 mg/g to 9—14 mg/g. In some of the experiments by EMMELOT and BOS (1956) the mitochondria of the cancer cell did not oxidize the pyruvates; some of these mitochondria actually hindered the utilization of oxygen by the mitochondria of the liver. BALO and BANGA (1957) investigated more than 50 metal compounds that might act as inhibitors of oxidation. They found that the ascorbic acid complex of Fe prevented the utilization of oxygen in the carcinoma of mice and rats, while that of cadmium caused an increase in it but the less toxic malate complex of Mn actually prevented the growth of the carcinoma.

The results of these investigations prompt the idea that the question might also be one of atrophy of the mitochondria after manganese has been absent from their membranes for an extended period of time, and that at an early stage the condition of the mitochondria could still be restored by means, say, of manganese.

MEDIGRECEANU (1913) found that there is a relative paucity of manganese in the tumors of mice and rats. They usually contain less manganese than 0.010 mg/100 g of fresh tumor tissue, varying between 0.004 and 0.012, while the normal tissue of a mother rat contains some 0.02 mg, its kidneys 0.063—0.238 and its liver 0.265—0.416 mg in fresh tissue.

The behaviour of manganese in metabolism, the partial possibilities of replacing it and especially of using it instead of other metals, as well as its susceptibility to leave the mitochondria may have impeded the recognition of its significance. In the reactions of the Krebs cycle, however, it is more important as an activating metal than is Mg, and in one section associated with the urea cycle (13) it is actually the only activating metal that has been mentioned. As the Krebs cycle produces intermediates, both for amino acid synthesis and for the respiratory chain, the significance of manganese is obvious although magnesium is otherwise the primary activating metal for cell metabolism. In spite of its being quite abundant, quantities of Mn obtained in food are small when compared with those of Mg, and the uptake of manganese from the soil by plants varies (ØDELIEN 1945) much more than does that of magnesium (LAKANEN 1969). This variability is presumably connected with the special replacing value of manganese.

Magnesium is clearly the most common activating metal of cell metabolism. It is also a component of chlorophyll in plants. A deficiency of magnesium is related to the incidence of cancer in many investigations (ROBINET 1930, 1934, DELBET 1934, FAVIER 1951, TROMP 1954,

VOISIN 1959). It was observed in connection with the investigation of MARJANEN (1969) that the highest magnesium content in the soil had not prevented an increase in the incidence of cancer in areas where there was a distinct deficiency of manganese. A distinct shortage of magnesium did seem to cause an increase in the incidence. According to VIRTANEN (1959 b and c), HEGGTVIET (1969) and NIEPER (1970) the significance of magnesium is important in preventing heart attacks. A deficiency of it can be assumed to cause an increase in the susceptibility to diseases of the heart and the blood circulatory system. It may have a concurrent effect upon cell activities. Though there is always some magnesium in products of the vegetable and animal kingdoms, its relative quantity is dependent on the amount of Mg available to the plant through soil and also on the balance of other plant nutrients (VOISIN 1959, RASMUSSEN 1964, MÄNTYLAHTI and MARJANEN 1971).

According to UNDERWOOD (1971), selenium is important particularly in the respiratory chain (1) in connection with coenzyme Q and perhaps in the transfer of hydrogen. It is also needed in the absorption of vitamin E and, together with this vitamin, in the metabolism of fats (6) (UNDERWOOD 1971), as a possible unstable linkage between SH groups and together with them in cell adhesion (FROST 1970), as a membrane protector in lysosomes (TAPPEL and CLADWELL 1967), as an enzyme catalyst (TUPPY 1959) and as a substitute for or antagonist of sulphur (FROST 1972). It is possible that selenium may function also in connection with the mitochondrial membrane-associated enzymes mentioned by WALLACE (1969), between the glycolysis and the respiratory chain, as a transporter of intermediates, perhaps in the same way as LINDBERG and ERNSTER (1954) describe the functioning of manganese.

In addition to what FROST (1970 and 1971) mentions about selenium as a cancer-resisting factor, it can moreover be shown that the Se content of the blood of cancer patients has, according to SHAMBERGER et al. (1971), been on average clearly lower than that of other patients. NORDMAN (1971) mentions that selenium isotopes make their way to cancer tumors and especially to the most malignant parts of these. Selenium is needed as a membrane protector to correct any adhesion deficiency in cancer cells (FROST 1970, TAPPEL and CLADWELL 1967, ABERCROMBIE 1967) and to enhance the biosynthesis of ubiquinones needed for host-defense mechanisms (HELLER 1971).

The blood serum of a person suffering from cancer has been found to contain a lower amount of SH groups than does the blood serum of a normal person, and this phenomenon is indeed so characteristic that STRICKS (1953) suggested quantitative determination of the SH groups in the serum as a diagnosis of cancer. WOOD and KRANYAK (1953) found that benzpyrene injections greatly reduced the sulfhydryl group content of the blood serum, and later, RONDONI (1955) concluded that it is precisely because they prevent the activity of the SH groups of certain enzymes that benzpyrenes are carcinogenic. FROST (1970) mentioned investigations on mice, in one of which the disulphide, cystine, caused an increase in the susceptibility to spontaneous or induced cancer (WHITE et al. 1947) and another in which cysteine with free SH groups, caused a decrease in spontaneous cancer and an increase in the lifespan of mice (HARMAN 1961). The supply of SH groups in man is associated with the SH groups contained in the sulfur amino-acids. That supply is not always certain. For example, the explicit purpose of additives mixed with flour to improve the baking properties of wheat bread in Finland is to oxidize almost half the SH groups of the flour into -S-S-bonds. According to BLOKSMA (1964) and TIPPLES (1967) a high level of oxidation is advantageous when high-speed baking machines are used. The hulling of the grain itself removes a large part of the protein with SH groups (NEUMAN and PELSHENKE 1954).

REVICI (1955) found a dualistic pattern in malignant tissue, a predominance of sterols in the terminal phase causing low sulfhydryl excretion, and a predominance of fatty acids

causing an increase in sulfhydryl excretion. As the need for SH groups seems to be smaller on the reaction line of glycolysis-lactic fermentation than on the line glycolysis acetyl-CoA fatty acid spiral, it is conceivable that the choice of reaction line when the Krebs cycle becomes restricted would depend on the supply of the SH groups and selenium or other activators. The variations in the excretion of SH groups could, again, be a consequence of the line along which the reactions are directed at any given time.

Iron is an important mineral in the respiratory chain (1) and is one of the primary activators both in glycolysis and in the direct oxidation of glucose (9). True, it can be obtained in quantity from products of the vegetable kingdom because it is indispensible to plants both for their photosynthesis and for the formation of nitrogenous substances, but a deficiency of iron occurs both in plants and in animals. In man, however, iron deficiency is rather easily perceived as anemia. Steps are usually taken to control anemia at a relatively early stage. A linkage has been established between the utilization of iron and the level of manganese in the body (COTZIAS 1958), and so has the significance of copper, cobalt and vitamin B_{12} in connection with manganese (HOWELL and DAVISON 1959, VIRTANEN 1959 a).

Copper is a component (HOWELL and DAVISON 1959) of the cytochrome oxidase of the respiratory chain (1) but it is also needed in the protein synthesis of plants. It also has other functions in animal cells (SCHÜTTE 1964). In several studies an increase in the copper content of the blood has been established in cancer patients, while the quantities of iron have decreased except in cases of acute leukemia (KEIDERLING and SCHARPF 1953). The excessive rise in copper in the blood may, for instance, be due to non-utilization of it in the respiratory chain. A deficiency in copper has been found to cause a disturbance in the metabolism of the SH groups of the wool cells of sheep and to cause paralysis in lambs and calves, and in Australia, ataxia of cattle (BERGMANN 1968) as well as anemia (GALLAGHER et al. 1956). In the early stages of the present study MARJANEN (1969) observed together with a deficiency in copper a relatively high incidence of cancer in a few areas in Finland where the manganese deficiency alone was not particularly severe.

According to ROSE (1968) there is a shortage of zinc in cultivated plants of the cancer regions of the Transkei. According to HYPPÖLÄ (1966) symptoms of zinc deficiency in animals in Finland are common, and when animals with such a deficiency have been given supplementary fodder containing Mg the symptoms of zinc deficiency have been aggravated and the disturbances in the reproductive capacity have shown a sharp increase. The significance of zinc for the maintenance of health is understandable, for zinc regulates the activity of the ribonuclease of cells (UNDERWOOD 1971) and is an activator in protein synthesis (12). It also acts in alcoholic fermentation (8) and as a replacement for other minerals in the urea cycle (13) and in glycolysis (5).

Molybdenum is mentioned as the activator in the nitrate reductase of plants (11). A deficiency of molybdenum causes malfunctions in the protein synthesis of plants. If, moreover, it occurs together with a deficiency of manganese, the nitrate content of the plant will be on the borders of toxicity. Also, phosphorus does not bind normally with organic matter: for instance, phosphorus from silage fodder fed to animals in Finland flows off with the expressed juice (MARJANEN 1972). It might be asked whether molybdenum might have an indirect effect on the phosphorus metabolism of man as well. In ruminants, molybdenum has been found to have a great effect on copper metabolism. Evidently, molybdenum and sulphate, in quantities that are either too great or too small, will impede the utilization of copper by preventing the tissues from receiving it or by increasing the excretion of copper, or in both of these ways (WOHL and COODHART 1964). Both CLEMMESEN (1965) and ROSE (1968) mention a deficiency of molybdenum as being a possible cause of susceptibility to cancer.

Vitamin deficiencies have frequently been

found in cancer patients, but even these seem to be connected with mineral supply. For example CLEMMESEN (1965) and QUISENBERRY (1961 a) mention a deficiency in vitamin B_1 and it has been found that manganese is metabolically related to vitamin B_1, i.e. thiamine (HILL and HOLTKAMP 1953). The storage of manganese in the liver is dependent upon the amount of thiamine in the diet, and conversely it appears that the storage of thiamine is dependent on the level of dietary manganese. As boron fertilization has been found to promote the formation of plant thiamine (LYON and BEESON 1948), a shortage of boron, too, can be regarded as being a factor indirectly causing an increase in the susceptibility to cancer (ROSE 1968, KMET and MAHBOUBI 1972).

In association with cancer CLEMMESEN (1965) also mentions vitamins A and C. According to SCHÜTTE (1964) increased dietary manganese promoted the synthesis of vitamin C in rat livers. The same has been shown for plants: it is possible to bring about an increase in the content of vitamin C by means of fertilization with manganese (ERKAMA 1947, SCHARRER and WERNER 1957). The quantities of vitamin A and carotin (provitamin A) have been observed to vary with the quantities of manganese and copper in plants (ERKAMA 1947, BURGER and HAUGE 1951, SCHÜTTE 1964).

Vitamin B_{12} deficiency may increase cancer (CLEMMESEN 1965, ROSE 1968) while it can cause malfunctions in nucleotide reactions. Vitamin B_{12} is important as an activating element when the DNA formation reactions depart from the RNA reaction pathways. Utilization of vitamin B_{12} is dependent on manganese, and cobalt is a constituent part of it. In cell metabolism, cobalt seems able to replace at least iron, magnesium and manganese in some of the reaction stages.

The significance of vitamin E, i.e. tocopherols, has been mentioned together with that of selenium in the synthesis of fatty acids; biotin is mentioned in connection with malonyl-CoA synthesis (3—6); pantothenic acid in the formation of coenzyme-A; riboflavin in that of $FADH_2$; and niacin in that of NADH and NADPH (AMBROSE and EASTY 1970).

The relations between mental factors and bodily health have been noticed ever since 1402 or earlier according to LESHAN (1959), and a quite obvious dependence has been established. Generally, the consequence of unbalanced emotions has been apparent as disturbances in the balance of the metabolism. For instance, a cancer patient next to a dying person has shown changes in the excretion of sulfhydryl groups, in surface tension and specific gravity of urine (LESHAN 1959) and slight increases in the serum acid-phosphatase level (TRUNNELL 1956). On the other hand, it has been both suggested and stated that changes in manganese concentration in the body fluids are associated with some neurological reactions (MEIRI and RAHAMIMOFF 1972).

The results of animal tests in which cancer is induced by means of unbalanced feeding and, conversely, is controlled by means of various additives in the diet (WHITE et al. 1964, HARMAN 1961), clearly suggest the importance of balance and imbalance caused by diet in the resistance and susceptibility to cancer. On this basis we can also interpret the illegal and penalized, but nevertheless significant, experiment done by a physician and mentioned by MÄKELÄ, O. (1967). Tissue-cultivated cancer cells were transplanted in some patients. These cells even formed metastases in persons previously suffering from cancer, but always died in patients with other difficult conditions.

Mineral and vitamin imbalances may impede both the formation of primary metabolites and the energy supply of the couplings. It can be assumed that divergent nucleotides and their enzymes may then be formed. Intracellular ratios such as the K^+/Mg^{++} ratio are important in ordinary protein synthesis. They may function as regulators insofar as, for instance, various concentrations of Mg^{++} may cause the formation of various proteins (MÄKELÄ, P. 1967, AMBROSE and EASTY 1970). It has probably not been established whether the deficiency in GTP energy, previously mentioned, is associated with

the membrane connection of the polysomes or merely with the coupling of mRNA and tRNA in the ribosome. It can be assumed, however, that conditions in the cell gradually change on account of various combined factors. Then the genes, which have remained inactive in the normal state of the cell, may become activated (HUEBNER et al. 1970), the activity ratios of the active forms of DNA may alter, or quite unusual nucleotides may be formed.

ATTARDI and ATTARDI (1967) investigated malignant HeLa cells and found that only 10—15 % of the polysomes are membrane bound as opposed to the normal 60—70 %. The base composition and the metabolic behaviour of these are distinct from those of the mRNA of free polysomes, and the RNA of the latter are synthesized on a mitochondrial DNA template. OKER-BLOM (1967) mentions both RNA and DNA as nucleic acids of tumor viruses. The behaviour of RNA and DNA of tumor viruses differs from the behaviours of the respective compounds of both ordinary viruses and the healthy organism. The tumor viruses resemble other viruses in being transmittable.

A characteristic of cells is that, when necessary, they are able to form new enzymes. For instance, microbes using glucose may, when supplied with lactose, quickly form enzymes that are capable of breaking down lactose which they did not previously possess (MENGEL 1968). If, for instance, there is a deficiency of vitamin B_{12} the formation of DNA is impeded (AMBROSE and EASTY 1970). Too much RNA may accumulate and it is conceivable that such an excess may in itself form an enzyme of its own. It may be similar, for example, to an enzyme used at the embryo stage, or it may be a new enzyme induced exclusively by the situation of malfunction. BALTIMORE (1970) and TEMIN and MIZUTANI (1970) have studied the Rauscher mouse leukemia virus which causes cancer in animals, and also the Rous sarcoma virus. They arrived at the conclusion that the information carried by the infecting RNA is transferred to a DNA copy which then serves as a template for the synthesis of viral RNA. This model requires a unique enzyme, an RNA-dependent DNA-polymerase. No such enzyme was present in supernatants of normal cells, but the experiments of TEMIN and MIZUTANI (1970) show that the Rauscher mouse leukemia virus and Rous sarcoma virus contain RNA-dependent DNA-polymerase. According to LEVISON et al. (1972), this polymerase is important in the reproduction of the virus, while the DNA of the tumor is not.

If imbalances are generally considered to be causes of cancer, the appearance of cancer should vary a great deal, as do malfunctions generally. Thus STEEL (1971) found that cancer cells in man are quite heterogeneous in terms of both their growth and their reproduction. The impeding of the Krebs cycle and the respiratory chain, for various reasons, may cause differences in the amounts of activators left unused, eq. Cu, and which can in some cases be further used in replacing others like Mn and Se. ŠEVČENKO and PANKOV (1970) have found accumulation of Cu and Mn in bone tumors and the accumulations of Cu has been much the greater so that the Cu:Mn ratio, normally $0{,}_4$ to $0{,}_6$ in bone rose to the range of $2{,}_2$ to $2{,}_9$ in human bone sarcoma. SHAMBERGER et al. (1972) have found low blood selenium values in males and females with gastrointestinal cancer or metastases in gastrointestinal organs and in patients with Hodgkins disease, but patients with carcinoma of the breast, most blood cancers, Crohn's disease, certain sarcomas and patients with rectal carcinoma had normal values of blood selenium. If, again, cancer is considered to be a virus disease, then YAMAFUJI (1964) may be correct in his conclusion that virus diseases can be regarded as consequences of specific nutritional or metabolic disturbances.

KUFE et al. (1972) established that human sarcomas contain RNAs that can hybridize with DNA homologous to the RNA known to cause sarcomas in mice. Human breast carcinoma RNA is unrelated to the RNA of mice leukemia but is homologous to the RNA that causes breast tumors in mice. On the other hand, human leukemia shows a unique homology to

the virus that causes leukemia in mice. In contrast, none of the RNAs from normal adult and foetal tissues give a reaction that could be designated as positive with sarcoma and leukemia RNA. It is thus conceivable that the quality of the imbalances and perhaps the genetical basis, e.g. the blood group (QUISENBERRY 1961 b), might determine the direction of the faulty reactions insofar as the equivalent diseases of animals and of men are more or less of the same type.

Discussion II: Cancer, the activators of cell metabolism and soil mineral resources in Finland

The economic results of farming have been improved by heavy fertilization of the soil. Farmyard manure and various milled natural products previously used contained some amounts of all the requisite minerals. Lime was the first substance of which the one-sided use was shown to have disadvantages. The good economic results obtained with three of the major plant nutrients (N, P and K) prompted the development of products of the chemical industry containing these substances alone for the purpose of fertilization. The consequences have materialized as various deficiency symptoms in plants and animals. Scientific investigation has consequently charted the deficiency, optimum and excessive quantities of numerous minerals in order to determine the fertilization requirements of plants and mineral requirements of animals. The human diet is varied so widely that it has not been easy to recognize the phenomena of deficiency in man.

Experimentally induced deficiency cancer in test animals does not prove that similar cancer incidence in man could be caused by the same deficiency. Recent research in cell biology has made it possible to assume that, just as in other animals, the absence of the necessary activators or a malfunction in their balance in man may cause malfunctions that induce actual illness.

In the present study an examination was made of the literature concerning the way in which plant nutrients described in connection with susceptibility to cancer may be concerned in the matter in terms of cell biology.

A deficiency especially of Mn and Se and accompanying deficiencies of Cu, Mg, Ca, P or SH groups may cause a malfunction in cell respiration. Of these, manganese deficiency may cause a restriction of the highly essential Krebs cycle and also impede the formation of proteins and especially of nucleotides. A deficiency of selenium hinders the reactions of the respiratory chain itself but also disturbs the functions of structural membranes and of many enzymes containing SH groups.

Deficiencies of some of the essential activators or the malfunctioning of intracellular relations may lead to a shortage of the energy requisite, or to divergent orientations of the reaction pathways. Genes that have controlled embryonic development may begin to function again (HUEBNER et al. 1970). Defective forms held to be viruses may be formed out of nucleotides. These defective forms in turn may create defective enzymes and finally form various tumors, perhaps even differing according to the type of malfunction in balance.

Psychological states in man differ in their nature from the nutrition of the body, but a deficiency in the balanced production of energy in the body easily drives the mental reactions in an unfavourable direction, and the converse may also occur, so that imbalances may be intensified in various ways.

In recent years deficiencies in the amounts of:

Mn (JAMALAINEN 1963, KURKI 1963, MARJANEN 1969),
Mg (HAARANEN 1970, MÄNTYLAHTI and MARJANEN 1971, KURKI 1972),
Se (OKSANEN and SANDHOLM 1970),
Cu (TAINIO 1945, KURKI 1963),
B (JAMALAINEN 1935, HÄNNINEN 1958, KURKI 1963, 1972, SIMOJOKI 1970),
Zn (HYPPÖLÄ 1966),
Co (LAKANEN and MARJANEN) and
Mo (MARJANEN 1972)

obtained from plants and animals have been ascertained in Finland. Their amounts, as well as those of SH (NEUMAN and PELSHENKE 1954) and Fe, have been further reduced, for instance by the hulling of grain (KOIVISTOINEN 1971, KOIVISTOINEN et al. 1970). In particular, Finnish consumers of sugar, white bread and sausages have obtained ample quantities of proteins and carbohydrates without obtaining the proportionate activating elements, SH groups and vitamins B_{12}, B_1, C, A and E for example, and have thus become susceptible to infectious diseases and particularly to malfunctioning of the cells. The recent very abundant harvests produced by dressings of N, P, K and Ca have exhausted reserves of the mentioned subsidiary and trace nutrients in cultivated soils. The increasing quantities of some nutrients and the decreasing quantities of others in soils (KURKI 1972), in plants and consumers of plants have brought about general imbalances, which are difficult to diagnose. According to FORSSÉN (1972) mineral contents of the organs of the human body in Finland are different from those in the U.S.A., Africa, the Middle East and the Far East. The quantities of Mn are on average distinctly lower in Finnish people than among others, and the quantities of Cu and Cd frequently so. Determinations of Se, Mg and P have not been made, but the averages for the quantities of K are clearly higher in Finnish people. The author points out that the question might be one of a systematic error, but the overly high quantities of K may also be a consequence of Mg deficiency in Finnish cultivated soils, or of the luxury consumption of potassium given in the form of fertilizer, with the resulting excess of K in the Finnish diet (KOIVISTOINEN et al. 1970). The quantities of Zn have been perhaps a little lower in Finnish people than in others in some cases, but distinctly abundant in the pancreas, which again may be due to the use of zinc as a substitute for manganese. Here might be a connection with the association of zinc excesses and the incidence of cancer (HALME 1968). Investigations of the regional incidence of cancer in other countries support the opinion that cancer may be a deficiency disease.

Acknowledgements. — This article is a survey of literature for the preliminary report by MARJANEN (1969) "Possible causal relationship between the easily soluble amount of manganese in arable mineral soil and susceptibility to cancer in Finland." The second author assisted in the collection of certain data from the literature. The authors wish to thank Dr. Douglas V. Frost, Brattleboro, Vt., U.S.A. for much information concerning the role of selenium in cell metabolism and cancer.

REFERENCES

ABERCROMBIE, M. 1967. Contact Inhibition: The Phenomenon and its Biological Implications. Discussion. Second Decennial Review Conference on Cell Tissue and Organ Culture. Nat. Cancer Inst. Monograph. 26: 249—273.

ALGALDE BLANCO, S. 1969. Physiologische und Chemische Untersuchungen über den Einfluss der Manganernährung auf die Beziehungen zwischen Wachstum und Chlorophyllgehalte bei Phaseolus vulgaris im Sprühkulturverfahren. Dissertation 122 p. Göttingen.

AMBROSE, E. J. & EASTY, D. M. 1970. Cell Biology. 500 p. London.

ATTARDI, B. & ATTARDI, G. 1967. A membrane-associated RNA of cytoplasmic origin in Hela cells. Proc. Nat. Acad. Sci. (Wash.) 58: 1051—1058. (Ref. Wallach, D. F. H. 1969.)

BALO, J. & BANGA, I. 1957. Effect of metal complexes upon experimental carsinoma. Acta U.I.C.C. 13: 463—465.

BALTIMORE, D. 1970. Viral RNA-dependent DNA Polymerase. Nature 226: 1209—1211.

BEEVERS, L. & HAGEMAN, R. H. 1969. Nitrate reduction in higher plants. Plant Physiol. 20: 495—592.

BERGMANN, W. 1968. Die Bedeutung der Mikronährstoffe in der Lantwirtschaft. 121 p. Berlin.

BLOKSMA, A. H. 1964. Oxidation by potassium iodate of thiol groups in unleavened wheat flour doughs. Reprint: J. Sci. Fol. Agric. 15, 2: 83—94. Wageningen.

BOUITER, D. 1970. Protein synthesis in plants. Plant Physiol. 21: 91—109.

BURGER, O. J. & HAUGE, S. M. 1951. Relation of manganese to the carotene and vitamin contents of growing crop plants. Soil Sci. 72: 303—313.

CHAPPELL, J. B., COHN, M. & GREVILLE, G. D. 1963. Energy Linked Functions of Mitochondria. Chance, B., Ed., Academic, p. 219—235. New York.

CLEMMESEN, J. 1965. Statistical studies in malignant neoplasms. 543 p. Munksgaard, Copenhagen.

COTZIAS, G. C. 1958. Manganese in Health and Disease. Physiol. Rev. 38, 4: 503—532.

COWAN, J. C. 1971. Personal communications. (Ref. Selenium in nutrition. National Academy of Sciences. 79 p. Washington).

DELBET, P. 1934. B. de l'Académie Nationale de Médecine 111, 393. (Ref. Voisin, A. 1959.)

—& ROBINET, L. 1934. B. de l'Académie Nationale de Médecine 111, 415. (Ref. Voisin, A. 1959.)

EMMELOT, P. & BOS, C. J. 1956. Enzymatic activities of tumor mitochondria as demonstrated by their inhibitory effect on the oxidative response of liver mitochondria towards fatty acids and pyruvate. Enzymologia Acta Biocatalytica 18: 149—178.

ERKAMA, J. 1947. Über die Rolle von Kupfer und Mangan im Leben der höheren Pflanzen. 90 p. Helsinki.

FAVIER, J. E. 1951. Equilibre Minéral et Santé. Paris. (Ref. Voisin, A. 1959.)

FORSSÉN, A. 1972. Occurrence of Ba, Br, Ca, Cd, Cs, Cu, K, Mn, Ni, Sn, Sr, Y and Zn in the human body. 70 p. Helsinki.

FROST, D. V. 1970. Tolerances for arsenic and selenium: a psycodynamic problem. World Rev. of Pest Control 9, 1: 6—28.

— 1971. The Case for Selenite as a Feed Additive. Feedstuffs 43, 31: 12.

— 1972. Selenium has great nutritional significance for man, animals; Should be cleared for feed. Feedstuffs 44, 4: 8, 58—59.

GALLAGHER, C. H., JUDAH, J. D. & REES, K. R. 1956. The Biochemistry of Copper Deficiency. I The Enzymological Disturbances, Blood Chemistry and Excretion of Amino Acids. Proc. Roy. Soc., B 145, 134—150 (Ref. Schütte, K. H. 1964.)

HAARANEN, S. 1970. Havaintoja magnesiumista nautakarjan ruokinnassa. Sementtiyhd. Tied. 15—20.

HAKAMA, M. & SAXÉN, E. A. 1967. Cereal consumption and gastric cancer. Intern. J. Cancer 2: 265—268.

HALME, E. 1968. Käsitykseni syövän synnystä IV. TEE (Terveiden Elämäntapojen Edistämistoiminnan Yhteislehti) 2: 14—18.

HARMAN, D. 1961. Prolongation of the normal lifespan and inhibition of spontaneous cancer by antioxidants. J. Geront. 16: 247—54. (Ref. Frost, D. V. 1970.)

HELLER, J. H. 1971. New England Institute Report 70: 71, Ridgefield, Conn. (Ref. Frost, D. V. 1972.)

HEGGTVIET, A. 1969. Magnesium in ischaemia is recommended. Medical Tribune 20. 2. 1969, p. 2.

HIGGINSON, J. 1967. Panel question. (Ref. Saxén, E. A. & Hakama, M. 1967.)

HILL, R. M. & HOLTKAMP, D. E. 1954. J. Nutrition 53: 73. (Ref. Cotzias, G. G. 1958.)

HOWELL, J. McC. & DAVISON, A. N. 1959. Copper and Cytochrome Oxidase in Swayback: Biochem. J. 72: 365—8. (Ref. Schütte, K. H. 1964.)

HUEBNER, R. J., KELLOFF, G. J., SARMA, P. S., LANE, W. T., TURNER, H. C., GILDEN, R. V., OROSZLAN, S., MEIER, H., MYERS, D. D. & PETERS, R. L. 1970. Group-specific Antigen Expression during Embryogenesis of the Genome of the C-type RNA Tumor Virus: Implications for Ontogenesis and Oncogenesis. Proc. Nat. Acad. Sci. 67, 1: 366—376.

HYPPÖLÄ, K. 1966. Interrelationship of zinc and magnesium in cattle. Maatal.tiet. Aikak. 60: 180—182.

HÄNNINEN, P. 1958. Boorista ja sen käytöstä puna-apilalla. Summary: Results of boron treatment on red clover-timothy leys. Maatal. ja Koetoim. 12: 235—249.

JAMALAINEN, E. A. 1935. Tutkimuksia lantun ruskotaudista. Referat: Über die "Ruskotauti" Krankheit der Kohlrübe. Valt. Maatal.koetoim. Julk. 72: 1—116.

— 1963. Boorin, mangaanin ja kuparin puutteesta aiheutuvista kasvitaudeista. Maatalous 29: 308—310, 312.

KATALYMOW, N. W. 1969. Mikronährstoffe-Mikronährstoffdüngung. 296 p. Berlin.

KEIDERLING, W. & SCHARPF, H. 1953. Über die klinische Bedeutung der Serumkupfer- und Serumeisenbestimmung bei neoplastischen Krankheiten. Münch. Med. Wschr. 95: 437—439.

KOIVISTOINEN, P. 1971. Viljan asema valtakunnan elintarvikehuollossa. Symposiumi Leipä 70 alustukset. p. 6—21. Helsinki.

—, AHLSTRÖM, A., NISSINEN, H., PEKKARINEN, M. & ROINE, P. 1970. Mineral element compositions of Finnish diets. Part 1: Fe, Cu, Mn, Zn, Mg, Na, K, Ca and P. Suom. Kemistilehti — The Finn. Chem. J. 43, 11: 426—430.

KMET, J. & MAHBOUBI, E. 1972. Esophageal Cancer in the Caspian Littoral of Iran: Initial Studies. Detailed studies of geographica pathology are opening new vistas in cancer epidemiology. Science 175, 4024: 846—853.

KRETOVICH, W. L. 1965. Some problems of aminoacid and amino Biosynthesis in plants. Ann. Rev. Pl. Physiol. 16: 141—154.

KUFE, D., HEHLMANN, R. & SPIEGELMAN, S. 1972. Human Sarcomas Contain RNA Related to the RNA of a Mouse Leukemia Virus. Science 175, 4018: 182—185.

KURKI, M. 1963. Suomen peltojen viljavuudesta. Viljavuuspalvelu Oy. 107 p. Helsinki.

— 1972. Suomen peltojen viljavuudesta II. Über die Fruchtbarkeit des finnischen Ackerbodens auf Grund der in den Jahren 1955—1970 durchgeführten Bodenfruchtbarkeitsuntersuchungen. Viljavuuspalvelu Oy. 182 p. Helsinki.

LAKANEN, E. 1969. Viljan kivennäisainekoostumuksesta. Koetoim. ja Käyt. 26, 8: 31.

—& MARJANEN, H. unpublished data.

LESHAN, L. 1959. Psychological states as Factors in the Development of Malignant Disease: A Critical Review. J. Nat. Cancer Inst. 22: 1—18.

LEVISON, W. E., VARMUS, V. E., GARAPIN, A-C. & BISHOP, Y. M. 1972. DNA of Rous Sarcoma Virus: Its Nature and Significance. Science 175, 4017: 76—78.

LIEBERMAN, M. & BAKER, J. E. 1965. Respiratory electron transport. Pl. Physiol. 16: 343—382.

LINDBERG, O. & ERNSTER, L. 1954. Manganese, a Cofactor of Oxidative Phosphorylation. Nature 173: 1038—39. Stockholm.

LYNEN, F. 1965. Der Weg der "aktivierten Essigsäure" zu den Terpenen und Fettsäuren. Angew. Chemie 77: 929.

—& DOMAGK, G. F., GOLDMANN, M. & KESSEL, I. 1962. Zur Biosynthese der Fettsäuren. 1. Nachweis von Malonyl CoA als Zwischenprodukt der Fettsäuresynthese in Hefeextrakten. Biochem. Z. 335: 519—539.

LYON, C. B. & BEESON, C. 1948. Influence of Toxic Concentrations of Micro-nutrient Elements in the Nutrient Medium on Vitamin Content of Turnips and Tomatoes. Bot. Gaz. 109: 506—520 (Ref. Schütte, K. H. 1964.)

MAHLER, H. R. & CORDES, E. H. 1971. Biological Chemistry. Second ed. 1009 p. New York.

MARJANEN, H. 1969. Possible causal relationship between the easily soluble amount of manganese on arable mineral soil and susceptibility to cancer in Finland. Ann. Agric. Fenn. 8: 326—334.

— 1972. Tuorerehunurmien molybdeenin tarpeesta. Karjatalous 48, 9: 354—355.

MEDIGRECEANU, F. 1913. Proc. Roy. Soc., B. 86: 174. (Ref. Tromp, S. W. 1954.)

MEIRI, U. & RAHAMIMOFF, R. 1972. Neuromuscular Transmission: Inhibition by Manganese Ions. Science 176, 4032: 308—309.

MENGEL, K. 1968. Ernährung und Stoffwechsel der Pflanze. Dritte, umgestaltete und verbesserte Auflage. 436 p. Jena.

MENGER, J. W. 1967. The manganese content of milk. A discussion on its relation to the bacteriological consumption of starters containing Leuconostoc (*Betacoccus*) cremoris. Nederlands Melk-en Zuiveltijdschr. 21, 2: 67—73.

MÄKELÄ, O. 1967. Immunologinen syöpäterapia. Duodecim 83: 177—183.

MÄKELÄ, P. H. 1967. Ribosomit. Duodecim 83: 1074—1080.

MÄNTYLAHTI, V. & MARJANEN, H. 1971. Tuorerehunurmen lannoitus. Summary: Fertilization of heys for silage. Ann. Agric. Fenn. 10: 153—175.

NEUMAN, M. & PELSHENKE, P. 1954. Brotgetreide und Brot. 622 p. Berlin.

NIEPER, H. A. 1970. Eine fruchtbare Diskussion auslösen. Bunte 48, 23. 11. 1971.

NORDMAN, E. 1971. Syöpäkudokseen hakeutuvat isotoopit. Syövän Torjunta. Kampen mot Kräfta 3, 6: 12—13.

OHE, K., MORRIS, H. P. & WEINHOUSE, S. 1967. B-hydroxybutyrate dehydrogenase activity in liver and liver tumors. Cancer Res. 27: 1360—1371. (Ref. Wallach, D. F. H. 1969.)

OKER-BLOM, N. 1967. Virukset syövän aiheuttajana. Duodecim 83: 184—191.

OKSANEN, H. E. & SANDHOLM, M. 1970. The selenium content of Finnish forage crops. Maatal.tiet. Aikak. 42, 4: 250—253.

PLUMLEE, M. P., THRASHER, D. M., BEESON, W. M., ANDREWS, F. N. & PARKER, H. E. 1965. The Effects of Manganese Deficiency upon the Growth, Development and Reproduction of Swine. J. Animal Sci. 15: 352—67. (Ref. Schütte, K. H. 1964.)

QUISENBERRY, W. B. 1961 a. Castro-intestinal Cancer in Hawaii. Acta U.I.C.C. 17: 324—329.

— 1961 b. Stomach cancer among Japanese in Hawaii and the United States mainland. Acta U.I.C.C. 17: 858—866.

RASMUSSEN, H., FISCHER, J. & ARNAUD, C. 1964. Proc. Nat. Acad. Sci. U.S., 52, 1198. (Ref. Sanadi, D.R. 1965.)

REVICI, E. 1955. The control of cancer with lipids. Based upon a paper presented at the Clinical Pathological Conference. Beth David Hospital, New York City. 11 p.

ROBINET, L. 1930. B. de l'Académie Nationale de Médecine, 103, 440 (Ref. Voisin, A. 1959.)

— 1934. B. de l'Académie Nationale de Médecine, 111, 501. (Ref. Voisin, A. 1959.)

RONDONI, P. 1955. Advances in Cancer Research 3: 171—222. (Ref. Voisin, A. 1959.)

ROSE, E. F. 1968. The Effects of Soil and Diet on Disease. Cancer Res. 28: 2390—2392.

SANADI, D. R. 1965. Energy-linked reactions in mitochondria. Ann. Rev. Biochem. 34: 21—48.

SAXÉN, E. & HAKAMA, M. 1967. The Different Incidence of Gastric Cancer all over the World and Possible Reasons for this Difference. Ninth Intern. Cancer. Congr. U.I.C.C. 10: 49—55.

SCHARRER, K. & WERNER, W. 1957. Über die Abhängigkeit des Ascorbinsäure-Gehaltes der Pflanze von ihrer Ernährung. Z. Pfl. Ernähr. Düng. 77: 97—110.

SCHÜTTE, K. H. 1964. The Biology of the Trace Elements. Their role in nutrition. Intern. Monographs: Aspects of Animal and Human Nutrition. 228 p. London.

SCHWEIGART, H. A. 1962. Vitalstofflehre — Vitalstoff-Tabellarium. 230 p. München.

SEUBERT, W., GREULL, G. & LYNEN, F. 1957. Die Synthese der Fettsäuren mit gereinigten Enzymen des Fettsäurecyclus. Angew. Chem. 69, 11: 359—361.

ŠEVČENKO, S. D. & PANKOV, E. Ja. 1970. Količestvennoe soderžanie medi i marganca v èksperimental' nyh opuholjah zivotnyh i sarkomah kostej čeloveka. (Cu and Mn contents in experimental tumors in animals and in human bone sarcoma.) Bjull. èksp. Biol. Med., 1970, 49, 1: 75—76. English summary. Nutr. Abst.& Rev. 40: 7693.

SHAMBERGER, R. J., TYTKO, S., PRICE, J. W. & WILLIS, C. E. 1971. Concentratio of Selenium in Blood of Normal and Cancer Patients. Clin. Chem. 17, 7: 643.

—, RUKOVENA, E. F. & WILLIS, C. E. 1972. Selenium in the blood of normals and cancer patients. Proc. Amer. Assoc. Cancer. Res. 13: 41.

SIMOJOKI, P. 1970. Lyhtyohra yleistyy. Pellervo 18: 1156—1158.

STEEL, G. G. 1971. Kinetic aspects of solid tumors. Cancer Chemotherapy by Elkerbout, F., Thomas, P. & Zwaveling, A. Leiden p. 16—25.

STRICKS, W., KOLTHOFF, I. M., BUSH, D. G. & KURODA, P. K. 1953. Lancet 73: 328.

STUMPF, P. K. & BRADBEER, C. 1959. Fat metabolism in higher plants. Ann. Rev. Pl. Physiol. 10: 197—222.

TAINIO, A. 1945. Kuparikuonasta ja sen käytöstä. Pellervo 9: 235.

TAPPEL, A. L. & CLADWELL, K. A. 1967. Symposium: Selenium in Biomedicine. AVI Publishing Co., Westport, Conn. p. 345—61. (Ref. Frost, D. V. 1970.)

TEMIN, H. M. & MIZUTANI, S. 1970. RNA-dependent DNA Polymerase in Virions of Rous sarkoma Virus. Nature 226: 1211—1213.

TIPPLES, K. H. 1967. Recent advances in baking technology. Bakers Digest Reprint. 41, 3: 18—20, 24, 26—27, 75.

TROMP, S. W. 1954. Statistical study of the possible relationship between mineral constituents in drinking-water and cancer mortality in the Netherlands (period 1900—1940). British Journal of Cancer 8, 1: 586—593.

—& DIEHL, J. C. 1955. A statistical study of the possible relationship between cancer of the stomach and soil. Brit. J. of Cancer 9, 3: 350—357.

TRUNNEL, J. B. 1956. Theories of the origing of cancer. Paper read at the Texas Conference on Psychosomatic Implications in Cancer. Dallas. (Ref. Leshan, L. 1959.)

TUPPY, H. 1959. Aminosäure-Sequenzen in Protein. Natur-wiss. 46: 35—43.

UNDERWOOD, E. J. 1971. Trace Elements in Human and Animal Nutrition. Third ed. 543 p. London.

VIRTANEN, O. E. 1959 a. Katsaus lääkeainetutkimuksen ja teollisuuden viimeaikaiseen kehitykseen. Suom. Kemistilehti A 32: 16—68.

— 1959 b. Äkillisen sydänlihaskuolion eli ns. sydäninfarktin biokemiasta. Suom. Kemistilehti A 32: 82—83.

— 1959 c. Ravinto ja sydänverisuonitaudit. Suom. Kemistilehti A 32: 280—281.

VOISIN, A. 1959. Soil, grass and cancer. 302 p. London.

WALLACH, D. F. H. 1969. Cellular Membrane Alternations in Neoplasia: A Review and a Unifying Hypothesis. Current Tropics in Microbiology and Immunology 47: 152—176.

WARBURG, O. 1956. On the Origin of Cancer Cells. Science 123: 309—314.

WEBB, T. E., BLOBEL, G. & POTTER, V. R. 1964. Polyribosomes in rat tissues. I. A study of in vitro patterus in liver and hepatomas. Cancer Res. 24: 1229—1237. (Ref. Wallach, D. F. H. 1969.)

— & MORRIS, H. P. 1965. Polyribosomes in rat tissues. II. The polyribosome distribution in the minimal deviation hepatomas. Cancer Res. 25: 1219—1224. (Ref. Wallach, D. F. H. 1969.)

WHITE, J., WHITE, F. R. & MINDER, G. B. 1947. Effects of diets deficient in certain amino acids on the introduction of leukemia in dba mice. J. Nat. Cancer Inst. 7: 199. (Ref. Frost, D. V. 1970.)

WOOD, J. L. & KRAYNAK, M. L. 1953. Cancer Research 13, 358—360 (Ref. Voisin, A. 1959.)

WOHL, M. G. & GOODHART, R. S. 1964. Modern nutrition in health and disease. 1282 p. Philadelphia.

YAMAFUJI, K. 1964. Nutritional Factors in Virus Formation. Intern. Monogr.: Aspects of Animal and Human Nutrition. 128 p. Great Britain.

ZILL, L. P. & CHENIAE, G. M. 1962. Lipid Metabolism. Pl. Physiol. 13: 225—263.

ØDELIEN, M. 1945. Jernmangel på myrjord og koppersulfatets virkning på plantenes jern- og manganforsyning. Særtrykk av Tidsskr. for Det norske Landbr. 3—4: 1—9.

MS received 28 February 1972

Helvi Marjanen and Sylvi Soini
Agricultural Research Centre
Bureau for Local Experiments
Erottajankatu 15—17
SF-00130 HELSINKI 13, Finland

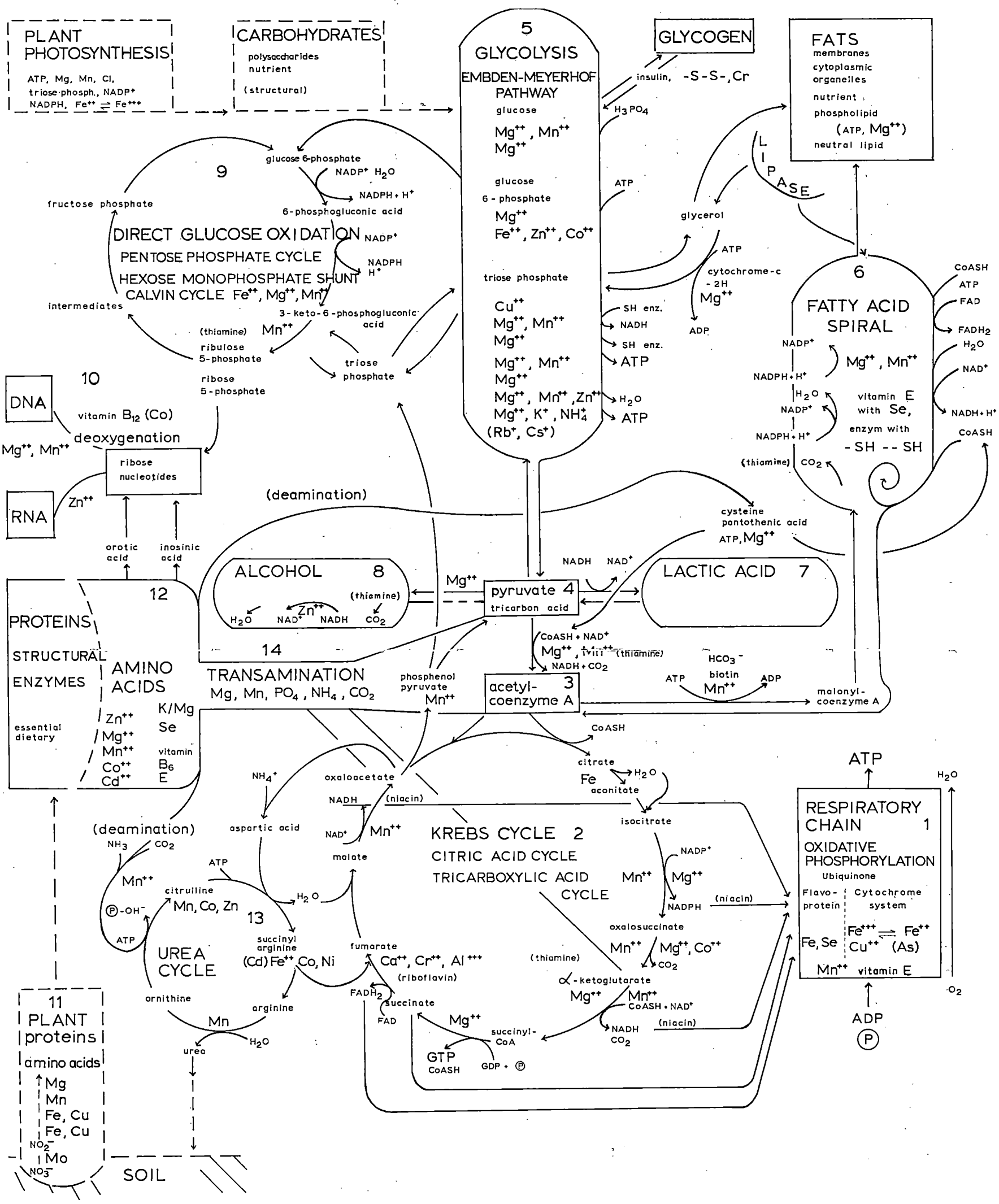


Fig. 1. Simplified assumed collocation of the reaction pathways of the cell, as drawn from the literature. The purpose is to illustrate the most important areas of function of various minerals in cell metabolism. The diagram has been made by combining information from the following sources, mentioned in the bibliography under: Alcalde Blanco (1969), Ambrose and Easty (1970), Beevers and Hageman (1969), Bouiter (1970), Frost (1970, 1972), Katalymow (1969), Kretovich (1965), Lynen (1965), Lynen et al. (1962), Mahler and Cordes (1971), Mengel (1968), Menger (1967), Sanadi (1965), Schweigart (1962), Seubert et al. (1957), Stumpf and Bradbeer (1959), Underwood (1971), Warburg (1956), Zill and Cheniae (1963).

SELOSTUS

Mahdollinen syy-yhteys ravinnetasapainohäiriöiden, etenkin mangaanin puutteen, ja syöpään sairastumisalttiuden välillä Suomessa

HELVI MARJANEN ja SYLVI SOINI

Maatalouden tutkimuskeskus, Paikalliskoetoimisto, Helsinki

Koe-eläimissä yksipuolisella ruokinnalla aiheutettu syöpä ei sinänsä ole riittänyt todistamaan, että ihmisen syöpään sairastuminen voisi samoin olla tulos yhden tai useamman aineen puuttumisesta. Viimeaikainen solubiologian tutkimus on tehnyt mahdolliseksi olettaa, että samaan tapaan kuin eläimillä voivat ihmisilläkin välttämättömien entsyymejä aktivoivien aineiden puuttumiset tai kivennäisaineiden tasapainosuhteiden häiriöt aiheuttaa suoranaisia sairautta aloittavia virhereaktioita.

Tässä katsauksessa on kirjallisuuteen nojautuen tarkasteltu, millä tavoin syöpään sairastumisalttiuden yhteydessä esitettyjen kasvinravinteiden on solubiologisesti mahdollista osallistua asiaan.

Soluhengityksen eli kehon tärkeimmän käyttöenergian tuottajan vaurioitumiseen voivat olla syynä ensisijaisesti mangaanin (Mn) ja seleenin (Se) puute sekä niiden ohella SH-ryhmien, kuparin (Cu), magnesiumin (Mg), kalkin (Ca) tai fosforin (P) puute. Näistä mangaanin puute voi aiheuttaa erittäin keskeisen Krebsin kierron ahtautumisen sekä vaikeuttaa lisäksi proteiinien ja etenkin nukleotidien muodostusta. Seleenin ja kuparin puute ehkäisevät itse hengitysketjun reaktioita. Seleenin puute vaikeuttaa lisäksi hienorakennekalvojen ja monien SH-ryhmäisten entsyymien toimintoja.

Joidenkin välttämättömien mineraalien ja vitamiinien puute tai solunsisäisten suhteiden häiriintyminen voi johtaa tarvittavan energian puutteeseen tai reaktioratojen poikkeaviin suuntautumisiin. Alkiokehitystä säädelleet geenit voivat uudelleen alkaa toimia (HUEBNER ym. 1970). Nukleotideista voi muodostua viroottisina pidettyjä virheellisiä muotoja. Nämä puolestaan rakennuttavat virheellisiä entsyymejä ja lopulta muodostavat erilaisia kasvaimia, mahdollisesti vielä sen mukaan erilaisia, minkä tyyppisiä tasapainohäiriöt ovat olleet.

Psykologiset tekijät ihmisissä poikkeavat olemukseltaan kehon ravitsemuksesta, mutta tasapainoisen energiantuoton puute kehossa painaa helposti sielullisia reaktioita epäedulliseen suuntaan ja päinvastoin niin, että tasapainohäiriöt voivat saada eri tyyppisiä lisäpiirteitä.

Suomessa on viime vuosina todettu vajausta kasvien ja eläinten saamissa ravinnemäärissä: Mn (JAMALAINEN 1936, KURKI 1963, 1972, MARJANEN 1969), Mg (HAARANEN 1970, MÄNTYLAHTI ja MARJANEN 1971, KURKI 1972), Se (OKSANEN ja SANDHOLM 1970), Cu (TAINIO 1945, KURKI 1963, 1972), B (JAMALAINEN 1935, HÄNNINEN 1958, KURKI 1963, 1972, SIMOJOKI 1970), Zn (HYPPÖLÄ 1966), Co (LAKANEN ja MARJANEN) ja Mo (MARJANEN 1972). Nämä samoin kuin SH- (NEUMAN ja PELSHENKE 1954) ja Fe-määrät ovat vielä vähentyneet esim. viljaa kuorittaessa (KOIVISTOINEN 1971). Leipomoteollisuus on leivontatulosta parantaakseen suorastaan pyrkinyt SH-ryhmien hapettamiseen leivontalisäaineita käyttämällä, jolloin muodostuu syöpää lisäävää disulfidia. Etenkin sokerin, valkoisen leivän ja makkaran syöjät ovat Suomessa saaneet runsaita valkuais- ja hiilihydraattimääriä ilman vastaavia entsyymejä aktivoivia kivennäisaineita, SH-ryhmiä sekä vitamiineja (esim. E, B_{12}, B_1, C ja A). Nämä ihmiset ovat olleet alttiita sekä tarttuville taudeille että solujen virhereaktioille. Yksipuolisella pääravinnelannoituksella (N, P, K ja Ca) tuotetut sadot ovat saaneet edellä mainittujen sivu- ja hivenravinteiden varastot viljelysmaista ehtymään. Toisten ravinteiden lisääntyvät ja toisten niukkenevat määrät viljelysmaissa (KURKI 1972) ovat saaneet aikaan vaikeasti todettavissa olevia yleisluonteisia tasapainohäiriöitä sekä kasveissa että niiden edelleen käyttäjissä. Yksinomaan Suomessa eläneiden tapaturmaisesti kuolleiden ihmisten elimissä on todettu (FORSSÉN 1972) mangaanimäärien olevan selvästi ja kadmium- sekä kuparimäärien useissa tapauksissa alempia kuin U.S.A:n, Afrikan, Lähi- ja Kauko-Idän vastaavat analyysitulokset osoittavat, kun taas kaliummäärät ovat suomalaisilla olleet keskimäärin suurempia kuin muilla.

MARJASEN (1969) tutkimus osoitti riippuvuussuhdetta maaperän helppoliukoisen mangaanin ja syöpään sairastuvuuden välillä. Nyt suoritetun tarkastelun perusteella voidaan olettaa, että muiden tasapainohäiriöiden kuten seleenin, SH-ryhmien, vitamiinien ja kuparin puutteen tai ravinnon kalium/magnesium -suhdevirheen ohella mangaanin puute Suomessa voi olla solujen tasapainon häiriintymisessä siinä määrin laukaiseva tekijä, että syöpään johtavat virhereaktiot ovat mahdollisia.

ANNALES AGRICULTURAE FENNIAE, VOL. 11: 407—416 (1972)
Seria PHYTOPATHOLOGIA N. 54 — Sarja KASVITAUDIT 54

PERUNAN VIRUSTAUTIEN ESIINTYMINEN SUOMESSA 1964—66

ESKO SEPPÄNEN

SEPPÄNEN, E. 1972. **Perunan virustautien esiintyminen Suomessa 1964—66.** [The occurrence of virus diseases of potatoes in Finland in 1964—66.] Ann. Agric. Fenn. 11: 407—416.

In 1964—65 a total of 719 samples of seed potatoes from 480 randomly chosen farms in 7 different regions of Finland were collected for a study of the value of seed potatoes used by farmers. In addition, in 1964—65 33 samples of commercial uncertified seed potatoes, and in 1966 some leaf samples taken from stands of certified seed potatoes and of variety trials were examined.

In the material collected from farmers tuber size varied greatly, the majority of tubers fell to the size class 41—60 g. Trueness to variety excluding the samples notified by farmers as unknown varieties and varietal mixtures was 88 per cent on the average, and including them 65 per cent only. Seed stocks were renewed or varieties changed no more often than every 15th year on the average. Viruses S and X were common while A and Y uncommon.

The quality of uncertified seed potatoes was poor. In the stands of certified seed potatoes and of variety trials only virus S was of any importance.

Siemenperunan käyttöarvoon vaikuttavat ensisijaisesti sen aitous ja terveys, mutta myös siemenmukuloiden koolla ja muillakin ominaisuuksilla on merkitystä. Meillä käytettävän siemenperunan arvosta on niukasti tietoja.

Vuonna 1950 suoritettu ensimmäinen tutkimus perunalajikkeistamme ja niiden viljelyaloista (SAKSA 1955) osoitti meillä vallitsevan lajikekirjavuuden. Lähes kolmannes tutkimusmateriaalista ilmoitettiin sekalajikkeiseksi tai lajikkeen nimeä ei tiedetty. Voidaan olettaa, että lajikenimellä ilmoitetuista eristä ainakin osa on ollut aitoudeltaan heikkoa, jopa kokonaan väärällä nimellä. Tätä olettamusta tukee VARIKSEN (1970) v. 1960 keräämien perunanäytteiden aitous, 1 161:stä Jaakko-, Kuningas Yrjö V- ja Vesijärvi-näytteestä 218 eli noin viidennes oli väärällä nimellä tai erän aitous oli alle 50 %.

Virustautien esiintymiseen ja merkitykseen ovat ensimmäisinä kiinnittäneet huomiota BRUMMER (1946, 1949) ja JAMALAINEN (1946, 1947). Tuohon aikaan ei vielä ollut kehitetty niiden määrittämiseen yksinkertaisia testausmenetelmiä, vaan määritykset perustuivat usein pelkästään silmävaraisiin havaintoihin. Näin ollen on ymmärrettävää, että huomiota kiinnitettiin ainoastaan ankariin, toisin sanoen helposti havaittaviin virooseihin, kuten kierre-, viiru-, kurttuja ainakin osittain myös kirjoviroosiin. Näistä kolme ensin mainittua ovat hyönteisten levittämiä ja niiden merkitys, huolimatta niiden voimakkaasta satoa-alentavasta vaikutuksesta (BRUMMER 1949), katsottiin niiden vähäisen esiintymisen vuoksi pieneksi (JAMALAINEN 1946). AURA (1957) käytti ensimmäisenä Suomessa serologista määritysmenetelmää ja osoitti tällä

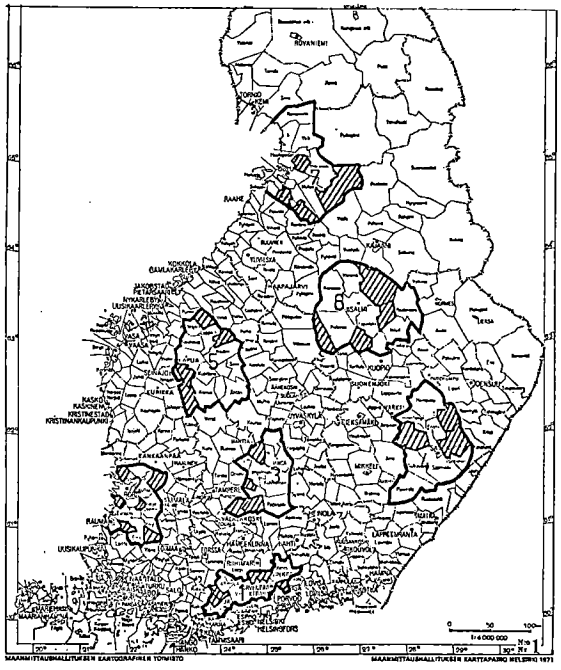
menetelmällä määritettävien X- ja S-virusten olevan meilläkin yleisiä kautta maan. Tätä käsitystä tukevia tuloksia ovat esittäneet POHJANHEIMO (1961, 1962) ja VARIS (1966) osittain silmävaraisiin havaintoihin, osittain serologisiin testauksiin perustuvissa tutkimuksissaan. Ankarampien virustautien esiintymiseen ja merkitykseen ovat kiinnittäneet huomiota edellä mainittujen lisäksi mm. VARIS (1958), POHJAKALLIO ym. (1961) ja YLLÖ (1966).

Kasvitautien tutkimuslaitoksella aloitettiin v. 1964 tutkimus, jonka tarkoituksena oli selvittää perunan virustautien levinneisyyttä ja yleisyyttä maassamme. Alustavia tuloksia tästä tutkimuksesta on julkaistu etenkin neuvonnallisissa kirjoituksissa (SEPPÄNEN 1965, 1966, 1967, SEPPÄNEN ja ULVINEN 1966). Tämän kirjoituksen tarkoituksena on tulosten esittäminen yhtenäisenä kokonaisuutena.

Aineisto ja tutkimusmenetelmät

Tutkimuksen tärkeimpänä tavoitteena oli selvittää viljelijöiden käyttämän siemenperunan käyttöarvoa, erityisesti virustautisuutta. Lisäksi tutkittiin 33 kauppasiemennäytettä, sekä vuoden 1966 valiosiemenviljelysten ja eräiden koekasvustojen virustautisuutta.

Viljelijöiltä kerättiin siemenperunanäytteitä seitsemältä alueelta maan eri puolilta (kuva 1).



Kuva 1. Seitsemän alueen arvalla valitut kunnat (viivoitetut alueet), joista näytteet kerättiin maataloushallituksen tilastoviljelmätiloilta. Vrt. taulukko 1.

Fig. 1. The regions and the local governments (lined areas) in which the samples were collected from farms chosen at random. Cf. Table 1.

Viljelmät valittiin siten, että kultakin määrätyltä alueelta otettiin arpomalla niin monta kuntaa, että niissä olevien ns. maataloustilastoviljelmien yhteisluku nousi vähintään 70:een. Koska useilla viljelmillä viljellään useampaa kuin yhtä lajiketta, arvioitiin kultakin alueelta saatavan noin sata näytettä. Kokonaistavoite oli 566 viljelmää 28 silloisen kunnan alueella. Vuonna 1964 kerättiin aineisto Uudeltamaalta (Hyvinkään mlk., Nummi, Pornainen ja Sammatti), Itä-Savosta (Kerimäki, Rantasalmi ja Savonranta), Etelä-Pohjanmaalta (Evijärvi, Nurmo, Peräseinäjoki ja Vimpeli) ja Pohjois-Pohjanmaalta (Kiiminki, Liminka ja Utajärvi). Vuonna 1965 olivat vuorossa Satakunta (Ahlainen, Harjavalta, Lappi, Lavia, Luvia ja Säkylä), Pohjois-Häme (Eräjärvi, Kuorevesi, Luopioinen ja Pälkäne) ja Pohjois-Savo (Keitele, Maaninka, Sonkajärvi ja Varpaisjärvi). Näytteitä saatiin kaikkiaan 480 viljelmältä, mikä on 85 % tavoitteesta.

Koska edellytykset virustestauksiin olivat rajoitetut ja koska toisaalta viljelysalat olivat yleensä pieniä, otettiin kutakin lajiketta vain 20 mukulan suuruinen näyte. Näin pieni näyte ei anna luotettavaa kuvaa tutkittavan erän hyvyydestä, mutta tutkimuksen päätarkoitusta, kokonaiskuvan saamista virustautien esiintymisestä, se palvelee riittävästi. Lisäksi 25 erästä otettiin 100 mukulan rinnakkaisnäytteet; 20 mukulan ja 100 mukulan antamat tulokset eivät olennaisesti poikenneet toisistaan, joten 20 mukulan näytteistä saatuja tuloksia voidaan pitää suuntaa antavina ko. erien käyttöarvosta.

Näytteiden keräyksen yhteydessä koottiin tietoja myös viljelyaloista, viljeltävän perunakannan alkuperästä sekä siitä, miten kauan kukin lajike oli viljelmällä ollut.

Näytteet punnittiin mukuloittain ja istutettiin näytteittäin metrin rivivälein ja noin puolen metrin taimivälein virusten kosketuslevinnän ehkäisemiseksi. Kasvustoista tehtiin silmävaraisesti aitous- ja virustautisuushavainnot.

Virustestaus aloitettiin nopeimmin kehittyneistä kasvustoista niiden saavutettua 20 cm:n korkeuden. Testaus suoritettiin rutiinimenetelmin. S- ja X-virukset testattiin serologisesti agglutinaatiomenetelmällä ja A- ja Y-virukset testikasvin A 6 (*Solanum demissum* x Akvila) avulla siten, että testattavan kasvin lehdestä puristettu mehu inokuloitiin carborundum-jauheen (400 meshiä) avulla A 6:n irtolehtiin, jotka pidettiin 8 päivän ajan kostealla suodatinpaperilla jatkuvassa noin 2500 luxin valossa ja konstantissa lämpötilassa. A-virus testattiin 16—18 ja Y-virus 20—22 °C:n lämpötilassa.

Tulosten laskennassa on otettu huomioon kunkin näytteen edustama viljelyala, joten tulokset ovat aina painotettuja keskiarvoja ellei nimenomaan toisin mainita.

Kauppasiemennäytteet ostettiin keväällä siemenalan liikkeistä. Ne eivät anna kuvaa koko siemenkaupasta, sillä suurin osa näytteistä oli palstaviljelijöille myytävää varhaisperunan siementä. Ne käsiteltiin samalla tavoin kuin viljelijöiltä hankitut näytteet.

Lajikekokeiden ja valiosiemenviljelysten virustestaukset tehtiin kasvustoista otetuista satunnaisnäytteistä. Valiosiemenviljelyksistä otettiin satunnaisnäytteet, joiden suuruus oli 30—50 kpl/ha viljelyksen koosta riippuen. Lajikekokeista otetut näytteet olivat pieniä, vain 10 kpl, mutta eri koepaikkojen näytteitä voidaan pitää toistensa kerranteina.

Tulokset

Lajikkeet, niiden viljelyalat ja lajikkeen uusiminen

Näytteistä oli 27 eri lajikenimellä ilmoitettuja 525 kpl (73 %), tuntemattomia 99 kpl (14 %) ja sekalajikkeisia 95 kpl (13 %). Vain muutama näyte oli ilmoitettu väärällä lajikenimellä. Taulukossa 1 esitetään näytteiden jakautuminen tärkeimpien lajikkeiden ja eri alueiden kesken.

Taulukko 1. Näytteiden jakautuminen lajikkeittain ja alueittain.
Table 1. The varietal and regional distribution of the samples.

Alue *Region*	Viljelmiä kpl *Number of farms*	Näytteitä kpl *Number of lots*	Lajikkeet — *Varieties*												
			Eigenheimer	Jaakko	Rekord *Record*	Siikli *Sieglinde*	Amyla	Nuutti *Frühnudel*	Olympia	Pauli *Paul Wagner*	K. Yrjö V *King George V*	Muut lajikkeet *Other vrs.*	Tuntemattomat *Unknown*	Sekalaiset *Mixtured*	
1. Uusimaa	66	107	2	12	2	8	2	3	2	8	7	38	11	12	
2. Satakunta	62	94	3	4	12	10	3	2	5	3	5	16	22	9	
3. Pohjois-Häme	73	113	8	7	8	9	5	4	15	1	4	28	14	10	
4. Itä-Savo	71	132	0	14	18	3	4	7	2	0	2	46	15	21	
5. Etelä-Pohjanmaa ..	71	90	53	3	3	1	6	7	0	0	0	7	3	7	
6. Pohjois-Savo	83	117	12	11	11	1	7	0	0	9	3	12	28	23	
7. Pohjois-Pohjanmaa	54	66	21	8	1	2	3	1	0	1	0	10	6	13	
Yhteensä — *Total*	480	719	99	59	55	34	30	24	24	22	21	157	99	95	
Näytteiden edustamat alat ha *Total area represented by the samples ha*		183.7	38.3	21.0	18.6	6.8	5.2	6.8	5.8	4.4	5.4	27.3	44.1		
Keskimääräinen ala/näyte ha *The mean of areas represented by the samples ha*		0.26	0.39	0.36	0.34	0.20	0.17	0.28	0.24	0.20	0.26	0.17	0.23		

Kaikkiaan 9:stä lajikkeesta oli vähintään 20 näytettä, ja vain näiden lajikkeiden tulokset esitetään lajikekohtaisesti. Todettakoon, että nämä samat lajikkeet olivat yleisimmät myös vuoden 1965 lajiketiedustelun (ANON. 1966) mukaan, ja niiden suhteelliset viljelyalat tässä aineistossa poikkeavat lajiketiedustelun tuloksista vain kahden lajikkeen, Rekordin ja Paulin, osalta enemmän kuin yhden prosenttiyksikön. Näin ollen aineisto hyvin edustaa meillä viljeltyjä lajikkeita, ja saatuja tuloksia voidaan ainakin yleisimpien lajikkeiden osalta pitää luotettavina.

Näytteiden edustamat viljelyalat olivat pieniä (taulukot 1, 2 ja 3). Ne suurenivat lähimain tilan peltoalan mukaan (taulukko 3), mutta yli 25 peltohehtaarin tiloilla ne jäivät suhteellisesti pienemmiksi. Eigenheimer-, Jaakko- ja Rekordnäytteiden keskimääräiset viljelyalat olivat suurimmat, 0.39, 0.36 ja 0.34 ha. Näytteiden keskimääräiset viljelyalat olivat pohjoisimmilla alueilla suurimmat, mikä johtuu ainakin osittain Eigenheimerin ja Jaakon yleisyydestä.

Taulukosta 4 voimme todeta, että perunansiemenen uusiminen, joko uuden kannan hankkiminen ennen viljellystä lajikkeesta tai kokonaan uuden lajikkeen hankkiminen, tapahtuu meillä verraten harvoin. Keskimäärin vain runsas kolmannes perunasta oli uusittu viimeisten 5 vuoden aikana ja suunnilleen viidennes kunkin muun luokan aikana. Tämän mukaan meillä

Taulukko 2. Näytteiden edustamat viljelyalat, keskimääräinen mukulakoko ja aitous alueittain.
Table 2. The regional growing areas represented by the samples and the means of growing areas, of tuber size and of genuineness.

	Näytteiden edustama viljelyala *Growing area represented by the lots* ha	Keskim. viljely-ala/näyte *Mean growing area/a lot* ha	Keskim. mukulakoko *Mean tuber size* g	Keskim. aitous *Mean genuineness* %	
				1	2
1. Uusimaa	22.8	0.21	73	83	(61)
2. Satakunta	20.7	0.22	56	86	(53)
3. Pohjois-Häme	22.4	0.20	55	85	(72)
4. Itä-Savo	27.3	0.21	71	90	(70)
5. Etelä-Pohjanmaa	31.0	0.34	58	96	(88)
6. Pohjois-Savo	28.8	0.25	51	81	(38)
7. Pohjois-Pohjanmaa	30.7	0.41	54	81	(67)
Yhteensä — *Total*	183.7				
Keskiarvo — *Mean*		0.25	59	88	65

[1] Aitousprosentti laskettu ilman tuntemattomien ja sekalaisten edustamaa alaa. — *Genuineness calculated without the areas represented by the unknown and mixtured varieties.*
[2] Tuntemattomien ja sekalaisten edustama ala mukaanluettuna. — *With the areas represented by unknown and mixtured.*

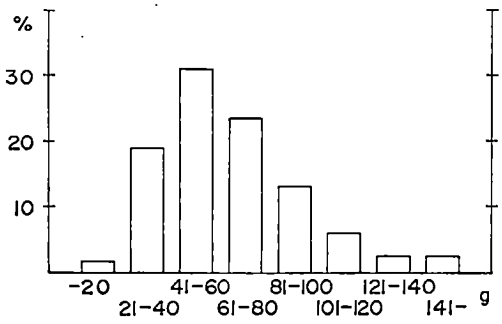
Taulukko 3. Tulosten tarkastelu tilan peltoalaan perustuvan suuruusluokittelun mukaan.
Table 3. The scrutiny of the results in the light of different size classes of the farms.

	Tilasuuruusluokat *Size class of farms* ha						Yhteensä/ keskim.
	1.00—5.00	5.01—10.00	10.01—15.00	15.01—25.00	25.01—50.00	50.01—	*Total/mean*
Kokonaisala ha *Total area represented by the lots ha*	13.5	32.6	48.2	30.1	22.3	37.0	183.7
Keskim. ala/näyte ha *Mean growing area/a lot ha*	0.09	0.17	0.26	0.35	0.39	0.67	0.26
Keskim. mukulakoko g *Mean tuber size g*	53	59	58	62	68	56	59
Keskim. aitous % *Mean of genuineness %*	82	85	91	84	89	85	88
Keskim. virustautisia % *Mean of incidence of virusdiseased %*	82	80	80	79	72	79	79

Taulukko 4. Siemenen uusiminen eri tilasuuruusluokissa.
Table 4. Change of seed stocks in different size class of farms.

Luokka *Size class*	Lajikkeen kantasiemen ollut tilalla vuosia *Stock seed grown at farm without change (years)* 1—5[1] %	1—5[2] %	6—10 %	11—20 %	20— %
1. 1.00— 5.00 ha	35	2	19	27	17
2. 5.01—10.00 „	37	2	17	28	16
3. 10.01—15.00 „	19	10	21	27	23
4. 15.01—25.00 „	27	9	26	24	14
5. 25.01—50.00 „	13	34	17	17	19
6. 50.01— „	8	25	23	22	22

[1] Kantasiemen hankittu toiselta viljelijältä. *Stock seed obtained from other farmers.*
[2] Kantasiemen hankittu kaupasta. *Stock seed from seedman's shop.*



Kuva 2. Viljelijöiltä kerättyjen siemenmukuloiden jakautuminen painoprosentteina eri kokoluokkien kesken.
Fig. 2. The size distribution of the seed tubers collected from farmers.

siemen uusitaan suunnilleen joka 15. vuosi. Tilasuuruusluokkien välillä ei ole havaittavissa eroja uusimisalttiudessa. Sen sijaan on selvästi todettavissa, että tilan suuretessa uusi siemen hankitaan yhä useammin kaupasta kuin toiselta viljelijältä. Pienimmissä tilasuuruusluokissa on lähes kaikissa tapauksissa uusi siemen hankittu toiselta viljelijältä, mutta suurimmassa luokassa vain joka neljännessä tapauksessa.

Mukulakoko

Mukulakoolle oli ominaista suuri näytteensisäinen vaihtelu. Vaikka näytteet otettiin myös mukulakoon suhteen valikoimatta, on 20 mukulan näyte liian pieni osoittamaan mukulakoon hajontaa. Vertailunäytteiksi otetuista 25:stä 100 mukulan erästä sen sijaan laskettiin keskihajonta, joka vaihteli 8.61—38.32, keskiarvo oli 21.82.

Luotettavan kokonaiskuvan saamista mukuloiden koosta haittasi mm. niiden erilainen itäneisyys punnitusaikaan, ja esitettyjä lukuja voidaan pitää vain suuntaa antavina. Näytteiden sisältämien siemenmukuloiden jakautuminen suuruusluokkiin ilman niiden edustamien viljelyalojen huomioon ottamista ilmenee kuvasta 2. Suurin osa mukuloista (87 %) on kokoa 21—100 g, ja yleisin on kokoluokka 41—60 g. Näytteiden keskimääräisiin mukulapainoihin ja viljelyaloihin perustuvissa laskelmissa päädytään suunnilleen samanlaisiin johtopäätöksiin (taulukot 2, 3 ja 5). Uudenmaan ja Itä-Savon näytteiden keskipainot poikkeavat yllättävän paljon muista, syy voi olla ainakin osaksi suurimukulaisten lajikkeiden viljelystä johtuva. Tilasuuruusluokittain keskipainoja tarkasteltaessa käy ilmi, että tilakoon suuretessa — lukuun ottamatta yli 50 ha:n tiloja — mukulakoko suurenee.

Aitous

Aitoutta on tarkasteltu yksittäisten näytteiden lisäksi tilasuuruusluokkien ja siemenen hankinta-aikojen valossa (taulukot 2, 3 ja 5). Yleensä aitous on ollut heikko, vain Etelä-Pohjanmaalla se on ollut tyydyttävä 96 %. Aitouden säilymiseen on ilmeisestikin ollut suuri merkitys sillä, että useimmilla Etelä-Pohjanmaan tiloilla viljellään vain yhta lajiketta, Eigenheimeria. Eri tilasuuruusluokkien enempää kuin siemenen hankinta-aikojenkaan välillä ei ole todettavissa selviä eroja.

Tavanmukaisen aitousmääritteen ohella on tarkasteltu, kuinka suuri osa näytteiden edustamasta koko peruna-alasta on aitoa. Koko aineiston aitous oli 88 %, mutta kun aitouden 'rasitteeksi' otetaan sekalaiset ja tuntemattomat erät, joiden aitous on 0, saadaan aitousluvuksi vain 65 %. Etelä-Pohjanmaalla on luku vieläkin niin hyvä kuin 88 %, mutta Pohjois-Savossa vain 38 %. Jos tarkastellaan tilannetta siemenen hankinta-ajan valossa ilmenee, että aitous ilman tuntemattomia ja sekalaisia ei ole riippuvainen

Taulukko 5. Tulosten tarkastelu siemenen hankinta-ajan mukaan.

Table 5. Scrutiny of the results in the light of times seed stock grown at farms without change.

	Lajikkeen kantasiemen ollut tilalla vuosia *Stock seed grown at farm without change (years)*				
	1—5[1]	1—5[2]	6—10	11—20	20—
Kokonaisala ha (%) *Total area ha* (%)	40.2 (22)	25.1 (14)	38.4 (21)	44.9 (24)	35.1 (19)
Keskim. ala/näyte ha *Mean growing area/a lot ha*	0.17	0.46	0.29	0.28	0.27
Keskim. mukulakoko g ..... *Mean tuber size g*	59	61	66	55	56
Keskim. aitous % .. *Mean of genuineness %*	83	89	84	89	89
Keskim. aitous %[3] *Mean of genuineuess %*[3]	67	87	66	65	43
Keskim. virustautisia % ..... *Mean of incidence of virus diseased %*	76	70	79	83	83

[1] Kantasiemen hankittu toiselta viljelijältä. *Stock seed obtained from other farmers.*

[2] Kantasiemen hankittu kaupasta. *Stock seed from seedman's shop.*

[3] Tuntemattomien ja sekalaisten edustama ala mukaan luettuna. — *Calculated including the areas represented by unknown and mixtures.*

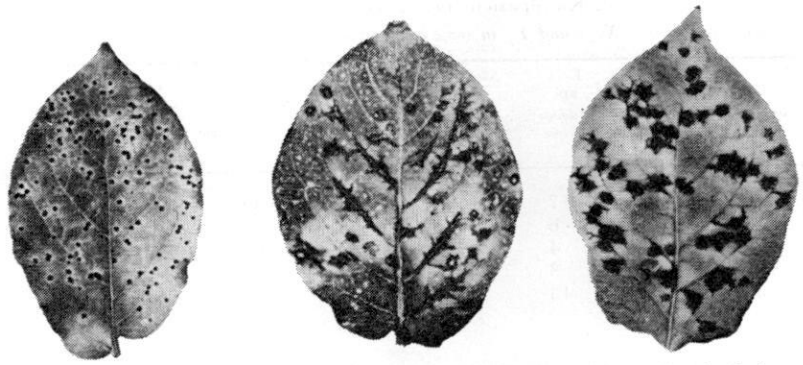
siemenen hankinta-ajasta, mutta ne mukaan luettuna se on sitä heikompi, mitä kauemmin lajiketta on viljelty. Yli 20 vuotta tilalla olleiden kantojen keskimääräinen aitous oli vain 43 %. Erittäin selvästi tulee esille ero toiselta viljelijältä ja kaupasta hankittujen kantojen välillä, kaupasta hankittujen erien aitous on ollut 20 prosenttiyksikköä parempi.

Virustautien esiintyminen

Kosketuslevintäiset virukset, X- ja S-virus osoittautuivat hyvin yleisiksi, X-viruksen saastuttamaksi todettiin 58 % ja S-viruksen saastuttamaksi 56 % meillä viljellystä perunasta (vrt. AURA 1957, VARIS 1966). Runsas kolmannes aineistosta sisälsi nämä molemmat virukset. Vastaavia tietoja ovat viimeisten 10 vuoden aikana esittäneet mm. FEDOTOVA ja SHCHERBAKOVA (1964), RANDALU (1966) ja TRUSKINOV (1970) Neuvostoliitosta, GABRIEL (1962) Puolasta, FROGNER (1964) sekä OPSAHL ja HERJE (1966) Norjasta ja LIHNELL (1971) Ruotsista.

Hyönteislevintäisten A- ja Y-virusten esiintyminen oli vähäistä, yhteensä vain 4 prosenttia aineistosta todettiin niiden saastuttamaksi (vrt. esim. GABRIEL 1962 ja LIHNELL 1971). On huomattava, että A- ja Y-virusten määrittäminen yksinomaan testikasvin A 6 avulla on osittain epäluotettavaa, koska niiden eri rotujen A 6:ssa aiheuttamat oireet ovat hyvin monenlaiset, vieläpä vaihtelevat (KÖHLER 1953, WENZL 1963, BARTHELS 1970 ja BOKX 1970). BARTHELS päätyykin toteamukseen, että tämä testi ei ole täysin luotettava näiden virusten määrittämiseksi. Tässä tutkimuksessa on taudin aiheuttajaksi määritetty A-virus niissä tapauksissa, joissa kuoliolaikut A 6:ssa olivat mustat, tähtimäiset, ja terveen solukon ja kuolion välinen raja oli epäselvä. Useimmissa tapauksissa laikut olivat pieniä (vrt. BARTHELS 1970, isolaatti Magna kuvassa 5) ja vain harvoin suurehkoja, kuten kuvassa 3. Y-viruksen aiheuttamiksi määritettiin kahdenlaiset oireet: pienehköt selvärajaiset laikut, jotka ovat ominaiset mm. Tammiston aikaisen kantamalle Y-viruksen rodulle, sekä rengasmaiset ja osittain lehtisuonia myötäilevät kuoliolaikut, jotka tulivat esille mm. Savon punaista testattaessa (kuva 3). On mahdollista, että joissakin tapauksissa myös Acuba-virus tai X-viruksen jokin rotu on ollut kysymyksessä, sillä käytetty testikasvikanta ei ollut X-viruksen saastuttama (vrt. esim. KÖHLER 1953, RAYMER ja MILBRATH 1957, BOKX 1964). Joka tapauksessa hyönteislevintäisten virusten esiintyminen oli vähäistä, useimmissa näytteissä merkityksetöntä. Kierreviroosia todettiin vain muutamissa yksilöissä.

Taulukossa 6 on esitetty tulokset virustautien alueittaisesta esiintymisestä. Voidaan todeta, että alueiden välillä on eroja, mutta ne eivät ole selitettävissä esimerkiksi alueittaisen ilmaston vaihtelun, vaan kullakin alueella viljeltyjen lajikkeiden perusteella. Taulukosta 7 voidaan todeta, että X- ja S-virusten esiintyminen eri lajikkeissa vaihtelee suuresti. Eigenheimer, Olympia ja Pauli olivat yleisemmin X-viruksen, kun taas Jaakko, Amyla ja Nuutti S-viruksen saastuttamia. Sama oli todettavissa myös AURAN (1957) tuloksissa. Hyönteisten levittämien virus-



Kuva 3. Vasemmalla ja keskellä kahden Y-virusrodun ja oikealla A-viruksen aiheuttamat oireet A 6:n lehdissä.

Fig. 3. The symptoms caused by two strains of virus Y (left and middle) and those of virus A in leaves of A 6 (right).

ten yleisempi esiintyminen Itä-Savossa selittyy ainakin osaksi sillä, että siellä vanhastaan viljellyt maatiaisperuna Savon punainen, Aikainen ruusu ja Upto ovat pahasti näiden virusten saastuttamia ja niistä on tartunta levinnyt muihinkin siellä viljeltyihin lajikkeisiin. Uudellamaalla ja Satakunnassa ovat vastaavanlaisina tartunnan levittäjinä toimineet Magnum bonum, Preussen ja Tammiston aikainen. Lisäksi muutamat tuntemattomien ja sekalaisten ryhmän erät olivat pahasti saastuneita. joten tartunnanlähteitä saattaa olla muitakin. Toisaalta hyönteislevintäisten viroosien esiintymisen jollakin seudulla ei tarvitse olla riippuvainen tartunnanlähteiden olemassaolosta enempää kuin ilmastostakaan, sillä alueelle viety kantasiemen on saattanut olla saastunutta jo sinne vietäessä. Näin selittyy esimerkiksi A- ja Y-virusten runsaampi esiintyminen Pohjois-Pohjanmaalla kuin Pohjois-Savossa ja Hämeessä.

Eri tilasuuruusluokkien kesken ei virustautien esiintymisessä todettu eroja (taul. 3), mutta tilalla kauemmin viljellyissä erissä oli tautisten osuus suurempi kuin äsken hankituissa erissä. Tilalla 1—5 vuotta viljellyissä erissä olivat kaupasta hankitut erät muita terveempiä (taul. 5).

Kauppasiemenerien testaus

Taulukossa 8 esitetään tulokset 33 siemenerän testauksesta. Koska näytteet ovat näidenkin

Taulukko 6. Virustautien esiintyminen alueittain.
Table 6. The occurrence of virus diseases in different regions.

Alue *Region*	Näytteiden luku kpl *Number*	edustama ala ha *and area (of the lots)*	Keskimäärin eri virusten saastuttamia % *Means of infected plants in the different regions %* X	S	A	Y	Yht. *Total*
Uusimaa	107	22.8	60	82	1.4	4.1	91
Satakunta	94	20.7	58	47	3.1	1.9	71
Pohjois-Häme	113	22.4	49	58	0.1	0.8	78
Itä-Savo	132	27.3	49	55	6.7	6.9	71
Etelä-Pohjanmaa ..	90	31.0	64	40	2.0	1.4	78
Pohjois-Savo	117	28.8	55	60	0.0	0.2	84
Pohjois-Pohjanmaa	66	30.7	66	52	1.9	0.8	81
Yhteensä — *Total*	719	183.7					
Keskiarvo — *Mean*			58	56	2.2	2.2	79

Taulukko 7. Lajikkeiden virustautisuus.
Table 7. Incidence of viruses in different varieties.

Lajike *Variety*	Näytteiden luku kpl *Number*	edustama ala ha *and area (of the lots)*	Eri virusten saastuttamia % *Infected with viruses %* X	S	A	Y	Yht. *Total*
Eigenheimer	100	38.3	77	41	1.8	1.5	84
Jaakko	58	21.0	75	88	2.6	1.0	97
Rekord — *Record* ..	55	18.6	13	18	1.0	3.0	28
Siikli — *Sieglinde* ..	34	6.8	49	41	0.7	2.4	64
Amyla	30	5.2	60	76	0.7	0.4	88
Nuutti — *Frühnudel*	24	6.8	15	72	0.8	0	75
Olympia	22	5.8	76	52	0	0.1	90
Pauli – *Paul Wagner*	22	4.4	78	49	0.4	0	92
K. Yrjö V — *King George V*	21	5.4	50	58	0.8	1.3	82
Muut lajikkeet — *Other varieties*	168	27.3	48	65	5.7	5.2	81
Sekalaiset ja tuntemattomat — *Varietal mixtures and unknown*	185	44.1	62	61	1.9	2.5	86

Taulukko 8. Kauppasiemenerien virustautisuus 1964—65.

Table 8. Incidence of viruses (X, S and Y) in some commercial seed potato lots (not-guaranted) in 1964—65.

Lajike — *Variety*	Eriä kpl *Number of lots*	Mukulain keskip. *Mean size of tubers* g	Aitous *Genuineness*	Viroottisuus % X-virus *Percentages of plants with viruses* X	S-virus S	Y-virus Y	Yhteensä *Total*
Siikli — *Sieglinde*	7	58	82	30	49	0	58
Jaakko	6	70	58	71	71	14	83
Amyla	4	62	96	60	91	1	99
Rekord — *Record*	3	107	94	10	8	0	13
Muut — *Other vars.*	13	62	67	51	56	4	73

Taulukko 9. Tulokset valiosiemenviljelyksiltä otettujen lehtinäytteiden virustestauksesta v. 1966 (SEPPÄNEN ja ULVINEN 1966).

Table 9. Results of virustests of leafsamples collected from stands of seed potatoes for elite class in 1966 (SEPPÄNEN and ULVINEN 1966).

Lajike *Variety*	Viljelysten luku kpl *Number of stands*	yht. ala ha *Total area of stands*	Testattu yksilöitä /näyte *Mean number of leaves of the samples*	Viroottisia X-virus *Porcentages of leave* X	S-virus S	Yht.[1] *Total* [2]
Pito	7	8.40	32	8	89	89
Valtti	7	19.15	40	7	90	91
Barima	3	10.00	42	0	0	0
Olympia	2	3.00	45	98	96	99
Rekord — *Record*	1	1.00	28	0	4	4
Siikli — *Sieglinde*	1	2.00	57	7	84	84

[1] A- ja Y-virusta ei todettu.
[2] *Viruses A and Y were not detected.*

osalta pieniä, 5 kg kukin, on tuloksia tarkasteltava samaan tapaan kuin viljelijöiltä kerätyn aineiston tuloksia, yhtenä kokonaisuutena.

Mukuloiden keskipaino, aitous ja viroottisuus ovat yhtä vaihtelevia ja keskimäärin samaa tasoa kuin viljelijäaineistossakin.

Valiosiemenviljelysten testaus

Vuonna 1966 testattiin valiosiemenviljelyksiltä otetut lehtinäytteet. A- ja Y-virusta ei todettu yhtään tapausta. X-virusta todettiin yleisesti vain Olympiassa, mutta S-virusta Olympian lisäksi myös Pidossa, Valtissa ja Siiklissä (taul. 9). Valiosiemenen tuotannossa näyttää S-virus olevan milteipä ainoa ongelma, etenkin kotimaisissa lajikkeissa.

Koekasvustojen viroottisuus

Taulukossa 10 on esitetty tärkeimmät tulokset lajikekokeiden kasvustoista otetuista näytteistä. Tulokset osoittavat, että S-virus oli useimmissa lajikkeissa hyvin yleinen. Myös X-virusta oli mainittavasti, mutta A- ja Y-viruksia ei todettu yhdessäkään tapauksessa.

Taulukko 10. Tuloksia perunan lajikekokeiden virustautitestauksista v. 1966.

Table 10. Incidence of viruses X and S in some varieties in trial fields of some trial localities in 1966.

Lajike — *Variety*	Näytteitä yhteensä *Number of lots*	Viroottisia keskimäärin % X-virus *Percentages of plants with viruses* X	S-virus S	Yhteensä *Total*
Ruusulehti — *Rosafolia (check var.)*	10	12	32	43
Pito	9	7	80	80
Veto (Jo 0179) ..	9	11	79	81
Valtti	8	15	84	85
Rekord — *Record*	6	13	53	53
Jaakko	5	22	94	98
Amyla	4	32	100	100
Eigenheimer	4	60	65	73

Tiivistelmä

Vuosina 1964—65 kerättiin 480 viljelmältä 7 alueelta maan eri puolilta kaikkiaan 719 siemenperunanäytettä. Niiden ohella hankittiin 33 kauppasiemennäytettä. Siemennäytteet kasvatettiin Tikkurilassa ja lehdistä testattiin niiden viroottisuus. Vuonna 1966 testattiin valiosiemenviljelysten ja lajikekoekasvustojen viroottisuutta.

Viljelijöiltä kerätyssä aineistossa mukuloiden koko vaihteli suuresti yleisimmän koon ollessa 41—60 g. Lajikeaitous oli lukuunottamatta eriä, jotka oli ilmoitettu tuntemattomiksi tai sekalajikkeisiksi, keskimäärin 88 %, mutta tuntemattomat ja sekalajikkeiset mukaan luettuna vain 65 %. Siemenkanta oli uusittu tai lajike vaihdettu keskimäärin vasta joka 15. vuosi. Virukset S ja X olivat erittäin yleisiä, kun taas virukset A ja Y esiintyivät vain harvoissa näytteissä.

Kauppasiemenen terveys oli heikko. Valiosiemenkasvustoissa ja lajikekoekasvustoissa ainoastaan virus S oli yleinen.

Kiitokset. — Agronomien Yhdistykselle olen kiitollinen saamastani apurahasta.

KIRJALLISUUTTA

ANON. 1966. Tärkeimpien vilja-, herne- ja perunalajikkeiden viljelylaajuudet vuonna 1965. Maatal.til. Kuukausikatsaus 1966: 23—37.

AURA, K. 1957. Suomessa viljellyn perunan virustautisuudesta. Summary: On potato virus diseases in Finland. Maatal.tiet. Aikak. 29: 103—110.

BARTHELS, R. 1970. Symptomausbildung von Viren der Kartoffel-Y-Gruppe auf A 6-Blättern. Potato Res. 13: 119—128.

BOKX, J. A. de 1964. Onderzoekingen over het aantonen van aardappel-Y^N-virus met behulp van toetsplanten. Summary: Detection of potato virus Y^N by means of test plants. Diss. 84 p. Baarn.

— 1970. Effect of temperature on the development of local lesions on 'A 6' leaves after inoculation with potato virus A. Potato Res. 13: 167—174.

BRUMMER, V. 1946. Tutkimuksia tärkeimmistä Tammistossa esiintyneistä perunavirooseista. Hankkijan Siemenjulk. 1946: 176—185.

— 1949. Vesijärven ja Tammiston aikaisen suhtautumisesta perunavirooseihin. Summary: On susceptibility to the virus diseases of Harbinger and Tammisto Early potatoes. Maatal.tiet. Aikak. 21: 17—28.

FEDOTOVA, T. & SHCHERBAKOVA, N. 1964. Ispol'zovanie serologicheskogo metoda v rabotakh po ozdorovlenyia semennogo Kartofelya. [The use of the serological method in producing healthy seed potatoes.] Trudy Vses. Inst. Zashch. Rast. 21: 51—56.

FROGNER, S. 1964. Potetforsøk på Opplandene 1945—1962. Forsk. Forsøk Landbr. 15: 311—339.

GABRIEL, W. 1962. Nowoczesne metody ograniczania szerzenia sie chorób wirusowych na plantacjach nasiennych Ziemniaka. [Modern methods of limiting the spread of virus diseases in seed Potato plantations.] Postepy Nauk. Rol. 9; 3: 23—42. (Ref. Rew. Appl. Myc. 43: 372.)

JAMALAINEN, E. A. 1946. The significance of potato virus diseases in Finland. Maatal.tiet. Aikak. 18: 134—146.

— 1947. Potatisvirosernas betydelse i Finland. Nord. Jordbr.forskn. 29: 568—570.

KÖHLER, E. 1953. Der Solanum demissum-Bastard 'A 6' als Testpflanze verschiedener Mosaikviren. Züchter 23: 173—176.

LIHNELL, D. 1971. Sorter och virussjukdomar i svensk matpotatisodling. Nord. Jordbr.forskn. 53: 311.

OPSAHL, B. & HERJE, K. 1966. Stamsaed jämført med ukontroller utsæd i potet. Forskn. Forsøk Landbr. 17: 21—32.

POHJAKALLIO, O., KARHUVAARA, L. & ANTILA, S. 1961. The effect of potato rugose on the yield of some potato varieties. Selostus: Viiruviroosin vaikutuksesta perunan sadon määrään. Maatal.tiet. Aikak. 33: 89—100.

POHJANHEIMO, O. 1961. Piilevinä esiintyvistä virustaudeista perunalajikkeissa. Referat: Über die latent austretenden Viruskrankheiten bei Kartoffelsorten. Maatal. ja Koetoim. 15: 242—250.

— 1962. Jaakko-perunalajike virustautien esiintymisen ja leviämisen ilmaisijana. Summary: The potato variety Jaakko as an indicator of potato virus disease. Maatal. ja Koetoim. 16: 153—158.

RANDALU, I. 1966. Andmeid Eesti NSV-S rajoonitud kartulisortide nakatatuse kohta X-, S-, K(M)- ja Y-viirusest 1954—1965 aastal. Teaduslike Tööde Kogumik 9: 91—104.

RAYMER, W. B. & MILBRATH, J. A. 1957. A local-lesion test for potato virus A in the presence of potato virus X. Phytopathology 47: 532.

SAKSA, P. J. 1955. Maamme perunalajikkeiden viljelylaajuus ja viljelyalueet. Summary: The extent and the regions of the cultivation of potato varieties in Finland. Maatal.tiet. Aikak. 27: 41—52.

SEPPÄNEN, E. 1965. Virustaudit verottavat perunasatojamme. Pellervo 66: 836—837.

— 1966. Virustaudit yleisiä perunaviljelyksillämme. Kylvösiemen 3: 22—24.

— 1967. Potatisens Y-virus i Finland. Nord. Jordbr.-forskn. 49: 314—319.

—& ULVINEN, O. 1966. Perunan valiosiemenviljelysten virustautisuudesta. Kylvösiemen 4: 33—35.

TRUSKINOV, E. 1970. Rasprostranenie virusa M v kollektsii Kartofelya. Sb. Trud. Aspir. Molod. Nauch. Sotrud. 16: 432—439.

VARIS, E. 1958. Perunalajikkeidemme virustautisuudesta. Koetoim. ja Käyt. 15: 19.

— 1966. Kylvätkö samaa mitä korjaat. Pellervo 67: 511—512.

— 1970. Variation in the quality of table potato and the factors influencing it in Finland. Acta Agr. Fenn. 118, 3: 1—99.

WENZL, H. 1963. Zur Unterscheidung von Y- und A-Virus im Abreibeverfahren (Schalentest) auf *Solanum demissum* A 6. Pfl.schutzber. 33: 161—172.

YLLÖ, L. 1966. Siemenperunan terveyden merkitys. Koetoim. ja Käyt. 23: 5, 7.

Saapunut 21. 3. 1972

Esko Seppänen
Maatalouden tutkimuskeskus
Kasvitautien tutkimuslaitos
01300 TIKKURILA

ANNALES AGRICULTURAE FENNIAE, VOL. 11: 417—423 (1972)
Seria ANIMALIA NOCENTIA N. 64 — Sarja TUHOELÄIMET n:o 64

RESISTANCE OF CEREALS TO THE APHIDS RHOPALOSIPHUM PADI (L.) AND MACROSIPHUM AVENAE (F.) AND FECUNDITY OF THESE APHIDS ON GRAMINAE, CYPERACEAE AND JUNCACEAE

MARTTI MARKKULA and KAISA ROUKKA

MARKKULA, M. & ROUKKA, K. 1972. **Resistance of cereals to the aphids Rhopalosiphum padi (L.) and Macrosiphum avenae (F.) and fecundity of these aphids on Graminae, Cyperaceae and Juncaceae.** Ann. Agric. Fenn. 11: 417—423.

Rhopalosiphum padi and *Macrosiphum avenae* reproduced abundantly on all varieties and lines of spring wheat, barley and oats, of which there were more than one hundred of each cereal species in the experiments. The number of progeny of *M. avenae* was significantly smaller and its life span briefer on Nip oats than on some of the other varieties. No other significant differences appeared. The fecundity of 28 strains of *R. padi* and 36 strains of *M. avenae* was investigated on spring wheat, barley and oats, but no differences were observed between these strains. *R. padi* reproduced on more species of the families Graminae, Cyperaceae and Juncaceae than did *M. avenae*, and its progeny were usually more numerous. *Avena fatua* proved to be at least as favourable a host plant for reproduction of *R. padi* as oats. *M. avenae* reproduced equally well on *Poa annua* and on *Bromus secalinus* as on oats but less on the other species of the above families.

Many researchers have studied the resistance of cereals to aphids, especially in the 1960's. A basis for such studies was created by the clarification of the reproduction and life history of cereal aphids on several plant species (e.g. COON 1961, ORLOB 1961, HSU and ROBINSON 1962, 1963, MARKKULA and MYLLYMÄKI 1963, VILLANUEVA and STRONG 1964, JESSEP 1967). Also, lists have been made of the plant species on which these aphids live (e.g. PATCH 1938, HARPAZ 1953, ITO 1960, RICHARDS 1960, ORLOB 1961, ORLOB and MEDLER 1961, HSU 1963, ROBINSON and HSU 1963).

Although differences in resistance have been observed between varieties, they have generally been slight (e.g. COON 1961, HSU and ROBINSON 1962).

The purpose on the present study was to test differences in resistance among various plant species, varieties and lines of cereals on the basis of the fecundity and life span of the aphid. The aphids used were the bird-cherry aphid *Rhopalosiphum padi* (L.) and the grain aphid *Macrosiphum avenae* (F.), and some tests also included the rose-grain aphid *Metopolophium dirhodum* (Wlk.). A look-out was kept for any occurrence of biotypes with varying host-plant relationships.

Material and methods

The cereals were grown in pots known as Multipots, and the other test plants in clay pots on peat with added fertilizers as recommended by Soil Testing Services Ltd., Helsinki.

In initial tests it was established that the numbers of progeny of the grain aphid and the bird-cherry aphid did not vary significantly whether they lived on ears, leaves of fullgrown plants or sprouts. All the testing of cereals was done on young sprouts. Each aphid culture was protected with a PVC cylinder (MARKKULA and RAUTAPÄÄ 1965). A total of 503 varieties and lines were tested:

	Varieties tested		
	wheat	oats	barley
Macrosiphum avenae	101	182	152
Rhopalosiphum padi	115	196	192
Metopolophium dirhodum	1	45	1

Many other species of Graminae and a few species of the families Cyperaceae and Juncaceae were included in the tests. The aphids were placed on these plants in rearing cages (see MARKKULA and ROUKKA 1970).

The aphids used in the tests belonged to clones reared from one virginoparous female. Before being transferred to the test plants, the aphids were allowed to multiply on Sisu oats in a greenhouse. The wingless virginoparous females were placed on the test plants as soon as they had become adult, one aphid per plant. When the aphids had reproduced for one week, the progeny were counted. On the basis of these tests, the varieties and strains of cereal on which the counts were highest or lowest were selected for further experimentation in greenhouses. In the tests the progeny were counted and removed at weekly checks, the females being left behind to reproduce. In this way the total number of progeny of the aphids was counted. There were 20 aphids on each variety in every experiment.

The number of grain aphids was counted on 13 wheat varieties, six oat varieties and three barley varieties in the experimental field of the Department of Plant Husbandry, Tikkurila. There were five plots of 5 m^2 for each variety. On each plot, counts were made of the aphids on 20 ears located evenly throughout the plot.

In a search for differing biotypes, 36 strains of grain aphids from 27 communes and 28 strains of bird-cherry aphids from 23 communes were investigated. Some of the strains were reared from samples sent to the Department of Pest Investigation, and others from aphids gathered in various parts of the country. The most northerly strains were from Salla (app. 67° N), and one grain aphid strain was from Bergen in Norway. Irrespective of their original host plant, the aphids were reared on Sisu oats. When the strains had reproduced sufficiently, the number of progeny and the life span of 10 specimens was investigated on Tammi spring wheat, Pirkka barley and Sisu oats.

Results and discussion

Antibiosis of cereals

In terms of resistance the varieties and lines of cereals proved to be highly uniform. The grain aphid, the bird-cherry aphid and the rose-grain aphid reproduced in fair abundance on all the varieties and lines tested. The differences between the varieties were small. As regards fecundity, the only significant difference found was the number of progeny of the grain aphid on Nip oats as compared with Ta b5656, Zandster and Jo 0761 oats (Fig. 1). Although the number of progeny on Nip oats was not much smaller than that on the other varieties, there was a sizeable difference in life span. The average life span was a couple of weeks shorter than on the other varieties.

When a study was made of the oat sterile dwarf virus and its vector the leafhopper *Javesella pellucida* (F.) in the 1950's, it was found in

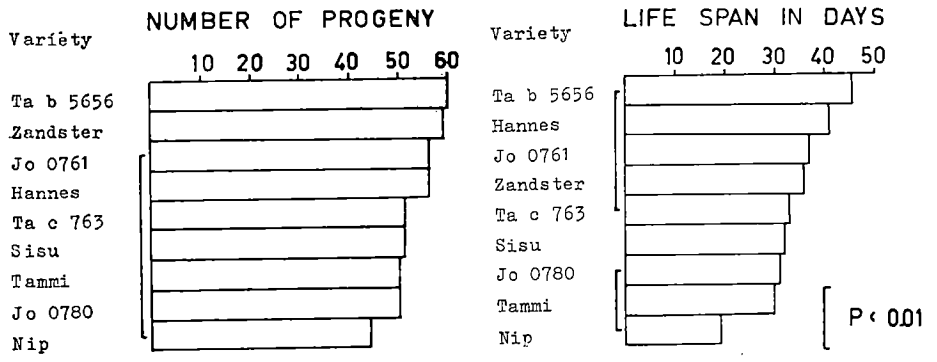


Fig. 1. Number of progeny and life-span of *Macrosiphum avenae* on some varieties of oats. The figures are averages for 20 specimens.

cage and field experiments that Nip oats were the most resistant variety to oat sterile dwarf virus. However, no significant differences appeared between the varieties in summers when damage was severe (M. Raatikainen, oral communication).

In the field, the largest number of grain aphids was found on the spring wheat Jo 0715 and the smallest number on the oat variety Hannes (Table 1). This result diverges from that of greenhouse tests, in which Jo 0715 seemed to be the most resistant, although the difference was not statistically significant.

Fecundity on Graminae, Cyperaceae and Juncaceae

The bird-cherry aphid reproduced on a greater number of plant species than did the grain aphid (Figs. 2 and 3). The numbers of progeny of the bird-cherry aphid were also usually higher than those of the grain aphid. In his experiments on host plant selection, RAUTAPÄÄ (1970) has shown that the host plant spectrum of the bird-cherry aphid is broader than that of the grain aphid. The grasses studied included species on which the grain aphid did not reproduce at all, as well as species on which its reproduction was as abundant as it was on Sisu oats. Whenever the average number of progeny on the plant fell below 10, almost all the females and their progeny died during the first week. *Poa annua* and *Bromus secalinus* were comparable to Sisu oats as host plants of the grain aphid. The aphid was also fairly prolific on some *Festuca* and *Lolium* species. RAUTAPÄÄ (1970), too, stated that *Poa annua*, *Bromus* and *Lolium* were preferents. Yet COON (1959) found that the grain aphid reproduced in small numbers on *Poa annua*.

Fairly large numbers of progeny were produced by the grain aphid on *Luzula pallescens* and *L. multiflora* (*Juncaceae*), although statistically very significantly fewer than on Sisu oats. Reproduction on *Carex* species was poor.

RAUTAPÄÄ (1970) found that Graminae were generally comparable to Sisu oats in preference value. In the present experiments, too, the num-

Table 1. Number of grain aphids in the field.

Spring wheat Variety	Number of aphids per ear	*Oats* Variety	Number of aphids per ear
Jo 0715	8.5	Pendek	2.3
Ta c2044	6.9	Sisu	1.5
Ta b3332	6.5	Jo 0721	0.4
Svenno	6.2	Guldregn II	0.2
Ring	5.9	Ta b8111	0.2
Diamant	5.6	Hannes	0.2
Diamant II	4.8		
Russian red seeded	4.8		
Erli	4.7	*Barley*	
Koga	4.1		
Norröna	4.1	Pirkka	3.5
Skala	2.7	Mari	3.4
Selkirk	2.3	Balder	2.3

P 0.05

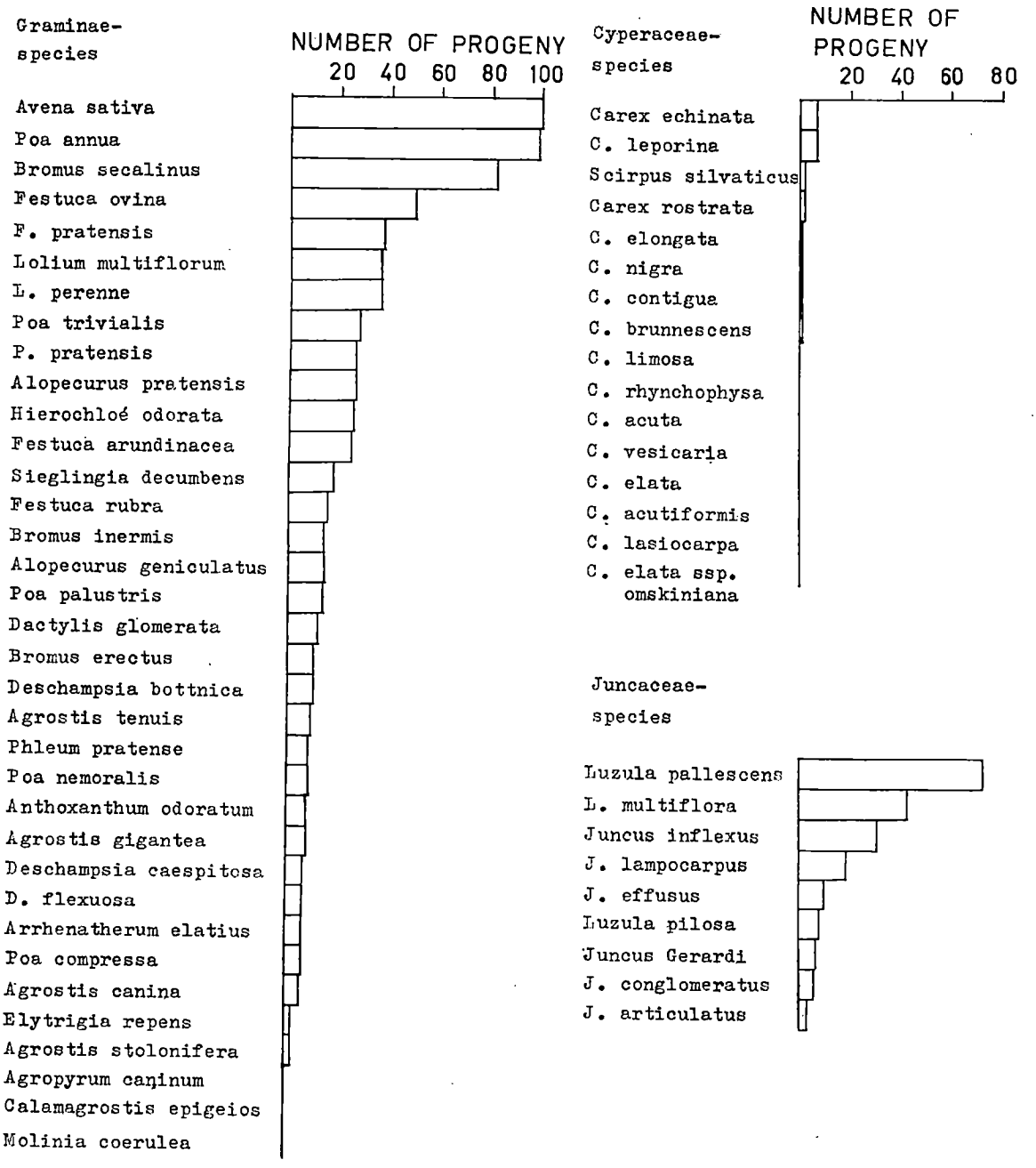


Fig. 2. Number of progeny of *Rhopalosiphum padi* on some plants of the families Graminae, Cyperaceae and Juncaceae. The number of progeny on *Avena sativa* (Sisu) = 100.

bers of progeny of the bird-cherry aphid were fairly high on many species of Graminae, although very significantly lower than on Sisu oats. *Avena fatua* proved to be at least as favourable a host plant for reproduction as Sisu oats. The reproduction of the bird-cherry aphid on the species of *Carex* was not abundant, although clearly more so than that of the grain aphid.

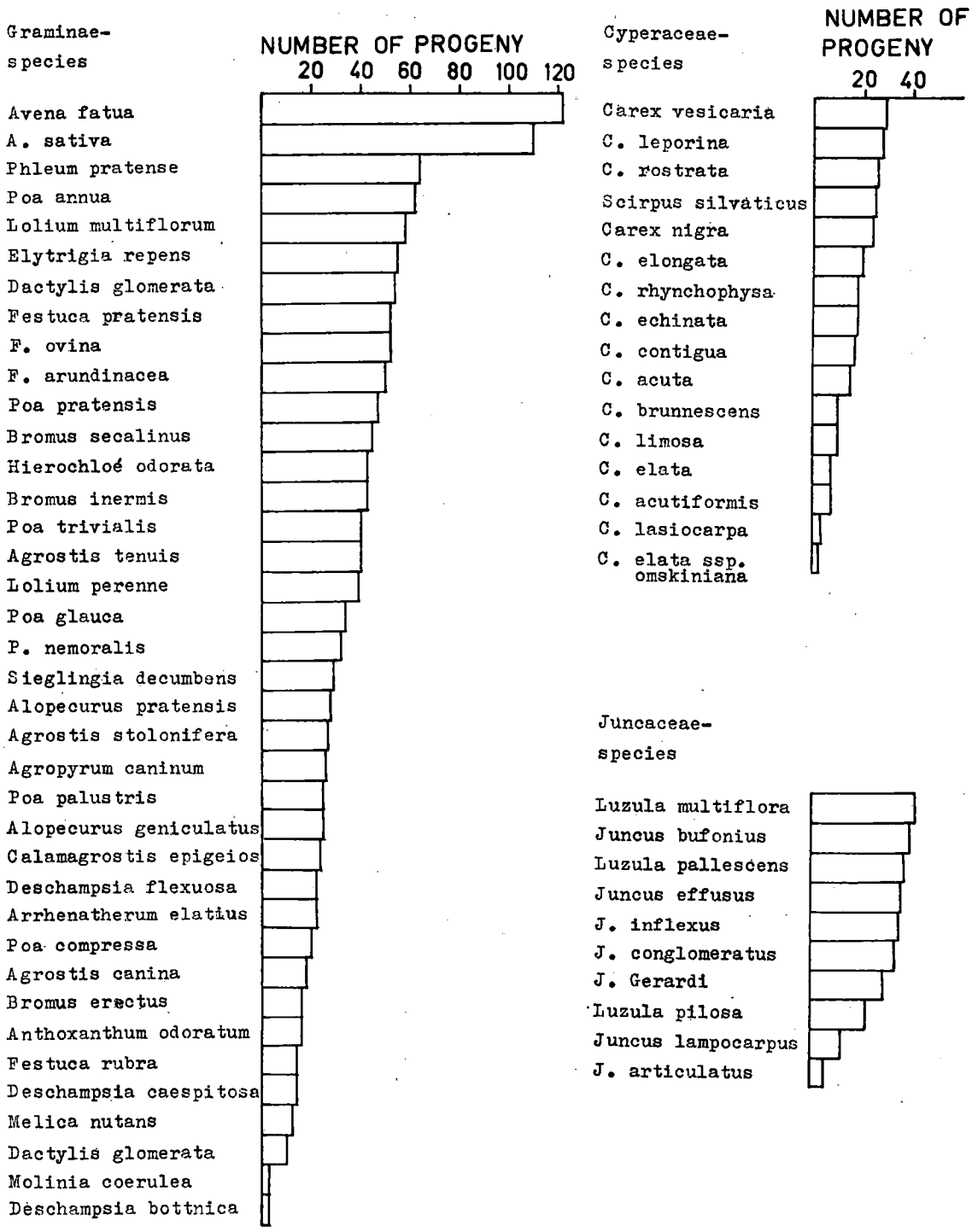


Fig. 3. Number of progeny of *Macrosiphum avenae* on some plants of the families Graminae, Cyperaceae and Juncaceae. The number of progeny on *Avena sativa* (Sisu) = 100.

Occurrence of biotypes

All the strains of grain aphid and bird-cherry aphid reproduced abundantly on the test plants. In both these species the numbers of progeny were highest on barley (bird-cherry aphid 59, grain aphid 64), slightly lower on oats (57, 57) and clearly lower on spring wheat (47, 46). The life span of the aphids was correlated with the number of progeny. In barley and oats the differences between the aphid strains were very small and not statistically significant. On spring wheat the number of progeny of the grain aphid strain originating in *Deschampsia caespitosa* from Bergen differed very significantly from most of the Finnish strains. In greenhouse tests the population did not thrive on *D. caespitosa*.

From the results of the experiments it would seem that the grain aphid and the bird-cherry aphid in Finland are both highly uniform species, and it is not possible to distinguish biotypes that diverge in their food plant relations on the basis of fecundity or life span.

REFERENCES

Coon, B. F. 1959. Grass hosts of cereal aphids. J. Econ. Ent. 49: 498—508.

Hsu, S.-J. 1963. Some notes on biology of *Rhopalosiphum padi* (L.). Plant Prot. Bull. 5: 247—254.

—& Robinson, A. G. 1962. Resistance of barley varieties to the aphid *Rhopalosiphum padi* (L.). Can. J. Pl. Sci. 42: 247—251.

—& Robinson, A. G. 1963. Further studies on resistance of barley varieties to the aphid *Rhopalosiphum padi* (L.). Can. J. Pl. Sci. 43: 343—348.

Jessep, C. T. 1967. A note on relative incidence of *Rhopalosiphum padi* (L.) on different varieties of ryegrass. N. Z. Ent. 3: 29.

Markkula, M. & Myllymäki, S. 1963. Biological studies on cereal aphids, *Rhopalosiphum padi* (L.), *Macrosiphum avenae* (F.), and *Acyrthosiphum dirhodum* (Wlk.) (*Hom., Aphididae*). Ann. Agric. Fenn. 2: 33—43.

—& Rautapää, J. 1963. PVC rearing cages for aphid investigations. Ann. Agric. Fenn. 2: 208—211.

—& Roukka, K. 1970. Resistance of plants to the pea aphid *Acyrthosiphon pisum* Harris (*Hom., Aphididae*). I. Fecundity of the biotypes on different host plants. Ann. Agric. Fenn. 9: 127—132.

Orlob, G. B. 1961. Biology and taxonomy of cereal and grass aphids in New Brunswick (*Homoptera: Aphididae*). Can. J. Zool. 39: 495—503.

—& Medler, J. T. 1961. Biology of cereal and grass aphids in Wisconsin (*Homoptera*). Can. Ent. 93: 703—714.

Patch, E. M. 1938. Food-plant cataloque of the aphids of the world. Bull. Maine Agric. Exp. Sta. 393: 1—431.

Rautapää, J. 1970. Preference of cereal aphids for various cereal varieties and species of *Graminae, Juncaceae*, and *Cyperaceae*. Ann. Agric. Fenn. 9: 267—277.

Richards, W. R. 1960. A synopsis of the genus *Rhopalosiphum* in Canada (*Homoptera: Aphididae*). Can. Ent. Suppl. 13: 1—51.

Robinson, A. G. & Hsu, S.-J. 1963. Host plant records and biology of aphids on cereal grains and grasses in Manitoba (*Homoptera: Aphididae*). Can. Ent. 95: 134—137.

Villanueva, J. R. & Strong, F. E. 1964. Laboratory studies on the biology of *Rhopalosiphum padi* (*Homoptera: Aphididae*). Ann. Ent. Soc. Amer. 57: 609—613.

MS received 31 July 1972

Martti Markkula and Kaisa Roukka
Agricultural Research Centre
Dept. of Pest Investigation
SF-01300 TIKKURILA, Finland

SELOSTUS

Kevätviljojen resistenssi tuomikirvaa ja viljakirvaa vastaan sekä näiden kirvojen lisääntyminen heinissä, saroissa ja vihvilöissä

MARTTI MARKKULA ja KAISA ROUKKA

Maatalouden tutkimuskeskus, Tuhoeläintutkimuslaitos, Tikkurila

Tuhoeläintutkimuslaitoksella on viime vuosien aikana suoritettu useita tutkimuksia kasvien resistenssistä. Tässä tutkimuksessa selvitettiin ensisijaisesti kevätviljojen resistenssiä tuomikirvaa ja viljakirvaa vastaan. Tutkittavana oli toistasataa kevätvehnän, ohran ja kauran lajiketta ja linjaa. Kirvojen lisääntyminen oli niissä runsasta. Vain Nip-kaurassa oli viljakirvan lisääntyminen tilastollisesti merkitsevästi vähäisempää kuin eräissä muissa lajikkeissa. Muita merkitseviä eroja ei ilmennyt. Nip kuuluu mustajyväisiin ja paksukuorisiin kauroihin eikä sitä enää suositella meillä viljeltäväksi.

Maamme eri osista hankittujen 28 tuomikirvakannan ja 36 viljakirvakannan lisääntymistä tutkittiin Tammikevätvehnässä, Pirkka-ohrassa ja Sisu-kaurassa. Kantojen välillä ei todettu merkitseviä eroja.

Tuomikirva lisääntyi useammissa heinä-, sara- ja vihvilälajeissa kuin viljakirva, ja myös sen jälkeläismäärä oli näissä kasveissa yleensä suurempi.

Tutkimuksen perusteella on pääteltävissä, että nykyisin viljeltävien kevätviljalajikkeiden joukossa ei ole viljan kirvoja kestäviä.

ANNALES AGRICULTURAE FENNIAE, VOL. 11: 424—436 (1972)
Seria ANIMALIA NOCENTIA N. 65 — Sarja TUHOELÄIMET n:o 65

THE IMPORTANCE OF COCCINELLA SEPTEMPUNCTATA L. (COL., COCCINELLIDAE) IN CONTROLLING CEREAL APHIDS, AND THE EFFECT OF APHIDS ON THE YIELD AND QUALITY OF BARLEY

JORMA RAUTAPÄÄ

RAUTAPÄÄ, J. 1972. **The importance of Coccinella septempunctata L. (Col., Coccinellidae) in controlling cereal aphids, and the effect of aphids on the yield and quality of barley.** Ann. Agric. Fenn. 11: 424—436.

There was a significant negative correlation between the numbers of coccinellid larvae and the population growth of *R. padi*. However, no correlation was found between the numbers of adults and *R. padi* nor between larvae or adults and *M. avenae*. While at the beginning of the experiments at the turn of June and July the total number of coccinellids (larvae or adults) was 100/m² in barley, and there were 25 aphids on each shoot, the aphid index (= the sum of the aphids living on one main shoot every day during the whole period of experiment) was 7 % lower than expected on the basis of the control cages. Likewise, when the number of coccinellids remained the same 100/m² but with 1.3 aphids per shoot, the aphid index decreased to 78 % units of what would have been expected. On the basis of the results regression equation was calculated which describes the effect of coccinellids on aphid populations.

The aphids reduced significantly the grain yield and the 1000-grain weight of barley. An aphid index of 1000 units (for example 50 aphids living on each main shoot for 20 days) corresponded to a 27 % loss in yield. The aphids reduced the extract content of malt but had no significant effect on other brewing qualities. The aphids did not affect the amounts of trace elements in the grain.

The effects of the oat bird cherry aphid *Rhopalosiphum padi* (L.) and the English grain aphid *Macrosiphum avenae* (F.) (*Hom., Aphididae*) on cereals have been studied earlier in Finland with cage experiments (RAUTAPÄÄ 1966, 1968 a and b). *M. avenae*, which had been living on wheat for long enough and in sufficient numbers, significantly reduced the yield but did not affect its quality, which was measured by its falling number and Pelshenke number. The yield of barley was reduced, too, and some changes were noted in the brewing quality. *R. padi* reduced the yield of oat and the protein quantity of the grain was lowered. Recent studies clarifying the effects of aphids on cereals have been reviewed by KOLBE (1969). LATTEUR (1970) has applied the same index as RAUTAPÄÄ (1966) to describe the abundance and presence of aphids in cereals. The population dynamics of cereal aphids has been studied in the field recently by e.g. LECLANT (1969), LATTEUR (1971), DEAN and LUURING (1970), and the effect of aphids on yield and its quality by e.g. ANGLADE (1969). Chemical control of aphids

and at the same time their effects on cereals have been studied by STERN and BOWEN (1967), GRIGOROV (1967), HARVEY and HACKEROTT (1970), WARD et al. (1970) and TWINE (1971).

The role of coccinellids in controlling the populations of cereal aphids has not been studied widely. On the basis of field observations KIECKHEFER and MILLER (1967), HAMILTON and KIECKHEFER (1969) and LATTEUR (1970) have compared numbers of cereal aphids and coccinellids and concluded the effects of coccinellids on the aphid populations. JONES (1972) caught by means of cages cereal aphids, their parasites and predators from the field and concluded that in various years bad weather, coccinellids, syrphids or *Chrysopidae* species had adverse effect on the aphid colonies. In this respect other aphid species than those living on cereals have been studied more (see e.g. HAGEN 1962, HODEK 1967, HAGEN and van den BOSCH 1968, GURNEY and HUSSEY 1970, HODEK 1970). The importance of coccinellids in controlling aphid populations in cages has been studied by HODEK et al. (1965) and SAILER (1966).

HODEK et al. (1965) concluded that temperature and other climatic factors have a great effect on the amount of aphids which the coccinellids are able to destroy. In 1964 the coccinellids were able to restrict the reproduction of aphids only when the number of aphids was less than 70 per one coccinellid. On the other hand, in 1965 the amount of aphids could be as much as 200 per coccinellid for growth of the aphid population to cease.

The aim of this study was to clarify the combined effect of the oat bird cherry aphid and the English grain aphid on barley and on the other hand to determine the ability of *Coccinella septempunctata* L. to limit the growth of the aphid populations.

Material and methods

The aphids belonged to parthenogenetic lines reared in a greenhouse for several years. Both lines were descended from one female. Aphids from the same lines have already been used in earlier studies (RAUTAPÄÄ 1966, 1968 a and b).

Non-viruliferous aphids were obtained in quantity as described earlier (RAUTAPÄÄ 1966). Until the start of the experiments the aphids were living on Sisu oat.

Adult coccinellids were collected from plants in the field. Before they were put into the experimental cages, the specimens were reared in the laboratory for a few days to separate the weak and sickly from the healthy ones. The sex of the coccinellids was not determined. The larvae were obtained from the laboratory rearings where they had been produced for other tests.

Pomo-barley was sown on May 17, 1971, in 26 cages set up on a loam soil. The cages (60 × 60 × 120 cm) consisted of a metal framework covered with terylene voile gauze. The number of grains evenly spaced in each cage was 3 × 64. The growing area of barley was 50 × 50 cm. The number of shoots was thinned on June 18 to 100 in each cage.

Four cages were chosen at random as controls without aphids or coccinellids. Other cages were grouped as follows (see also Table 1). All the aphids placed in cages were alate females.

A — Cages 1—4. Controls.
B — Cages 5—8. On June 29, either 1 or 3 *R. padi* were placed on each plant.
C — Cages 9—11. On July 8, either 1 or 3 *R. padi* or *M. avenae* were placed on each plant. No coccinellids were released into these cages.
D — Cages 12—18. On July 13, one *R. padi* was placed on each plant and on July 14 10, 15 or 20 coccinellid larvae were released into each cage.
E — Cages 19—26. On July 8, either 1 or 3 *R. padi* or *M. avenae* were placed on each plant and on July 12 five adult coccinellids were released into each cage.

In Table 1 the ratios between initial numbers of aphids and coccinellids are presented as fol-

Table 1. The initial numbers of aphids and coccinellids released into each cage. The ratios of the numbers at the start of the experiments are expressed as follows:

I = Number of coccinellids per 100 aphids.
II = Number of aphids per one coccinellid.
III = Number of aphids (per plant) per 100 coccinellids (per m^2).

Cage	Date	Number of aphids and coccinellids released into cages					Aphids per coccinellids		
		M. avenae	*R. padi*	Coccinellids					
				Date	Larvae	Adults	I	II	III
A 1		0	0		0	0			
2		0	0		0	0			
3		0	0		0	0			
4		0	0		0	0			
B 5	29. VI	0	100		0	0	0	0	0
6	29. VI	0	100		0	0	0	0	0
7	29. VI	0	300		0	0	0	0	0
8	29. VI	0	300		0	0	0	0	0
C 9	8. VII	100	300		0	0	0	0	0
10	8. VII	100	300		0	0	0	0	0
11	8. VII	300	0		0	0	0	0	0
D 12	13. VII	0	100	14. VII	10	0	10	10	2.5
13	13. VII	0	100	14. VII	20	0	20	5	1.3
14	13. VII	0	100	14. VII	10	0	10	10	2.5
15	13. VII	0	100	14. VII	10	0	10	10	2.5
16	13. VII	0	100	14. VII	15	0	15	6.5	1.7
17	13. VII	0	100	14. VII	15	0	15	6.5	1.7
18	13. VII	0	100	14. VII	15	0	15	6.5	1.7
E 19	8. VII	100	100	12. VII	0	5	2.5	40	10
20	8. VII	100	0	12. VII	0	5	5	20	5
21	8. VII	100	100	12. VII	0	5	2.5	40	10
22	8. VII	100	0	12. VII	0	5	5	20	5
23	8. VII	300	100	12. VII	0	5	1.3	80	20
24	8. VII	300	0	12. VII	0	5	1.5	60	15
25	8. VII	300	0	12. VII	0	5	1.5	60	15
26	8. VII	300	200	12. VII	0	5	1	100	25

lows: I) The number of coccinellids per 100 aphids, II) The number of aphids per coccinellid, III) The number of aphids (per plant) per 100 coccinellids (per m^2).

At the start of the experiments 30 plants were selected and marked at random in each cage. The number of aphids on the main shoot of these plants was counted at 3—7 days intervals (see Table 2). At the same time all the coccinellids in the cages were counted. Plants, soil and also the walls of the cage were checked. The yield was harvested on August 16. The heads were dried in a heating chamber at a temperature of 35 °C for over two weeks.

The brewing quality of selected samples of the yield was tested at the Brewing Laboratory Ltd. by EBC standard methods. The trace elements of the same samples were analysed at the Isotope Laboratory of the Agricultural Research Centre at Tikkurila. As it was impossible to make these tests for all the cages, cages were chosen which represented as well as possible various aphid indices (see Table 7). The yield of some cages were not sufficient for making the analyses and so the grain yields of some similar cages were bulked and mixed before sampling (combinations of cages presented in Table 7).

An index was calculated for each cage on the basis of the average number of aphids on one shoot and the time over which the aphids were present. The index represents the sum of the aphids living on one shoot on each day of the experiment. The method of calculating the

index has been explained earlier (RAUTAPÄÄ 1966), and it has been used also in other studies (RAUTAPÄÄ 1968 a and b, 1969, 1970).

The coccinellid index was calculated for each cage by the same method. It represents the sum of coccinellids (either larvae or adults) per square metre on each day of the experiment.

Results

Numbers of aphids on shoots

As a rule the numbers of aphids were the highest at the turn of July and August. The populations declined after the maximum was reached and no aphids were found at harvest time.

In general, there were more aphids of both species in the cages without coccinellids than in the ones with coccinellids (Table 2). The abundance of *R. padi* was the highest in the cages where three aphids were placed on each plant, i.e. in cages 9 (176.6 per shoot), 7 (90.8 per shoot) and 10 (85.5 per shoot).

Numbers of *M. avenae* were smaller than those of *R. padi* the maximum for the former being 54.2 per shoot (cage 22).

The aphid indices calculated for each cage are presented in Table 5. The index of *R. padi* was highest in cage 9 (2289). In cage 7 the index was almost as high (2275) and in cage 8 the corresponding value was 1325. In the other cages indices for *R. padi* were less than one thousand.

The highest index for *M. avenae* was in cage 22 (591).

Numbers of coccinellids in cages

Only a proportion of the initial number of coccinellids released into the cages could be found when checking (Table 2). No explanation could be found for even great variations (see for instance cages 16 and 18, Table 2).

In those cages (12—18) where only larvae were released, adults appeared after August 3 (see Table 2). In general, the numbers of adults in these cages were highest when the experiments were stopped in mid-August. However, in some cages no adults appeared (14 and 17). In cages where adult coccinellids were initially released (19—26), the first larvae were noticed on July 28. In all cages but number 28, larvae appeared on August 3 at the latest.

The maximum number of larvae observed when checking was 168/m^2 (cage 23) and in cage 20 the corresponding number was nearly the same (144/m^2). In other cages the maximum was at most a few dozen per square metre.

The coccinellid indices were calculated separately over the times when *R. padi* and *M. avenae* were present in the cage. As can be seen from Table 2 the start and end of *R. padi* and *M. avenae* populations did not always coincide. Also the coccinellid indices were calculated separately on the basis of the growth period of aphid populations (from the start to the maximum) and of the whole time the aphid populations was present (from the start till the aphids had disappeared).

Coccinellid indices calculated over the whole time the aphids were present are higher than those based on the time of growth of the aphid populations (Table 3). On the basis of the whole period the greatest total coccinellid index value for larvae and adults of *R. padi* is 1064 (cage 23). The corresponding figure for *M. avenae* is 1769 (cage 20).

The effect of coccinellids on the abundance of aphids

The correlation coefficients between aphid indices and coccinellid indices are presented in Table 4. *R. padi* indices are significantly correlated with some coccinellid indices, but the *M. avenae* indices are not. The correlation coefficients between the summed *R. padi* and *M. avenae* indices and the coccinellid indices are small

Table 2. Number of aphids (per shoot) and coccinellids (per m²) in cages. C-larvae = coccinellid larvae, C-ad = coccinellid adults.

	Cages		Aphids (per shoot) and coccinellids (per m²) released in cages	Number of aphids and coccinellids 29.6.	2.7.	6.7.	9.7.	12.7.	13.7.	15.7.	21.7.	28.7.	3.8.	9.8.	17.8.
B	5	*R. padi*	1	0	0.3	0.1	1.7			0.4	6.7	14.6	72.4	20.9	0
	6	*R. padi*	1	0	0.1	0.2	2.1			2.4	8.9	22.3	63.1	10.8	0
	7	*R. padi*	3	0	5.9	11.4	63.3			32.8	81.9	90.8	90.8	13.5	0
	8	*R. padi*	3	0	0.7	1.3	9.2			22.3	34.1	83.9	61.9	3.0	0
C	9	*R. padi*	3							10.9	33.1	176.6	133.3	0	0
		M. avenae	1							0.5	0.8	0.7	13.0	11.0	0
	10	*R. padi*	3							26.9	8.6	26.9	85.5	0	0
		M. avenae	1							4.0	1.2	0.4	12.9	16.7	0
	11	*M. avenae*	3							0.1	0.6	0.7	19.7	37.7	0
D	12	*R. padi*	1							0.3	2.4	5.7	29.9	8.3	0
		C-larvae	40						40	4	0	12	12	32	12
		C-ad	0										0	36	24
	13	*R. padi*	1							0.1	0.6	6.5	28.5	10.9	0
		C-larvae	80						80	4	0	28	32	16	8
		C-ad	0										0	8	20
	14	*R. padi*	1							0.1	0.0	1.2	4.4	67.3	0
		C-larvae	40						40	0	0	0	0	8	0
	15	*R. padi*	1							0.1	1.8	2.1	18.9	6.4	0
		C-larvae	40						40	0	0	12	8	0	0
		C-ad	0											0	8
	16	*R. padi*	1								0.4	0.9	6.3	41.4	0
		C-larvae	60						60		0	12	0	16	4
		C-ad	0										0	12	24
	17	*R. padi*	1							0.0	0.4	1.8	10.9	14.8	0
		C-larvae	60						60		30	0	4	0	0
	18	*R. padi*	1							0.8	0.7	0.5	10.4	26.5	0
		C-larvae	60						60		0	12	12	0	0
		C-ad	0										0	4	12
E	19	*R. padi*	1								0	5.4	64.9	0	0
		M. avenae	1							0.5	0.9	1.7	21.3	12.9	0
		C-larvae	0										0	12	4
		C-ad	20					20	8	4	0	0	0	0	0
	20	*M. avenae*	1							0.4	0.3	0.4	13.9	23.0	0
		C-larvae	0									0	44	72	144
		C-ad	20					20		4	8	4	0	8	36
	21	*R. padi*	1							0	17.2	21.5	45.1	0	0
		M. avenae	1							0.6	1.1	0.8	8.9	1.5	0
		C-larvae	0								0	8	8	16	0
		C-ad	20					20		8	16	4	8	4	0
	22	*M. avenae*	1							0.4	0.6	1.3	31.9	54.2	0
		C-ad	20					20					0	4	8
	23	*R. padi*	1									0	26.7	0	0
		M. avenae	3							0.8	0.9	1.9	32.7	13.0	0
		C-larvae	0									0	56	168	0
		C-ad	20					20		8	8	4	0	0	0
	24	*M. avenae*	3							1.0	1.6	1.4	26.2	45.9	0
		C-larvae	0								0	32	20	28	44
		C-ad	20					20		16	8	8	4	0	0
	25	*M. avenae*	3							0.5	0.5	0.8	13.8	28.3	0
		C-larvae	0									0	12	12	8
		C-ad	20					20		4	12	4	8	0	0
	26	*R. padi*	2							0	1.3	3.8	67.2	0	0
		M. avenae	3							1.1	2.9	5.2	36.8	28.1	0
		C-larvae	0									0	12	20	20
		C-ad	20					20		4	0	12	12	4	0

Table 3. Indices of coccinellids. For detailed information, see text. In cages 1—11 there were no coccinellids (see Table 1). C-larvae = coccinellid larvae, C-ad = coccinellid adults.

Cages	From the start to the maximum of aphid population						From the start to the end of aphid population					
	R. padi			*M. avenae*			*R. padi*			*M. avenae*		
	C-larvae	C-ad	Total	C-larvae	C-ad	Total	C-larvae	C-ad	Total	C-larvae	C-ad	Total
D 12	66	0	66	0	0	0	482	360	841	0	0	0
13	348	0	348	0	0	0	617	146	722	0	0	0
14	68	0	68	0	0	0	96	0	96	0	0	0
15	146	0	146	0	0	0	166	36	202	0	0	0
16	451	42	493	0	0	0	525	192	717	0	0	0
17	475	0	475	0	0	0	475	0	475	0	0	0
18	467	14	481	0	0	0	467	82	549	0	0	0
E 19	0	102	102	0	102	102	42	102	144	102	102	404
20	0	0	0	516	163	679	0	0	0	1416	353	1769
21	72	234	306	72	234	306	146	268	414	202	282	484
22	0	0	0	0	154	154	0	0	0	0	204	204
23	196	140	336	196	140	336	924	140	1064	1512	140	1652
24	0	276	276	434	222	656	0	0	0	738	222	969
25	0	0	0	114	198	312	0	0	0	192	198	390
26	42	180	222	42	180	222	142	218	360	302	232	534

and approach the lower limit of significance. It is characteristic that aphid indices are not correlated at all with adult coccinellid indices.

Indices for *R. padi* are best correlated with those coccinellid larvae indices determined on the basis of the whole time the *R. padi* populations were present. The correlation coefficient $r = -0.786$ ($P < 0.01$) and the regression equation $Y = 1021.7 - 1.368\ X$ (see Fig. 1). The aphid index thus decreases by about 137 units when the coccinellid index increases by 100 units.

R. padi indices are also significantly correlated with the summed indices for coccinellid larvae and adults, indices based on the complete duration of *R. padi* populations ($r = -0.610$, $P < 0.01$, Fig. 1). According to the equation $Y = 1083.5 - 1.170\ X$, the aphid index decreases by 117 units when the summed index of the coccinellid larvae and adults increases by 100 units.

Also correlations between the coccinellid indices calculated on the basis of the growth

Table 4. Correlation between aphid indices and coccinellid indices. A = coccinellid indices were calculated on the basis of the growth period of the aphid populations (from start of the experiment until the maximum), B = coccinellid indices were calculated on the basis of the whole time of aphid populations existed (from start untill the aphids had disappeared).
* $P < 0.05$, ** $P < 0.01$.

Aphid indices	Coccinellid indices	Correlation coefficients A	B
Only *R. padi*	larvae	-0.535*	-0.786**
	adults	± 0	± 0
	larvae + adults	-0.620**	-0.610**
Only *M. avenae*	larvae	± 0	± 0
	adults	± 0	± 0
	larvae + adults	± 0	± 0
R. padi + *M. avenae*	larvae	± 0	-0.416*
	adults	± 0	± 0
	larvae + adults	-0.414*	-0.451*

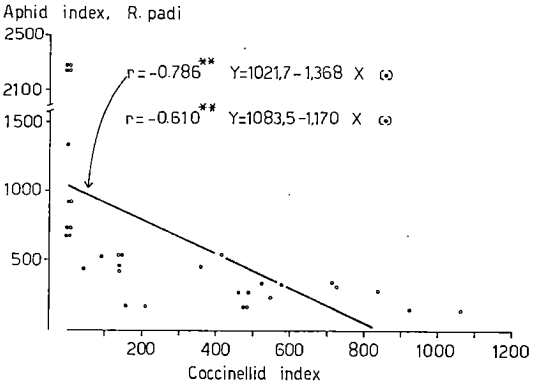


Fig. 1. The correlation between *R. padi* – indices and indices of coccinellid larvae (●) or the summed indices of larvae and adults (○). The coccinellid-indices are calculated on the bases of the total lifetime of aphid populations. Only the correlation between *R. padi* – indices and coccinellid larvae is presented by straight line.

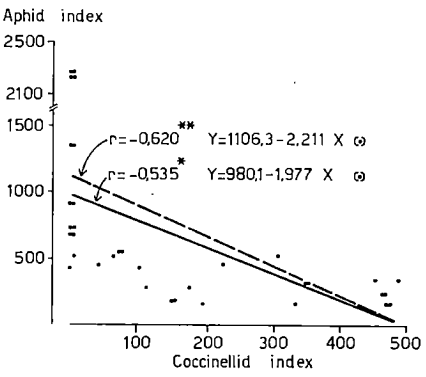


Fig. 2. The correlation between *R. padi*-indices and indices of coccinellid larvae (●) or the summed indices of larvae and adults (O). The coccinellid indices are calculated on the bases of the growth period of aphid populations (from the beginning of the experiments untill the greatest number of aphids on plants).

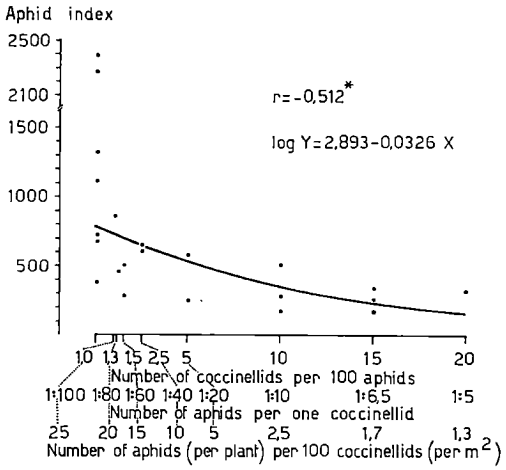


Fig. 3. The correlation between the summed (log) *R. padi*- and *M. avenae*-indices and the relation of the initial numbers of aphids and coccinellids released into the cages at the start of the experiments. For the detailed explanation, see the text.

phase of *R. padi* and the aphid populations are significant (Table 4, Fig. 2). When the *R. padi* indices and the coccinellid larvae indices are compared, $r = -0.535$ ($P < 0.05$) and the regression equation $Y = 980.1 - 1.977\ X$. This means that when the coccinellid larvae index increases by 100 units, the *R. padi* index decreases by 198. Correspondingly, the correlation between the *R. padi* index and the summed index for the coccinellid larvae and adults is significant ($r = -0.620$, $P < 0.01$). According to the regression equation $Y = 1106.3 - 2.211\ X$, the aphid index decreases by 221 units when the coccinellid index increases by a hundred.

The influence of coccinellids on the aphid populations was also studied by correlating the aphid indices with the numerical ratios of initial numbers of aphids to coccinellids released into cages at the beginning of the experiments (see Table 1). The logarithms of the summed indices of both aphid species are significantly correlated with the ratio of the initial numbers of aphids to coccinellids (larvae or adults), put into the cages. The curvilinear regression $\log Y = 2.893 - 0.0326\ X$ ($r = -0.512$; $P < 0.05$) is presented in Fig. 3. The regression equation was calculated by using numbers of coccinellids released into each cage per 100 aphids as values the independent variable (see Fig. 3).

According to the regression shown in Fig. 3, it is also possible to calculate by how many percent the aphid index decreases when the ratios between the initial numbers of coccinellids and aphids released into each cage changes. The decrease of the aphid index was calculated using the equation $X = 100\ (A-B)/A$, where X = the percentage decrease of the aphid index; $A = 2.893$, i.e. the value of log Y when $X = 0$ in the equation in the Fig. 3; B = the aphid index corresponding to a given value of X. The aphid indices decrease as follows:

Coccinellids per 100 aphids	Aphids per 1 coccinellid	Aphids (per plant) per 100 coccinellids (per m²)	Aphid index	% decrease of aphid index
0	0	0	782	0
1.0	100	25	725	7.3
1.3	80	20	709	9.3
1.5	60	15	699	10.6
2.5	40	10	647	17.3
5.0	20	5	538	31.2
10.0	10	2.5	369	52.8
15.0	6.5	1.7	254	67.5
20.0	5	1.3	174	77.8

According to this calculation the aphid index decreased by 7.3 percent when one coccinellid per 100 aphids was released into the cages. In other words, at the start of the tests there

Table 5. Effect of *R. padi* and *M. avenae* on the yield of Pomo-barley. For explanation of the aphid index, see text. r = coefficient of correlation between the aphid index and the yield. * $P < 0.05$, ** $P < 0.01$.

Cages	Aphid index			Grain yield per cage, g	Grain yield per head, g	Grain yield per plant, g	1000-grain weight, g	Number of plants per cage	Number of shoots per plant
	R. padi	*M. avenae*	Total						
A 1	0	0	0	165	1.21	2.07	33.4	80	1.8
2	0	0	0	99	0.81	0.96	27.3	103	1.2
3	0	0	0	99	0.74	0.93	24.4	107	1.4
4	0	0	0	257	0.96	2.36	27.6	109	2.9
B 5	731	0	731	134	1.06	1.33	31.4	101	1.4
6	680	0	680	130	0.70	1.34	21.2	97	2.2
7	2275	0	2275	69	0.59	0.66	17.1	104	1.2
8	1325	0	1325	55	0.45	0.52	17.6	105	1.3
C 9	2289	166	2395	102	0.85	1.13	24.7	91	1.51
10	901	201	1102	114	0.73	1.30	22.8	88	1.9
11	0	395	395	197	0.80	1.74	29.2	113	2.5
D 12	297	0	297	155	1.01	1.35	30.3	115	1.5
13	308	0	308	178	1.05	1.73	32.3	103	1.7
14	507	0	507	140	0.79	1.28	27.6	109	1.8
15	188	0	188	136	1.14	1.32	31.9	103	1.6
16	340	0	340	135	0.79	1.22	24.9	111	1.7
17	183		183	134	0.81	1.34	25.4	100	1.9
18	261		261	123	0.88	1.19	26.5	103	1.7
E 19	425	238	663	202	1.33	2.41	32.2	84	1.9
20	0	251	251	316	1.26	3.39	35.1	93	2.9
21	522	80	602	123	0.86	1.20	24.7	102	1.6
22	0	591	591	131	1.08	1.31	34.4	100	1.3
23	161	309	469	245	1.22	2.29	30.2	107	1.9
24	0	505	505	276	0.99	2.53	28.1	109	2.8
25	0	291	291	156	0.78	1.56	28.6	100	1.9
26	448	428	867	140	0.83	1.32	24.4	106	1.8
r Indices of *R. padi*				—0.625**	—0.450*	—0.018	—0.638**		
Indices of *R. padi* + *M. avenae*				—0.514**	—0.379	—0.108	—0.559**		

were 25 aphids on every plant and 100 coccinellids on an area of one square metre. The aphid index decreased logarithmically when the initial numbers of aphids put into cages were smaller. When there were 5 aphids per one coccinellid (at that time there were 1.3 aphids on each plant for 100 coccinellids on an area of m^2), the aphid indices decreased by approximately 77.8 percent.

The effect of aphids on the yield of barley

The grain yield of the cages is correlated significantly with both the *R. padi* indices and the summed indices for the both aphid species (Table 5). On the other hand, there is no significant correlation between yield and indices of *M. avenae*.

The logarithmic regression equation for the correlation of the *R. padi* indices with grain yields from the cages is $\log Y = 2.231 - 0.000175\,X$ ($r = -0.625$, $P < 0.01$, Fig. 5). The corresponding linear regression is $Y = 179.19 - 0.053\,X$ ($r = -0.533$, $P < 0.01$). Correspondingly, the correlation coefficient between the grain yield of the cages and the logarithm of the sum of *R. padi* and *M. avenae* indices is greater than the coefficient based on non-logarithmic values. The logarithmic equation is $\log Y = 2.234 - 0.00014\,X$ ($r = -0.514$, $P < 0.01$) and the corresponding linear equation $Y = 179.2 - 0.0427\,X$ ($r = -0.419$, $P < 0.05$). In Fig. 5 only curves for the logarithmic equations are presented.

The yield loss caused by aphids was calculated by means of the equation $X = 100\,(A - B)/A$. In this equation X = yield loss, A = the value of Y in the logarithmic equations above when $X = 0$, i.e. 2.234 or 2.231. Again B = the yield

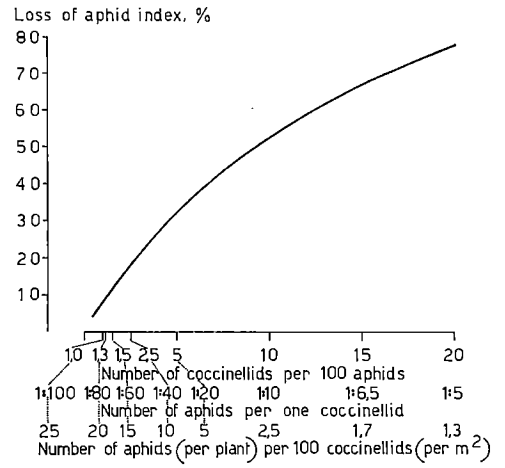


Fig. 4. The loss of aphid index (in percentage units) as a function of the relation of the initial numbers of aphids and coccinellids released into the cages.

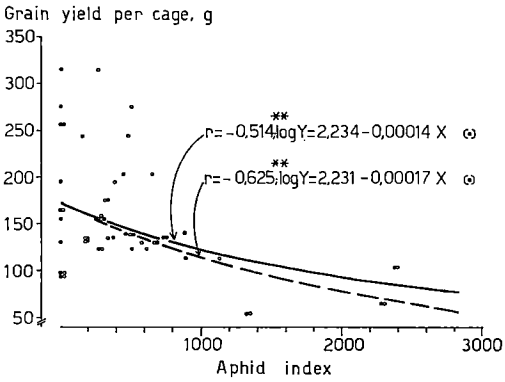


Fig. 5. The correlation between the grain yield of cages and *R. padi*- indices (●- - -●) or the summed indices of *R. padi* and *M. avenae* (O— —O).

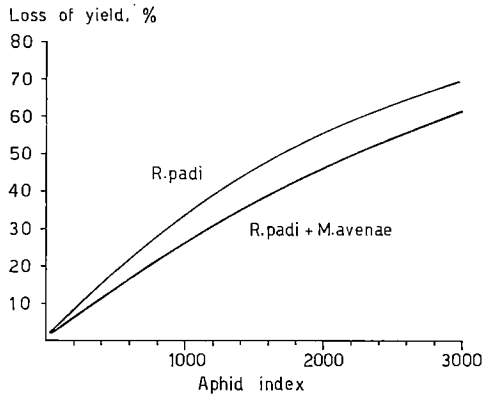


Fig. 6. Loss of grain yield as a function of *R. padi*-indices or the summed indices of *R. padi* and *M. avenae*.

corresponding to a given aphid index calculated with the aid of logarithmic equations presented earlier. In Fig. 6 the percentage yield loss is presented as a function of the aphid index.

When the yield of the cages is correlated only with *R. padi* indices, a variation of a given unit in the index causes a greater change in yield than if the yield were correlated with the sum of the indices for both aphid species.

The negative correlation between the *R. padi* indices and grain yield per head is significant ($r = -0.450$, $P < 0.05$) (Table 5). On the other hand, the grain yield per head is not correlated significantly with the sum of indices for both the aphid species ($r = -0.379$, $P > 0.05$).

Correlations between the yield of plants and the indices are not significant (see Table 5).

Neither the number of plants nor the number of shoots on the plants in cages at the harvest time are correlated significantly with aphid indices.

The 1000-grain weights are correlated significantly with the aphid indices (Table 5 and Fig. 7). The correlation coefficient between the *R. padi* index and 1000 grain weights is higher ($r = -0.638$, $P < 0.01$) than that for the summed incides for both the aphid species and the 1000 grain weight ($r = -0.559$, $P < 0.01$). The corresponding regression equations are $Y = 29.68 - 0.0048\ X$ (*R. padi* indices) and $Y = 29.94 - 0.0043\ X$ (the sum of incides for both the aphid species). An increase of a thousand units in the index thus causes either a 4.8 or a 4.3 g. decrease in the 1000 grain weight respectively.

The yields of the cages are correlated with the corresponding coccinellid indices. The correlation proved to be positive and highly significant ($r = 0.584$, $P < 0.01$, Fig. 8). According

Table 6. The percentage yield losses corresponding to some *R. padi* indices and summed indices for *R. padi and M. avenae*.

Index for *R. padi*	Loss of yield, %	Total of indices for *R. padi* and *M. avenae*	Loss of yield, %
100	3.9	100	3.1
500	17.8	500	14.4
1000	33.7	1000	26.9
2000	53.5	2000	46.6
3000	69.7	3000	61.2

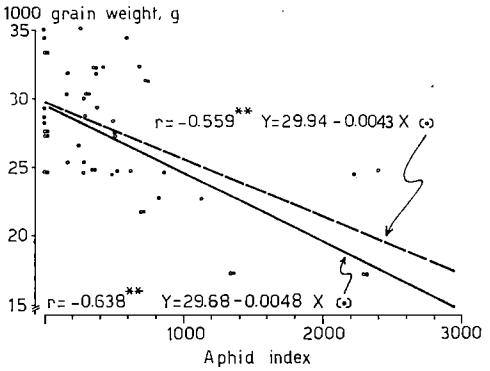


Fig. 7. The correlation between the 1000-grain weight and *R. padi*- indices (●—●) or the summed indices of *R. padi* and *M. avenae* (O—O)

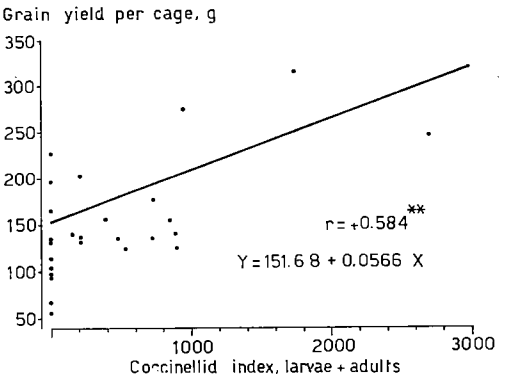


Fig. 8. The correlation between the grain yield per cage and the summed indices of coccinellid larvae and adults. The coccinellid-indices are calculated on the bases of the total lifetime of aphid populations.

to the regression equation $Y = 151.68 - 0.0566\,X$, an increase in the coccinellid index of 1000 units causes an average increase of 57 g in the grain yield of the cages.

Effect of aphids on the quality of barley

Only the extract content of malt is correlated significantly with both *R. padi* indices ($r = 0.738$, $P < 0.01$, Table 7) and with the sum of indices for both the aphid species ($r = -0.690$, $P < 0.01$). On the other hand, the correlation between the extract content of malt and *M. avenae* index is not significant ($r = -0.175$, $P > 0.05$).

The protein content, alpha-amylase activity, diastatic power of the malt and the malt yield

Table 7. Effect of *R. padi* and *M. avenae* on the brewing quality of Pomo-barley. For detailed information of the samples of grain and aphid index, see text. r = coefficient of correlation between the aphid index and the results of analyses. *P < 0.05, **P < 0.01.

Sample from cages	Extract content %	Protein content %	Alfa-amylase 20° D.U.	Diastatic power W.K.	Malt yield %	Aphid indices			1000-grain weight, g
						R. padi	*M. avenae*	Total	
1	79.1	10.1	91	270	86	0	0	0	33.4
4	74.9	12.9	128	470	85	0	0	0	27.6
2 + 3	75.1	9.5	100	340	84	0	0	0	25.9
5	78.0	10.7	104	310	85	731	0	731	31.4
7 + 8 + 9	67.8	13.2	131	380	78	1963	55	2018	19.8
10 + 6	72.2	9.6	104	300	83	780	100	880	22.0
11	74.8	12.4	125	430	85	0	395	395	29.2
12 + 13	76.3	11.1	113	360	86	302	0	302	31.3
15 + 17	76.6	11.3	116	400	86	185	0	185	28.7
18 + 16	73.0	10.8	131	460	85	300	0	300	25.7
19	77.2	10.5	108	390	86	425	238	663	32.2
20	78.1	11.3	111	400	87	0	251	251	35.1
21 + 14	73.4	13.1	131	450	84	514	40	554	26.2
22	77.9	11.8	104	360	86	0	591	591	34.4
23	76.5	12.4	119	400	85	161	309	469	30.2
24	75.9	11.1	113	380	85	0	505	505	28.1
25	76.2	11.1	104	380	84	0	291	291	28.6
26	73.3	10.3	119	390	83	448	428	867	24.4
Indices of	Correlation coefficients, r								
R. padi	— 0.738**	+ 0.227	+ 0.322	+ 0.105	— 0.263				
M. avenae	— 0.175	+ 0.086	— 0.070	+ 0.083	+ 0.048				
R. padi + *M. avenae*	— 0.690**	+ 0.274	+ 0.314	— 0.090	— 0.256				
1000-grain weight	+ 0.915**	+ 0.015	— 0.471*	— 0.010	+ 0.882**				

Table 8. The amounts of trace elements in barley grains (mg/kg dry weight). The figures represent the mean (in parenthesis minimum and maximum) for samples taken from control cages (A) and for cages with aphids (B—E). For detailed information of the samples, see text.

Samples	Mn	Fe	Na	Zn	Cu
A Control without aphids	24.1 (20.8—30.2)	37.9 (34.6—44.0)	46.2 (38.5—55.7)	40.0 (34.5—46.2)	5.8 (5.2— 6.4)
B—E Cages with aphids	26.3 (17.7—34.2)	38.3 (32.5—51.4)	43.7 (38.8—52.1)	43.6 (35.2—59.8)	5.7 (5.0—20.0)

are not correlated significantly with the aphid indices.

The lowest protein content of the barley samples was 9.5 % (cages 2 + 3; i.e. a control) and the highest 13.2 % (cages 7 + 8 + 9). The corresponding limits for the unit of alpha-amylase activity (20 D.U.) were 91 (cage 1; i.e. control) and 131 (several cages). The limits for the unit of diastatic power were 270 (cage 1; a control) and 470 (cage 4). Malt yield varied between 78 % and 87 %.

The extract content of malt, its alpha-amylase activity and malt yield are correlated significantly with the 1000 grain weight, i.e. the size of the grains. As the 1000 grain weight increases, the extract content of the malt and the malt yield increase but the alpha-amylase activity decreases. The protein content of the malt and the diastatic power are not correlated with the 1000 grain weight.

No connection was observed between the amounts of trace elements in the grain and the aphid indices. The quantities of all analysed trace elements varied within relatively wide limits (Table 8). Nor are the results of trace element analyses correlated significantly with the 1000 grain weight of the yield.

Conclusions

It is generally known that coccinellid larvae reduce the population growth of aphids more effectively than adults. Therefore, the significant negative correlation between certain aphid indices and coccinellid larvae indices is not an unexpected result. It seems extraordinary, however, that even in the closed environment of a cage the adult coccinellids had no measurable effect on aphid populations.

The coccinellid indices are correlated better with *R. padi* indices than with *M. avenae* indices or with the summed indices for both aphid species. Combining the indices for both species perhaps increased the range of the results. The aphid species may differ in their nutritive value for coccinellids, too. In the cages there were more *R. padi* than *M. avenae* and this difference in the abundance of the species also might have had an effect on the preference of coccinellids for aphids.

Coccinellids were not able to prevent entirely the growth of aphid populations but they suppressed it. The logarithm of the summed aphid indices is correlated significantly with the ratio of initial numbers of coccinellids to aphids released into cages at the beginning of experiments. Also, it is remarkable that those coccinellid indices based on the growing period of the aphid populations (from the start of tests to the aphid population maxima) are correlated significantly with the aphid indices. This implies that by means of adequate and consistent information it might be possible to prognose the index of the aphid population at an early stage and to predict the effects of aphids on cereals.

Until now, the effects of aphids on the yield and quality of cereals have been studied in four studies belonging to the same series. According to this experiment the effect of *R. padi* and the combined effect of both *R. padi* and *M. avenae* on barley are similar to the results obtained previously when studying the effects of *M. avenae* on barley (RAUTAPÄÄ 1968 a). An aphid index of one thousand units corresponds

in the present research to an yield loss of 33.7 percent (*R. padi*) or 26.9 percentage units (*R. padi* + *M. avenae*). The corresponding yield loss of barley was previously 27.3 percent. However, in earlier studies *M. avenae* decreased the yield of wheat less and *R. padi* had a smaller effect on oats, too (RAUTAPÄÄ 1966 and 1968 b). Yield losses corresponding to aphid indices of 1000 units were 10 and 9 percentage units, respectively, in these tests.

The aphids had no noticeable effect on the brewing quality of barley. The most important quality requirement for brewing malt is a high alpha-amylase activity. The alpha-amylase activity of all the samples analysed was rather high. Some values of the diastatic power were slightly low but the high alpha-amylase activity made the variations in diastatic power insignificant. The extract contents and the protein contents were quite low but the malt yield was still sufficient for practical needs. In general, all the analysed samples were suitable as distillery malts on the basis of their alpha-amylase activity, but because of the inconsistent and small grain size they would not have been suitable for brewing.

Acknowledgements. — I wish to express my sincerest thanks to Professor T.-M. Enari, D. Sc., Head of the Brewing Laboratory, for determining the brewing quality of barley. My warmest thanks are also due to the late Director of the Isotope Laboratory, Mr. Esko Lakanen, M. Sc., for analyses of the trace elements in barley samples, as well as to Mr. Martti Markkula, D. Sc., Dept. of Pest Investigation, and his colleagues for providing coccinellid larvae from laboratory rearings.

REFERENCES

ANGLADE, M. P. 1969. Essais d'estimation des dégate provoqués sur le Blé par la presence de colonies abondantes de "*Macrosiphum avenae*" Fab. Revue Zool. Agricole 68: 17—25.

DEAN, G. J. & LUURING, B. B. 1970. Distribution of aphids in cereal crops. Ann. Appl. Biol. 66: 485—496.

GRIGOROV, S. 1967. The effects of chemicals used for the control of cereal bugs and other cereal pests. Rastit. Zasht. 15: 23—27. (Ref. Rev. Appl. Ent. 58: 612).

GURNEY, B. & HUSSEY, N. W. 1970. Evaluation of some coccinellid species for the biological control of aphids in protected cropping. Ann. Appl. Biol. 65: 451—458.

HAGEN, K. S. 1962. Biology and ecology of predaceous *Coccinellidae*. Ann. Rev. Ent. 7: 289—326.

—& van den BOSCH, R. 1968. Impact of pathogens, parasites, and predators on aphids. Ann. Rev. Ent. 13: 325—384.

HAMILTON, E. W. & KIECKHEFER, R. W. 1969. Toxicity of malathion and parathion to predators of the English grain aphid. J. Econ. Ent. 62: 1190—1192.

HARVEY, T. L. & HACKEROTT, H. L. 1970. Chemical control of a greenbug on sorghum and infestation effects on yields. J. Econ. Ent. 63: 1536—1539.

HODEK, I. 1967. Bionomics and ecology of predaceous *Coccinellidae*. Ann. Rev. Ent. 12: 79—104.

— 1970. Coccinellids and the modern pest management. Bio Science 20: 543—552.

— NOVAK, K., SKUHRAVY, V. & HOLMAN, J. 1965. The predation of *Coccinella septempunctata* L. on *Aphis fabae* Scop. on sugar beet. Acta Ent. Bohemosl. 62: 241—253.

JONES, M. G. 1972. Cereal aphids, their parasites and predators caught in cages over oat and winter wheat crops. Ann. Appl. Biol. 72: 13—25.

KIECKHEFER, R. W. & MILLER, E. L. 1967. Trends of populations of aphid predators in South Dakota cereal crops — 1963—65. Ann. Ent. Soc. Amer. 60: 516—518.

KOLBE, W. 1969. Untersuchungen über das Auftreten verschiedener Blattlausarten als Ursache von Ertrags- und Qualitätsminderungen im Getreidebau. Pfl. schutz-Nachr. "Bayer" 22: 177—211.

LATTEUR, G. 1970. Les pucerons des cereales. Rev. l'Agric. 23: 1633—1646.

— 1971. Evolution des populations aphidiennes sur froments d'hiver (Gembloux, 1970). Med. Fac. Landbouw Rijksuniv. Gent 36: 928—939.

LECLANT, M. F. 1969. Une pullulation exeptionelle du Puceron "*Macrosiphum* (*Sitobion*) *avenae*" Fabricius sur Blé dans le Sud-Ouest de la France. Rev. Zool. Agricole 68: 13—16.

RAUTAPÄÄ, J. 1966. The effect of the English grain aphid *Macrosiphum avenae* (F.) (*Hom., Aphididae*) on the yield and quality of wheat. Ann. Agric. Fenn. 5: 334—341.

— 1968 a. Reduction in yield and changes in brewing quality of barley caused by *Macrosiphum avenae* (F.) (*Hom., Aphididae*). Acta Agric. Scand. 18: 233—241.

— 1968 b. Changes in the yield and protein quantity

of oat caused by *Rhopalosiphum padi* (L.) (*Hom., Aphididae*). Ann. Agric. Fenn. 7: 95—104.
— 1969. Effect of *Lygus rugulipennis* Popp. (*Hem., Capsidae*) on the yield and quality of wheat. Ann. Ent. Fenn. 35: 168—175.
— 1970. Effect of the meadow capsid bug, *Leptopterna dolabrata* (L.) (*Het., Capsidae*), on the yield and quality of wheat. Ann. Ent. Fenn. 36: 145—152.
SAILER, R. I. 1966. An aphid predator exclusion test. Proc. Symp. "Ecology of aphidophagous insects": 263. Academia, Prague.
STERN, V. M. & BOWEN, W. R. 1967. Control of aphids on barley. Economic treatment levels. Analysis of yield increase. Calif. Agric. 21: 14—15.
SANDERSON, F. R.& MULHOLLAND, R. I. 1970. The grain aphid and its control. New Zealand Wheat Rev. 11: 44—47.
TWINE, P. H. 1971. An insecticide trial against corn aphid (*Rhopalosiphum maidis*) (Fitch) on sorghum. Queensl. J. Agric.& Animal. Sci. 28: 19—22.
WARD, C. R., HUDDLESTON, E. W., ASHDOWN, D., OWENS, J. C. & POLK, K. L. 1970. Greenbug control on grain sorghum and the affects of tested insecticides on other insects. J. Econ. Ent. 63: 1929—1934.
WRIGHT, G. M. 1970. Effect of grain aphid on yield of wheat. New Zealand Wheat Rev. 11: 48.

MS received 20 November 1972

Jorma Rautapää
Agricultural Research Centre
Dept. of Pest Investigation
SF-01300 TIKKURILA, Finland

SELOSTUS

Seitsenpistepirkko tuomikirvan ja viljakirvan runsauden rajoittajana sekä kirvojen vaikutus ohran satoon ja laatuun

JORMA RAUTAPÄÄ

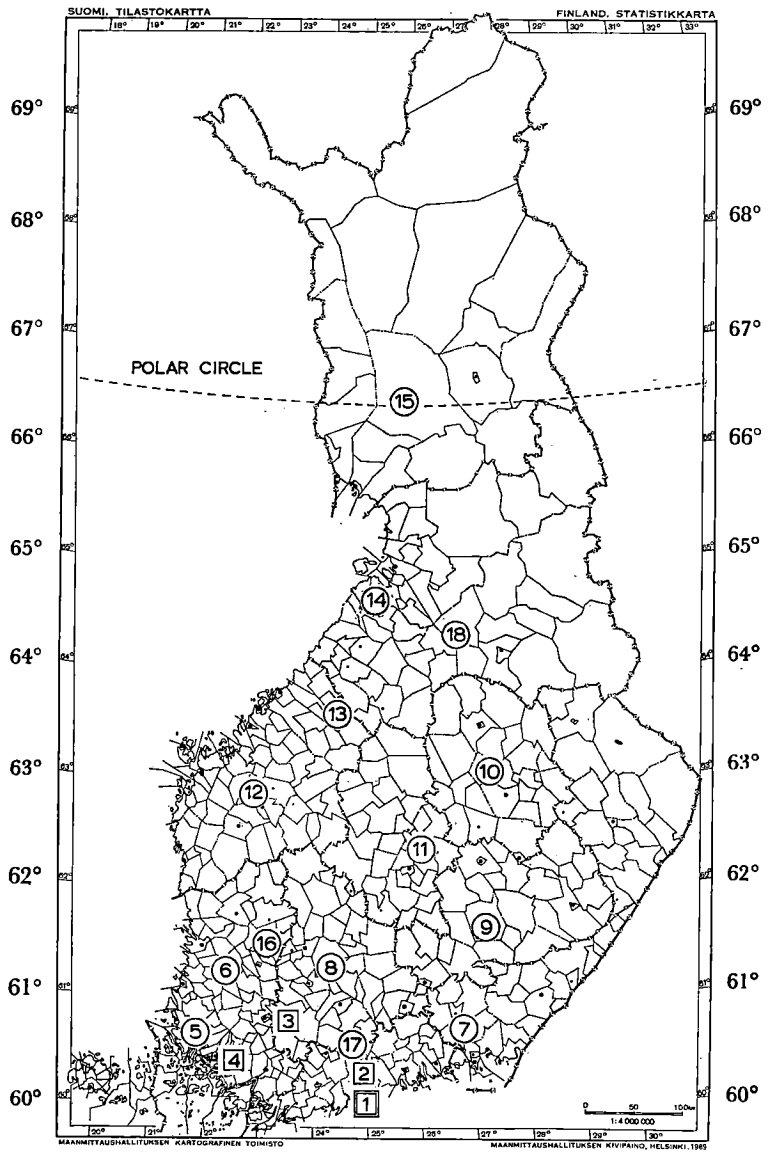
Maatalouden tutkimuskeskus, Tuhoeläintutkimuslaitos, Tikkurila

Tutkimuksessa selvitettiin häkkikokein yhtäältä seitsenpistepirkkojen (*Coccinella septempunctata* L.) kykyä rajoittaa tuomikirvan ja viljakirvan lisääntymistä ohrissa sekä toisaalta kirvojen vaikutusta ohran satoon sekä jyvien mallastumiseen ja hivenainepitoisuuteen.

Ohran versoissa eläneiden kirvojen määrän ja koeajan perusteella laskettiin häkeille indeksit, joilla tarkoitetaan koeajan kunakin päivänä yhdessä pääversossa eläneiden kirvojen summaa. Vastaavasti laskettiin häkeissä olleiden leppäpirkkojen määrien perusteella indeksit, jotka tarkoittavat koeajan jokaisena päivänä neliömetrin alueella olleiden leppäpirkkojen summaa. Leppäpirkkojen vaikutusta kirvojen runsauteen selvitettiin korreloimalla kirvaindeksit leppäpirkkoindekseihin. Vastaavasti tutkittiin kirvojen vaikutusta ohraan korreloimalla sadon määrä ja laatu kirvaindekseihin. Samalla tavoin laskettuja indeksejä on käytetty aikaisemmissa tutkimuksissa, joissa on selvitetty viljoissa elävien kirvojen sekä luteiden vaikutusta vehnän, kauran ja ohran satoon sekä laatuun. Näyttää siltä, että tällaisia indeksejä voidaan käyttää hyväksi arvioitaessa kirvojen tai luteiden mahdollista vaikutusta viljoihin.

Leppäpirkkojen toukat hidastivat tuomikirvan lisääntymistä, mutta leppäpirkkoaikuiset eivät vaikuttaneet kirvojen määriin. Viljakirvan lisääntymistä leppäpirkot eivät haitanneet. Kun leppäpirkkojen määrä häkissä oli kokeiden alkaessa kesä- ja heinäkuun vaihteessa 100/m² ja jokaisessa pääversossa oli 25 kirvaa, pieneni kirvaindeksi 7 %-yksikköä siitä, miksi se verrannehäkkien perusteella todennäköisesti olisi muodostunut. Vastaavasti kun leppäpirkkojen määrä oli sama 100/m² ja kirvoja oli 1.3/verso, jäi kirvaindeksi 78 %-yksikköä odotettua pienemmäksi. Tulosten perusteella laskettiin yhtälö, joka kuvaa leppäpirkkojen vaikutusta kirvapopulaatioihin.

Kirvat alensivat häkkien jyväsatoa ja 1000-jyvän painoa merkitsevästi. 1000-yksikön suuruista kirvaindeksiä (esim. 50 kirvaa elää kasveissa 20 päivän ajan) vastasi 27 %:n sadonalennus. Kirvat vähensivät maltaan uutepitoisuutta, mutta eivät muuttaneet muita mallastumisominaisuuksia. Ohrien hivenainepitoisuuteen kirvat eivät vaikuttaneet.



DEPARTMENTS, EXPERIMENT STATIONS AND BUREAUX OF THE AGRICULTURAL RESEARCH CENTRE IN FINLAND

1. Administrative Bureau, Bureau for Local Experiments (HELSINKI) — 2. Departments of Soil Science, Agricultural Chemistry and Physics, Plant Husbandry, Plant Pathology, Pest Investigation, Animal Husbandry and Animal Breeding; Isotope Laboratory, Pesticide Regulation (TIKKURILA) — 3. Dept. of Plant Breeding (JOKIOINEN) — 4. Dept. of Horticulture (PIIKKIÖ) — 5. Southwest Finland Agr. Exp. Sta. (HIETAMÄKI) — 6. Satakunta Agr. Exp. Sta. (PEIPOHJA) — 7. Karelia Agr. Exp. Sta. (ANJALA) — 8. Häme Agr. Exp. Sta. (PÄLKÄNE) — 9. South Savo Agr. Exp. Sta. (Karila, MIKKELI) — 10. North Savo Agr. Exp. Sta. (MAANINKA) — 11. Central Finland Agr. Exp. Sta. (VATIA) — 12. South Ostrobothnia Agr. Exp. Sta. (PELMA) — 13. Central Ostrobothnia Agr. Exp. Sta. (LAITALA) — 14. North Ostrobothnia Agr. Exp. Sta. (RUUKKI) — 15. Arctic Circle Agr. Exp. Sta. (ROVANIEMI) — 16. Pasture Exp. Sta. (MOUHIJÄRVI) — 17. Pig Husbandry Exp. Sta. (HYVINKÄÄ) — 18. Frost Research Sta. (PELSONSUO)

SISÄLLYS — CONTENTS

Suomalaisen Kirjallisuuden Kirjapaino Oy Helsinki 1972