"Though a documentary, it's dramatic enough to be be reminiscent of 'The Insider,' the whistleblowing thriller about Big Tobacco."

Graham Fuller, New York Daily News – 8/28/14

SECOND OPINION

LAETRILE AT SLOAN-KETTERING

"Lying to the American people wasn't part of my job description."

Ralph W. Moss, PhD

The following pages contain the "leaked documents package" showing the positive Laetrile experiments at Memorial Sloan-Kettering Cancer Center, under Dr. Kanematsu Sugiura.

Second Opinion: Laetrile At Sloan-Kettering is available on Blu-ray, DVD, and Video On Demand.
Also available is ***Doctored Results***, a new book & companion guide to this documentary by Ralph W. Moss, PhD

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ANATOMY OF A COVERUP

SUCCESSFUL SLOAN - KETTERING AMYGDALIN (LAETRILE) ANIMAL STUDIES

SLOAN - KETTERING

ANIMAL TEST

MEMORIAL SLOAN-KETTERING CANCER CENTER
1275 YORK AVENUE, NEW YORK, NEW YORK 10021
(212) 879-3000

AUG 2 3 1975

Dear Mr. Culbert:

Here are some the results of Sloan-Kettering's continuing experiments with Laetrile. Due to political pressure these results are being suppressed. Please do your best to bring these important findings to the attention of the people.

Krebs' theory is very promising, and Laetrile should be tested clinically to see if it really holds water.

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Memorial Hospital for Cancer and Allied Diseases
Sloan-Kettering Institute for Cancer Research
Sloan-Kettering Division, Graduate School of Medical Sciences, Cornell University

We received 60 female CD_8F_1 mice from Dr. Daniel S. Martin of the Catholic Medical Center of Brooklyn and Queens, New York, on May 4th 1973 for our experiment with Amygdalin. These female mice were born in December 1972.

We separated these mice into two groups--30 mice for controls which received daily intraperitoneal injections (except Sundays) of saline for 8 weeks or more and the other 30 mice received 2000 mg/kg/day/mouse of Amygdalin for the same period. These animals were weighed once weekly and examined for development of tumors. About 30% of these animals were pregnant.

The purpose of this experiment will be to find out the effect of Amygdalin on the development of spontaneous mammary cancer and lung metastases. The experiment was started on May 8, 1973.

On May 8 to July 9 (62 days) both control and experimental animals maintained body weight well. General health and appearance of Amygdalin-treated animals and that of the controls were good. However, 5 of 30 mice in the experimental group died during this period. Therefore, the dose was reduced to 1000 mg/kg/day. The sudden deaths of these animals might be due to the insertion of the needle into the intestine or uterine horn of these pregnant mice. Therefore, 1/2 inch, 23 gauge hypodermic needles (Becton, Dickinson and Company) were changed to 1/4 inch needles.

During the course of experimenting, we determined the effect of oral administration of Amygdalin on mice.

Each test consisted of 2 Balb x C57 Bl. mice. Amygdalin solutions were given once daily. Results showed that oral administration of 2000 and 1000 mg/kg/day of Amygdalin caused the death of animals in 1 hour. With a dose of 500 mg/kg/day animals lived for 1 hour but died between 2 to 3 hours after oral administration. All animals showed lung hemorrhage. With doses of 250, 100, and 50 mg/kg/day animals lived indefinitely.

Daily examination of Amygdalin treated animals and control animals (August 2, 1973 or 86 days since the start of the experiment) revealed no evidence of development of spontaneous mammary tumors in these animals. In August the mice will be 8 months old, and I expect appearance of spontaneous mammary tumors in the control group.

Histological examinations of mammary tumors of the First Experiment (September 12, 1972 show all adenocarcinomas. Tumor cells of untreated controls are very active and have many mitotic figures. On the other hand tumor cells of Amygdalin treated animals are not very active, more hemorrhagic and degenerated and contain less mitotic figures.

Histological examinations of lungs of the control animals and Amygdalin-treated animals for lung metastases revealed good agreement with that of gross findings.

I will prepare shortly an observation summary on the effect of Amygdalin on spontaneous mammary tumors in Swiss albino mice.

Kanematsu Sugiura

August 3, 1973

SLOAN-KETTERING INSTITUTE *for* CANCER RESEARCH

DONALD S. WALKER LABORATORY, 145 BOSTON POST RD., RYE, N.Y. 10580 OWENS 8-1100

Effect of Amygdalin on Spontaneous Mammary Tumors in CD_8F_1 Mice

This report consists of observations on the effects of prolonged treatment with Amygdalin (SK 1691B) on the growth of spontaneous mammary tumors (adenocarcinomas) in female CD_8F_1 mice. The diagnoses of the tumor tissues were made from biopsied tissues or by postmortem microscopic examination of tissues at the end of the experimental period. The controls received carboxymethylcellulose (CMC) daily and the experimental animals received 1000 mg/kg/day of Amygdalin daily intraperitoneally (6 times weekly). The animals were kept on a normal diet (Purina Laboratory Chow) and water.

The results obtained in the September 12, 1972 experiment are summarized in Tables 1 and 2. Nine control mice with 17 tumors (2.8 x 2.1 cm., the largest to 0.9 x 0.6 cm., the smallest) and ten experimental mice with 15 tumors (1.8 x 1.5 cm., the largest to 0.7 x 0.9 cm., the smallest) were used.

Mouse No. 4 died within 7 days after start of the experiment, and therefore, it was not included in the results.

Table 2 shows that repeated intraperitoneal injections of 1000 mg/kg/day of Amygdalin for 2 to 15 weeks failed to destroy the spontaneous cancer in mice. However, it caused an inhibition in about 50 percent of the tumors. It also shows Amygdalin had a strong inhibitory effect on the development of new tumors and on lung metastases (11% against 89%) in mice. The general health and appearance of the Amygdalin-treated animals with tumors was much better than that of the controls.

Kanematsu Sugiura

Kanematsu Sugiura
March 1, 1974

Table 1

CD_8F_1 Mammary Tumors (Adenocarcinomas) Controls

Mouse No.	Size of tumor (cm)	No. of injections	Duration of experiment (days)	Tumor growth	Final tumor size (cm).	Lung metas.*	Terminated
1	0.2 x 0.2 0.8 x 1.0 11/7‡	65	77	all grew	4.3 x 2.9 1.5 x 1.7	++	died
	0.9 x 0.6	72	86	grew	4.7 x 3.0	++	died
3	1.1 x 1.0 0.9 x 0.7 9/19 0.8 x 0.8 9/26	59	64	all grew	2.6 x 2.5 1.8 x 2.7 3.6 x 2.9	+	died
4	2.6 x 3.0	2	2	grew	3.0 x 3.5	-	died
5	1.5 x 1.2 0.8 x 1.0 10/24	39	46	all grew	4.3 x 3.7 1.0 x 1.0	++	died
6	1.3 x 0.9	79	92	grew	4.4 x 3.6	+++	died
-	2.8 x 2.1	17	20	grew	4.4 x 2.8	-	died
8	0.7 x 0.5 1.1 x 1.4 10/10	42	50	all grew	3.3 x 3.8 1.9 x 2.4	++	died
9	1.2 x 1.3 0.9 x 1.2 10/17	49	58	all grew	3.1 x 3.7 1.9 x 1.6	+++	died
0	1.1 x 0.9 2.0 x 1.5 9/26 0.6 x 0.6 9/26	17	20	all grew	1.5 x 1.3 3.3 x 2.6 1.4 x 1.6	+	died

Injections of CMC were started on September 12, 1972 and ended December 13, 1972 or when animals died.

* Evaluation of lung metastases: (+++) = More than 10 nodules in the lung.
(++) = More than 5 nodules in the lung.
(+) = Less than 5 nodules in the lung.
(-) = No nodules in the lung.

‡ Date new tumor found.

Table 2

CD8F1 Mammary Tumors (Adenocarcinomas) treated with 1000 mg/kg/day of Amygdalin

Size of tumor (cm)	No. of injections	Duration of experiment (days)	Tumor Growth	Final tumor size (cm)	Lung metas.*	Terminated
1.4 x 1.5 0.7 x 0.8	63	74	all grew	4.1 x 3.1 2.8 x 2.2	-	Died
1.3 x 1.2	18	21	stopped 21d Δ	1.0 x 1.2	-	Died
1.3 x 1.2 0.8 x 1.1 9/14‡	24	28	all grew	1.8 x 1.8 2.9 x 3.0	-	Died
1.0 x 0.6	68	80	stopped 21d	4.8 x 2.7	+	Died
0.7 x 0.9	105	140	stopped 56d	2.5 x 2.8	-	Died
0.9 x 0.9	14	16	grew	1.0 x 1.6	-	Died
1.8 x 1.5	57	66	stopped 21d	4.3 x 2.8	+	Died
0.9 x 0.8	28	32	grew	2.7 x 1.7	+	Died
1.0 x 0.8	29	34	stopped 34 d	1.1 x 0.9	-	Died
0.8 x 0.7 0.4 x 0.4 0.9 x 1.0 9/17 1.1 x 0.7 10/10	42	50	all grew	2.1 x 1.7 1.9 x 1.6 2.2 x 3.1 1.2 x 0.9	-	Died

Injections of Amygdalin were started on Sept. 12, 1972 and ended on Jan. 30, 1973 or when animals died.

‡ Date for new tumor found.

Δ Tumor growth stopped for indicated number of days, then growth resumed.

SLOAN-KETTERING INSTITUTE *for* CANCER RESEARCH

DONALD S. WALKER LABORATORY, 145 BOSTON POST RD., RYE, N.Y. 10580 OWENS 6-1100

Effect of Amygdalin on Spontaneous Mammary Tumors in CD8F1 Mice

On April 13, 1973 we received 20 female CD8F1 mice bearing spontaneous mammary tumors from Dr. D. S. Martin of Catholic Medical Center of Brooklyn and Queens, New York. Fourteen of 20 mice or 70% had already 2 to 3 spontaneous mammary carcinomas, indicating that these mice are older than those used in the previous two experiments (September 12, 1972 and February 20, 1973). Primary tumors in this group were definitely larger than those of the previous two groups.

Ten control mice with 19 tumors (2.6 x 2.4 cm., the largest to 0.6 x 0.5 cm., the smallest) received CMC daily intraperitoneally and 10 experimental mice with 18 tumors (3.4 x 2.7 cm., the largest to 1.1 x 0.8 cm., the smallest) received 2000 mg/kg/day of Amygdalin daily intraperitoneally except Sundays for 4 weeks. Four control animals and 1 experimental animal died within 7 days after start of the experiment and, therefore, they were not included in the results.

The results obtained are summarized in Tables 1 and 2 (April 19, 1973). It shows that repeated intraperitoneal injections of 2000 mg/kg/day of Amygdalin for 4 weeks failed to destroy the spontaneous mammary cancer in mice. All tumors grew normally (see Table 2). However, it shows a strong inhibitory effect on the development of lung metastases in mice - 22% against 100%. The general health and appearance of the Amygdalin-treated animals was much better than those of the controls.

Kanematsu Sugiura

Kanematsu Sugiura
March 5, 1974

CD8F1 Mammary Tumors (Adenocarcinomas) Controls

Size of tumor (cm)	No. of injections	Duration of experiment (days)	Tumor growth	Final tumor size (cm)	Lung metas.*	Terminated
1.5 x 1.4 0.9 x 1.3	31	36	all grew	2.4 x 2.3 1.8 x 1.9	+	Sac.
2.1 x 1.6 0.8 x 0.8 0.8 x 0.7	16	19	all grew	3.0 x 2.4 1.8 x 1.4 1.7 x 1.3	++	Sac.
2.3 x 2.0 2.0 x 1.6	6	7			-	Died
2.0 x 1.7	30	35	grew	4.1 x 3.3	+	Sac.
2.6 x 2.4 0.6 x 0.5	10	12	grew	3.0 x 2.9 0.9 x 0.7	++	Died
1.9 x 1.5	4	5			-	Died
1.8 x 2.6 1.2 x 1.2 1.0 x 1.0	13	15	all grew	2.1 x 3.1 1.5 x 1.6 1.6 x 1.4	++	Died
1.3 x 1.5 0.9 x 0.8 1.4 x 1.15/10‡	30	35	all grew	3.1 x 3.5 1.8 x 1.5 1.5 x 1.4	++	Died
1.6 x 1.6	5	6			-	Died
2.1 x 2.3 1.9 x 1.7	1	2			+	Died

ections of CMC were started on April 19, 1973 and ended on May 24, 1973 or when animals died.

* Evaluation of lung metastases: (+++) = more than 10 nodules in the lung; (++)=more than 5 nodules in the lung; (+) = less than 5 nodules in the lung; (-)=no nodules in the lung.

‡ Date for new tumor found.

CD_8F_1 Mammary Tumors (Adenocarcinomas) Treated with 2000 mg/kg/day of Amygdalin

Size of tumor (cm)	No. of injections	Duration of experiment (days)	Tumor Growth	Final tumor size (cm)	Lung metas.*	Terminated
1.1 x 1.3 1.4 x 1.3	12	14	All grew	1.4 x 1.7 1.9 x 2.0	-	Died
1.0 x 1.0 1.6 x 1.5	29	35	All grew	1.7 x 2.3 2.4 x 3.7	+	Sac.
1.9 x 1.9 1.2 x 1.4 1.7 x 1.1	14	18	All grew	2.5 x 2.2 2.0 x 1.5 3.1 x 1.8	-	Died
0.9 x 0.9 0.9 x 1.2 5/17‡ 0.8 x 0.6 5/17	25	30	Stopped 7d$^{\Delta}$	1.4 x 1.6 1.1 x 1.5 0.8 x 0.7	-	Died
1.6 x 1.4 1.4 x 0.9	17	21	All grew	2.0 x 1.4 1.8 x 1.3	-	Died
1.5 x 1.6 1.1 x 1.0	6	6	All stopped	1.4 x 1.6 0.9 x 1.0	-	Died
3.4 x 2.7 1.5 x 1.2	26	30	All grew	4.2 x 4.4 2.2 x 1.7	-	Died
1.8 x 1.4	16	19	Grew	3.1 x 2.2	+	Died
1.2 x 0.9 1.0 x 1.0	20	25	All grew	1.6 x 1.5 2.0 x 1.6	-	Died
1.1 x 0.8 1.1 x 0.7 5/3	30	36	Stopped 7d Grew	1.9 x 1.4 1.3 x 1.0	-	Sac.

ections of Amygdalin were started on April 19, 1973 and ended on May 24, 1973 or when animals died.

‡ Date for new tumor found.

Δ Tumor growth stopped for indicated number of days, then growth resumed.

SLOAN-KETTERING INSTITUTE *for* CANCER RESEARCH

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Effect of Amygdalin on Spontaneous Mammary Tumors in CD_8F_1 Mice

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Recently we undertook 3 separate experiments (2/22/74, 3/4/74, and 3/11/74) on the effects of prolonged treatment with amygdalin of Mexican origin and German origin (racemic compound) on the growth of spontaneous mammary tumors (adenocarcinomas) in female CD8F1 mice. Each set consisted of 10 controls receiving 0.5cc of saline daily (except Sundays) intraperitoneally and 10 experimental animals which received 2000 mg/kg/day of amygdalin (Mexican or German). The animals were kept on normal diet (Purina Laboratory Chow) and water.

When primary tumors became large (generally more than 4 weeks from the start of the experiments and having tumors more than 2.5 cm. in diameter) animals are sacrificed and negative lungs are bioassayed (1) for the presence or absence of metastases. However, when animals died the lungs were examined grossly with the aid of a magnifying glass and histologically for metastases.

It is interesting to note that 29 negative lungs examined by bioassay 5 or 17% developed tumors or incorrectly diagnosed by gross examinations. Therefore, the positive lung metastases were corrected in the results.

The results in the February 22, 1974 experiment in respect to lung metastases are summarized in Tables 1, 2 and 3.

The Table results show that repeated intraperitoneal injections of 2000 mg/kg/day of Amygdalin for 4 to 9 weeks had a strong inhibitory effect on the development of lung metasatses.

Controls=8 positive, 2 negative or 20% no metastases; amygdalin (Mexican)=3 positive, 7 negative or 70% no metastases; amygdalin (German)=2 positive, 8 negative or 80% no metastases.

The preceeding experiment (February 22, 1974) was repeated (March 4, 1974) using 30 female CD_8F_1 mice bearing spontaneous mammary tumors. Controls received saline daily except Sundays and experimental animals received 2000 mg/kg/day of amygdalin (Mexican) or amygdalin (German) daily intraperitoneally.

The results obtained in the March 4, 1974 experiment in respect to lung metastases, are summarized in Tables 4,5 and 6.

The Table results show that repeated intraperitoneal injections of 2000 mg/kg/day of amygdalin for 4 to 9 weeks had a strong inhibitory effect on the development of lung metastases. Controls=8 positive, 1 negative or 11% no metastases; amygdalin (Mexican)=2 positive, 7 negative or 78% no metastases; amygdalin (German)=3 positive, 7 negative or 70% no metastases.

The results in the March 11, 1974 experiment in respect to lung metastases are summarized in Tables 7, 8 and 9.

The Table results show that repeated intraperitoneal injections of 2000 mg/kg/day of amygdalin for 4 to 9 weeks had a strong inhibitory effect on the development of lung metastases. Controls=9 positive, 1 negative or 10% no metastases; amygdalin (Mexican)=4 positive, 5 negative or 56% no metastases; amygdalin (German)=3 positive, 7 negative or 70% no metastases.

The present 3 experiments show that the anti-lung metastasis activity of amygdalin of Mexican or German product appears to be the same - 68 and 73% no metastases, respectively, against 14% no metastases for controls.

On May 31, 1974, one animal in the control group and 2 animals in the amygdalin (Mexican)-treated group out of 90 animals are still living.

Kanematsu Sugiura

Kanematsu Sugiura
May 31, 1974

1) Anderson, J. C., Fugmann, R. A., Stolfi, R. L., and Martin, D. S. Metastatic Incidence of a Spontaneous Murine Mammary Adenocarcinoma. Cancer Research, 1974 (in press).

Table 1 (First experiment 2/22/74)

CD8F1 Mammary Tumors (Adenocarcinomas) Controls

Mouse No.	Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Lung metastasis: Gross exam.*	Lung metastasis: Microscopic exam.	Lung metastasis: Biopsy†	Terminal
1	0.7 × 0.9	2.5 × 2.9	90	+++	+		Sac.
2	0.9 × 1.0	2.0 × 2.4	32	++	+		Died
3	0.4 × 0.4	2.4 × 3.0	55	+	—		Sac.
4	0.8 × 1.3 0.7 × 1.1 (3/8)Δ	1.9 × 2.8 1.0 × 1.6	32	++	+		Sac.
5	1.0 × 0.9	2.2 × 2.8	32	—		—	Sac.
6	1.4 × 1.1	2.9 × 2.4	39	—		—	Sac.
7	1.0 × 1.1 0.9 × 0.7 (5/2)Δ	1.8 × 1.9 3.4 × 2.5	38	++	+		Died
8	1.0 × 0.8	3.9 × 2.8	55	++	+		Sac.
9	0.7 × 0.6 0.4 × 0.4 (5/1)Δ	2.2 × 2.4 1.2 × 1.3	39	+	—		Sac.
10	0.8 × 0.8	2.0 × 2.0	28	+	+		Died

* Evaluation of lung metastasis: (+++) = More than 10 nodules in the lung; (++) = more than 5 nodules; (+) = less than 5 nodules; (—) = no nodules.

† Biopsyed one negative lung in 2 male mice (2 implants in one mouse bilaterally.

Δ Date for new tumor found.

Table 2 (First Experiment 2/22/74)

CD8 F1 Mammary Tumors (Adenocarcinoma) Treated with 2000 mg/kg/day of Cyclophosphamide (Mpfac)

Animal No.	Initial Size of Tumor (cm)	Final Size of Tumor (cm)	Duration of Experiment (days)	Lung metastases: Gross * Appearance	Lung metastases: Microscopic Appearance	Kidney ‡	Terminated
1	0.8 X 0.7	1.8 X 1.4	+7	—	—		Died
2	1.0 X 0.8	2.1 X 1.6	36	—	—		Died
3	0.8 X 0.8	3.0 X 2.7	77	+	+		Sac.
4	1.0 X 0.7	3.3 X 2.6	90	+	+		Sac.
5	0.7 X 0.5	2.2 X 1.4	69	—	+		Died
6	1.0 X 0.7	2.7 X 1.8	55	—		+	Sac.
	0.8 X 1.0 3/4 △	1.6 X 1.6					
7	1.0 X 1.1	1.9 X 2.3	26	—	—		Died
	0.8 X 0.7 3/5 △	0.8 X 1.2					
8	1.1 X 1.0	3.0 X 2.7	75	—		—	Sac.
9	1.1 X 0.9	3.1 X 2.0	28	—		—	Sac.
10	0.9 X 0.8	1.8 X 2.2	59	—	—		Died

*

‡

△

Table 3 (First Experiment 2/22/74)

CD8F1 Mammary Tumors (Adenocarcinomas) Treated with 2000 mg/kg/day of Cyclophosphamide (Geomax)

Mouse No.	Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Lung metastasis: Gross appear.*	Lung metastasis: Microscopic appear.	Lung metastasis: Bleeding†	Termination
1	0.8×0.9 △ 0.7×0.7 3/8 ○ 0.8×0.9 3/4	2.2×2.1 1.2×1.3 0.8×0.9	39	—		—	Sac.
2	0.7×1.1	2.2×3.6	55	+++	+		Sac.
3	1.1×0.8 △ 0.7×0.8 3/8	1.6×1.6 2.2×2.6	39	—		—	Sac.
4	1.8×1.3	2.5×2.9	32	—		—	Sac.
5	0.9×1.1 △ 0.4×0.5 3/4	2.4×1.8 2.5×2.1	60	—		—	Sac.
6	0.9×1.1	3.1×2.4	67	—		—	Sac.
7	1.0×1.4	2.7×2.0	32	—		—	Sac.
8	0.8×0.8 △ 1.1×0.9 4/5	2.6×2.3 1.4×1.4	60	—		—	Sac.
9	0.9×0.8	2.3×3.3	51	—	—		Died
10	0.8×1.1	2.3×2.8	55	+++	+		Sac.

*

†

Effect of Amygdalin on the Development of Mammary Tumors (Adenocarcinomas) and Lung Metastasis in CD8F1 Mice.

On May 8, 1973, we started a new experiment to find out the effect of amygdalin (Mexican) on the development of spontaneous mammary cancer and lung metastasis in female CD8F1 mice. At the start of the experiment these mice were approximately 5 months old and had no spontaneous tumors. These mice had at least one pregnancy.

Thirty mice (8 mice were pregnant) for controls which received daily intraperitoneal injections of 0.5 cc saline (6 times weekly) for a prolonged period and the other 30 mice (8 mice were pregnant) received 1000 mg/kg/day of amygdalin daily intraperitoneally for the same period as controls. These female mice were born during December 1972. When tumors developed in these anaimals they were allowed to grow to a large size which took more than 21 days. The presence or absence of lung metastases was determined by gross and histologic examination. The animals were kept on a normal diet (Purina Laboratory Chow) and water.

When animals appeared to be weak due to the presence of lung metastases or due to toxemia from large tumors (2.0 cm. diameter or more) animals were sacrificed and gross examination was made for the presence and absence of metastases.*

Results: Daily examination of amygdalin-treated animals as well as controls (the last examination was made on September 30, 1974 or 510 days since the start of the experiment) revealed development of 19 spontaneous mammary tumors and 2 abdominal tumors in 30 mice among the control group. First tumor appeared on 10/11/73, followed by 12/8/73, 12/20/73, etc., - see Table 1. By September 30, 1974, 21 of 30 control animals developed tumors or 70 per cent. Three of them had second tumors. Of the 18 animals that died or were sacrificed because of large tumors, 14 had lung metastases in various degrees, or 78 per cent. Twelve animals are still alive with or without tumors.

Among 30 experimental animals, 5 animals were killed by accidental injection of amygdalin into the intestine within a short period of time after the start of the experiment and therefore these animals were not included in the results.

On December 28, 1973, one of the amygdalin-treated animals developed a spontaneous mammary tumor or 79 days later than that of the first control tumor, followed by 10 more mice with mammary tumors and one abdominal tumor - on 2/14/74, 3/20/74, 3/22/74, etc., or 48 per cent of animals had spontaneous tumors. Twelve animals died or were sacrificed because of weakness from large tumors. Post mortem examination revealed 3 animals had lung metastases or 25 per cent. Thirteen animals are still alive with or without tumors.

The present study shows that for the three quarters of their life span (21 months) the daily prolonged intraperitoneal injections of a large amount of amygdalin did not prevent the development of mammary cancers in mice completely. However, it had a definite deduction in development of mammary tumors - 70% in controls against 48% in amygdalin-treated mice. It also shows amygdalin had a strong inhibitory effect on the development of lung metastases in mice - 75 per cent inhibiton against 22 per cent in controls. The general health and appearance of the amygdalin-treated animals were as good as that of the controls in spite of 16 months of injections. The body weights of control animals without tumors and that of amygdalin-treated animals without tumors all gained weight. The surviving animals are approximately 21 months old.

* Evaluation of lung metastases:
(+++) = more than 10 nodules in the lung.
(++) = more than 5 nodules in the lung.
(+) = less than 5 nodules in the lung.
(-) = no nodules in the lung.

† Killed by accidental injection of amygdalin into the intestine.

‡ Date for second tumor found.

Kanematsu Sugiura

Kanematsu Sugiura
September 30, 1974

Development of Mammary Tumors (Adenocarcinomas) in CL

Mouse No.	Tumor development: Date of tumor development	Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Survival time with tumor (days)	Lung: Gross exam.
1	5-30-74	0.6×0.8	2.0×2.9	448	86	+
2				510		
3	3-27-74	0.7×0.6	1.1×1.6	~~47~~ 343	47	++
	4-6-74†	1.1×1.0	2.3×2.0			
4	2-5-74	1.0×0.9	2.8×3.7	304	~~34~~ 31	+
5	5-7-74	0.8×0.9	3.1×2.4	401	37	+++
6	5-16-74	0.7×1.2	2.6×3.1	412	39	++
7	4-16-74	0.8×0.9	2.4×2.3	401	58	+
8	6-18-74	abdominal tumor 1.2×1.1	(malignant tumor) 1.7×2.5	468	62	+
9	10-11-73	0.5×0.5	1.9×1.6	224	68	+
	11-14-73†	0.7×0.9	3.0×2.5			
10	3-31-74	0.4×0.3	2.7×2.9	370	43	+
11	4-30-74	0.4×0.5	2.0×2.4	426	95	+
12	9-21-74	0.3×0.2	0.8×0.6	510	9	
13	8-13-74	0.6×0.6	1.1×1.3	510	48	
14	12-18-73	0.6×0.7	4.1×3.1	~~70~~ 284	70	+++
15				139		—
16	5-17-74	0.6×0.7	2.6×3.2	412	38	++
17	3-8-74	0.6×0.6	2.4×2.2	334	30	+
18				510		
19				445		—
20	12-20-73	0.8×0.7	3.6×2.6	272	15	++

Table I

...ry Tumors (Adenocarcinomas) in CD8F1 Mice—Controls (May 8, 1973– Sept. 30, 1974)

...size ...)	Final size of tumor (cm)	Duration of experiment (days)	Survival time with tumor (days)	Lung metastasis: Gross exam. *	Lung metastasis: Microscopic exam.	Termination	Δ No. of days
.8	2.0X2.9	448	86	+	+	Sac.	357
		510				Alive	–
.6 .0	1.1X1.6 2.3X2.0	~~47~~ 343	47	++	+	Died	323
.9	2.8X3.7	304	~~34~~ 31	+	+	Died	273
9	3.1X2.4	401	37	+++	+	Sac.	364
2	2.6X3.1	412	39	++	+	Sac.	373
.9	2.4X2.3	401	58	+	—	Sac.	343
[illegible] (malignant tumor) [illegible]	1.7X2.5	468	62	+	—	Sac.	406
.5	1.9X1.6	224	68	+	+	Died	156
.9	3.0X2.5						
.3	2.7X2.9	370	43	+	—	Sac.	327
.5	2.0X2.4	426	95	+	—	Died	357
.2	0.8X0.6	510	9			Alive	501
.6	1.1X1.3	510	48			Alive	462
.7	4.1X3.1	~~70~~ 284	70	+++	+	Sac.	214
		139		—	—	Died	
.7	2.6X3.2	412	38	++	+	Died	374
.6	2.4X2.2	334	30	+	—	Died	304
		510				Alive	
		445		—	—	Died	
0.7	3.6X2.6	272	15	++	+	Died	226

Table 1 (cont.)

Mouse No.	Tumor development: Date of tumor	Tumor development: No. of days	Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Survival time with tumor (days)
21					510	
22					510	
23					510	
24					510	
25	7-26-74	444 ~~0.3×0.2~~	0.3×0.2	0.5×0.6	510	
26	9-3-74 ~~0.3×0.2~~ 444-483		0.3×0.2	0.8×1.5	510	
	9-5-74† ~~0.6×0.8~~		0.6×0.8	1.1×1.0		
27	5-23-74	388 At conclusion	2.3×2.0	2.3×2.1	399	19
28					510	
29	7-19-74	437	0.3×0.7	0.9×1.1	478	41
30	9-3-74	483	0.6×1.1	1.8×2.4	510	27

Table 1 (cont.)

Age	Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Survival time with tumor (days)	Lung metastases: Gross exam. *	Lung metastases: Microscopic exam.	Terminated
			510				Alive
			510				Alive
			510				Alive
			510				Alive
44	0.3 X 0.2	0.5 X 0.6	510				Alive
44-483	0.3 X 0.2	0.8 X 1.5	510				Alive
0.8	0.6 X 0.8	1.1 X 1.0					
58	2.3 X 2.0	2.3 X 2.1	399	19	Reticulosarcoma or lymphoma	—	Sac.
			510				Alive
37	0.3 X 0.7	0.9 X 1.1	478	41	—	—	Died
83	0.6 X 1.1	1.8 X 2.4	510	27			Alive

Table 2

Effect of Amygdalin on the Development of Mammary Tumors and Lung Metastasis in CD8F1 mice (May 8, 1973 – September

Mouse No.	Tumor development		Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Survival time of tumor (found dead)
	Date of tumor	No. of days				
1					456	
2					30	
3	12-28-73	234	1.0 X 0.9	3.5 X 3.6	295	30
4					510	
5					189	
6	7-18-74 8-17-74†	436	0.8 X 1.1 1.0 X 1.1	1.3 X 1.6 2.6 X 2.3	468 ~~293~~	32
7					293	
8					510	
9	8-5-74	454	0.4 X 0.2	1.8 X 2.5	510	56
10					60	
11					51	
12	3-20-74 4-3-74†	316	0.9 X 1.0 0.6 X 0.6	2.8 X 3.2 0.8 X 1.1	356	40
13	6-3-74 6-10-74 †	391	0.9 X 1.4 0.7 X 0.5	2.4 X 3.2 1.3 X 1.6	423	32
14	3-22-74	318	0.6 X 0.7	2.3 X 2.0	378	61
15	8-30-74	479	0.9 X 0.7	1.9 X 2.2	510	31
16					510	
17	2-14-74	479	0.8 X 1.2	2.8 X 4.2	331	49
18	9-12-74	492	0.6 X 0.8	0.9 X 1.7	510	
19					510	
20					510	

Table 2

[illegible] on the Development of Mammary Tumor (Adenocarcinomas)

[illegible] in CD8F1 mice (May 8, 1973 – September 30, 1974)

[illegible]elopment No. of days	Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Survival time with tumor (days)	Lung metastasis * Gross exam.	Lung metastasis * Microscopic exam.	Terminated
-			456	found adenocarcinoma, ### no primary mammary tumor			Died
			30	in the lung, no primary [illegible] tumor			Killed +
234	1.0X0.9	3.5X3.6	295	30	—	—	Sac.
			510				Alive
			189		—	—	Killed +
436	0.8X1.1 1.0X1.1	1.3X1.6 2.6X2.3	468 ~~293~~	32	+	+	Sac.
			293		—	—	Died
			510				Alive
[illegible]54	0.4X0.2	1.8X2.5	510	56			Alive
			60		—	—	Killed +
			51		—	—	Killed +
[illegible]16	0.9X1.0 0.6X0.6	2.8X3.2 0.8X1.1	356	40	++	+	Sac.
91	0.9X1.4 0.7X0.5	2.4X3.2 1.3X1.6	423	32	+	—	Sac.
13	0.6X0.7	2.3X2.0	378	61	—	—	Died
74?	0.9X0.7	1.9X2.2	510	31			Alive
			510				Alive
79	0.8X1.2	2.8X4.2	331	49	—	—	Sac.
12	0.6X0.8	0.9X1.7	510				Alive
			510				Alive
			510				Alive

Table 2 (Cont.)

Mouse No.	Tumor development		Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Survival time with tumor (d
	Date of tumor	No. of days				
21					510	
22					510	
23					510	
24	7-18-74	436	0.4X0.6	1.7X 2.4	484	48
25					510	
26					510	
27	8-26-74	Additional tumor 475		2.$\sqrt{2}$X 2.8	507	32
28					50	
29	6-5-74	393	0.4X0.8	2.1X 2.4	435	42
30					10	

Table 2 (Cont.)

Experiment No. of days	Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Survival time with tumor (days)	Lung metastases: Gross exam. *	Lung metastases: Microscopic exam.	Termination
			510				Alive
			510				Alive
			510				Alive
436	0.4×0.6	1.7×2.4	484	48	—	—	Sac.
			510				Alive
			510				Alive
475		$2.\sqrt{2}$×2.8	507	32	—	—	Died
			50		—	—	Killed +
393	0.4×0.8	2.1×2.4	435	42	—	—	Sac.
			10		—	—	Killed +

Effect of Amygdalin on Spontaneous Mammary Tumors in Swiss Albino Mice.

This report consists of observations on the effects of prolonged treatment with amygdalin (Mexican) on the growth of spontaneous mammary tumors (adenocarcinomas) in female Swiss-Webster albino mice (Taconic Farms, New York). The diagnoses of tumor tissues were made from a post-mortem microscopic examination of tissues at the end of the experimental period. Occasionally small growths regressed completely under injections of saline or amygdalin. These undiagnosed growths were not included in the results. Spontaneous tumors other than mammary adenocarcinomas were not included in the results. The animals were kept on a normal diet (Purina Laboratory Chow) and water. Since we received only 2 to 5 tumor-bearing mice at each time from Taconic Farms the experimental group and control group were performed separately. The controls received 0.5 cc of saline (S) daily except mouse No. 1 which received 0.5cc of carboxymethyl cellulose (CMC) and the experimental animals received amygdalin daily intraperitoneally (except Sundays).

The results obtained from this study are summarized in Tables 1 and 2. The experimental results in Tables 1 and 2 are in the order of experiments performed. Twenty eight control mice with 35 tumors and 2 new tumors (2.5 x 2.9 cm., the largest to 0.6 x 0.6 cm., the smallest) and thirty five experimental mice with 37 tumors and 5 new tumors (2.4 x 1.9 cm., the largest to 0.7 x 0.7 cm., the smallest) were used.

Table 2 shows that repeated intraperitoneal injections of 1000 to 3000 mg/kg/day of amygdalin for 2 to 18 weeks failed to destroy the spontaneous breast cancers in mice. However, it caused to stop the continuous growth of small tumors (about 1.5 cm. diameter or less) more often than that of the control group - 8 out of 28 tumors in controls stopped growth or 29 per cent against 18 out of 35 tumors in amygdalin-treated animals stopped growth or 51 per cent.

It also shows that amygdalin had a strong inhibitory effect on the development of lung metastases in mice - 77 per cent inhibition against 7 per cent inhibition in controls. Undoubtedly mice with large tumors had lung metastases. It is possible that

these metastatic growths have been destroyed by the repeated treatment with amygdalin. The general health and appearance of the amygdalin-treated animals were much better than that of the controls.

Results obtained with mammary tumors occurring in Swiss albino mice are essentially the same as those obtained with mammary tumors occurring in CD_8F_1 mice - that repeated intraperitoneal injections of 2000 mg/kg/day of amygdalin inhibited the growth of small tumors and development of lung metastases in mice.

Kanematsu Sugiura

Kanematsu Sugiura
February 8, 1975

Footnotes for Tables 1 and 2

* Evaluation of lung metastases: (+++)=more than 10 nodules in the lung; (++)=more than 5 nodules; (+)=less than 5 nodules; (-)=no nodules.

† Amygdalin was dissolved in CMC, elsewhere it was dissolved in saline.

‡ New tumor found, days.

Δ Tumor growth stopped for indicated number of days, then growth resumed.

Table 1

Lewis Miller - Mammary Tumors (Adeno-carcinoma) - Cont'd

Animal No.	Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Tumor growth	Lung metastases: Gross exam.	Lung metastases: Microscopic exam.	Type
1	1.0 × 1.7 0.9 × 1.1	3.4 × 3.5 1.8 × 1.5	49	Both grew	+++	+	Duct
2	0.5 × 0.8	1.6 × 1.7	48	Injected 13 d Δ	—	—	Duct
3	1.3 × 1.3	1.9 × 2.8	48	Grew	+	+	Duct
4	1.4 × 1.3	2.8 × 2.3	71	Injected 49 d Δ	+++	+	Duct
5	1.8 × 1.8 1.6 × 0.8	5.1 × 4.4 1.3 × 1.0	51	Both grew	+	+	Duct
6	0.9 × 0.9 * 0.6 × 0.5 9 d	1.7 × 2.2 1.0 × 1.3 9 d *	45	Injected 14 d Δ	+++	+	Duct
7	0.9 × 0.9	5.8 × 4.0	45	Grew	+	—	Duct
8	1.3 × 1.1	1.2 × 1.1	84	Injected 84 d Δ	+++	+	Duct
9	0.6 × 0.6 0.8 × 0.9 24 d *	1.7 × 1.5 1.4 × 1.7	114	Both grew	—	—	Duct
10	1.8 × 1.8	2.7 × 2.7	47	Grew	+++	+	Duct
11	1.2 × 1.6 1.0 × 1.3	2.1 × 2.3 3.8 × 4.9	51	Both grew	+	—	Duct
12	2.0 × 1.5	5.4 × 4.0	40	Grew	+++	+	Duct
13	2.2 × 1.4	3.4 × 3.3	51	Grew	+++	+	Duct
14	1.4 × 1.3 1.7 × 1.6	2.7 × 2.1 2.6 × 2.1	51	Grew	+	—	Duct
15	1.8 × 1.9	3.6 × 3.0	70	Grew	+	—	Duct
16	1.8 × 1.8	3.6 × 2.4	37	Grew	+	+	Duct
17	1.8 × 1.8 1.2 × 1.5	2.5 × 3.1 2.8 × 2.1	37	Grew	+++	+	Duct

Table 1 (cont.)

Controls

Mouse No.	Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Tumor growth	Living metastases: Gross exam.	Living metastases: Microscopic exam.	Terminated
18	0.9×0.9	5.9×4.1	45	Grew	+	—	Sac.
19	0.9×1.3	~~0.6×1.1~~ 2.4×2.8	~~41~~ 76	~~Stopped 41.6~~ Grew	++	+	Sac.
20	1.0×1.4	2.4×2.9	76	Grew	+	+	Sac.
21	0.9×1.3	0.6×0.9	93	Stopped 93 Δ	+	—	Sac.
22	1.1×1.4	~~2.9×2.1~~ 3.8×4.2	~~93~~ 45	Grew	++	+	Sac.
23	1.1×1.7	4.1×4.4	49	Grew	+	—	Died
24	0.9×0.7	2.3×2.1	93	Grew	+	—	Sac.
25	1.5×1.7	0.5×0.5	93	Stopped 93.2 Δ	+	+	Sac.
26	2.1×1.9	3.0×4.0	42	Stopped 14 Δ	++	+	Sac.
27	2.5×2.6 1.7×1.5	3.5×3.7 3.1×2.5	19	Both grew	++	+	Died
28	2.5×2.9 1.6×1.9	3.5×2.6 2.8×2.0	14	Both grew	+++	+	Died

Table 2

Acute Albino Hamsters by Topical (Adrenocortical) Treated with

Hamster No.	Dose mg/kg/day	Initial Size of Tumor (cm)	Final Size of Tumor (cm)	Duration of Experiment (days)	Tumor Growth	Lung Metastases	
						Gross Exam. *	Microscopic Exam.
1	2000 †	1.6 × 1.3	4.1 × 2.9	40	Grew	—	—
2	1000 †	1.5 × 1.6	2.3 × 2.1	19	Grew	—	—
3	1000 †	1.5 × 1.5	1.3 × 1.7	127	Stopped 127 d Δ	—	—
4	2000 †	1.5 × 1.3	3.3 × 3.6	33	Grew	+	+
5	2000 †	1.9 × 1.4 1.0 × 0.5 22 d ‡	3.2 × 2.3 1.1 × 1.7	35	Both grew	+++	+
6	2000	0.9 × 0.7	1.0 × 0.8	71	Stopped 64 d Δ	—	—
7	2000	1.1 × 0.7	0.7 × 0.7	71	Stopped 71 d Δ	—	—
8	2000	1.6 × 2.1	2.6 × 2.4	20	Grew	—	—
9	2000	1.1 × 0.9	1.8 × 2.4	28	Grew	+	+
10	1000	2.0 × 1.7	1.9 × 1.5	19	Stopped 19 d Δ	—	—
11	3000 †	1.3 × 1.3	2.0 × 2.4	55	Stopped 34 d Δ	—	—
12	3000 †	1.9 × 1.8 0.7 × 1.1	3.0 × 3.1 0.8 × 1.1	29	Both grew	—	—
13	3000 †	1.1 × 0.9	1.3 × 1.0	81	Stopped 69 d Δ	—	—
14	3000	0.8 × 1.2	0.6 × 1.0	11	Stopped 11 d Δ	—	—
15	2000	1.0 × 1.3	0.9 × 0.9	51	Stopped 51 d Δ	—	—
16	2000	1.0 × 1.0	2.0 × 2.3	51	Grew	—	—
17	2000	1.4 × 1.6	3.2 × 3.7	51	Grew	—	+
18	2000	2.0 × 2.0	2.4 × 2.1	23	Grew	—	—
19	2000	1.8 × 1.9 1.9 × 1.4	2.6 × 2.4 4.0 × 3.0	23	Both grew	—	+
20	2000	0.7 × 1.0	0.9 × 1.2	23	Grew	—	—
21	2000	1.1 × 1.5	1.2 × 2.0	12	Grew	—	—

Table 2

Pulmonary Tumors (Adenocarcinoma) Treated with Cryosurgery

Age	Initial size of Tumor (cm)	Final size of Tumor (cm)	Duration of experiment (days)	Tumor growth	Lung metastasis: Gross exam.	Lung metastasis: Microscopic exam.	Tumor
7	1.6×1.3	4.1×2.9	4.0	Grew	—	—	Died
+	1.5×1.6	2.3×2.1	14	Grew	—	—	Died
+	1.5×1.5	1.3×1.7	127	Stayed 127 d	—	—	Sac.
+	1.5×1.3	3.3×3.6	33	Grew	+	+	Died
+	1.4×1.4 ‡ 1.0×0.5 22 d	3.2×2.3 1.1×1.7	35	Both grew	++	+	Died
	0.9×0.7	1.0×0.8	71	Stayed 64 d	—	—	Sac.
	1.1×0.7	0.7×0.7	71	Stayed 71 d	—	—	Sac.
	1.6×2.1	2.6×2.4	20	Grew	—	—	Died
0	1.1×0.9	1.8×2.4	28	Grew	+	+	Died
	2.0×1.7	1.4×1.5	14	Stayed 14 d	—	—	Died
+	1.3×1.3	2.0×2.4	55	Stayed 37 d	—	—	Died
+	1.4×1.3 0.7×1.1	3.0×3.1 0.8×1.1	29	Both grew	—	—	Died
+	1.1×0.9	1.3×1.0	81	Stayed 69 d	—	—	Sac.
	0.8×1.2	0.6×1.0	11	Stayed 11 d	—	—	Died
	1.0×1.3	0.9×0.9	51	Stayed 51 d	—	—	Sac.
	1.0×1.0	2.0×2.3	51	Grew	—	—	Died
	1.4×1.6	3.2×3.7	51	Grew	—	+	Sac.
	2.0×2.0	2.4×2.1	23	Grew	—	—	Sac.
	1.8×1.9 1.9×1.4	2.6×2.4 4.0×3.0	23	Both grew	—	+	Sac.
0	0.7×1.0	0.9×1.2	23	Grew	—	—	Sac.
	1.1×1.5	1.2×2.0	12	Grew	—	—	Sac.

Amygdaline – Treated.

Mouse No.	Dose mg/kg/day	Initial size of tumor (cm)	Final size of tumor (cm)	Duration of experiment (days)	Tumor growth	Lung metastases: Gross exam.	Micro exam.
22	2000	0.4 X 1.4	0.2 X 0.5	41	stopped 41st	—	—
23	2000	1.1 X 1.3 1.5 X 1.6 15th	3.6 X 2.7 1.8 X 1.9	30	Both grew	—	—
24	2000	0.8 X 1.1	0.8 X 0.9	115	stopped 115th	—	—
25	2000	1.1 X 1.4	1.0 X 1.6	112	stopped 91st	—	—
26	2000	1.1 X 1.7	3.4 X 3.5	51	stopped 14th	—	—
27	2000	1.4 X 1.4	1.5 X 2.5	55	stopped 34th	—	—
28	2000	0.9 X 0.9	2.1 X 3.1	38	grew	+	+
29	2000	1.3 X 1.6	1.4 X 2.3	78	stopped 42nd	—	+
30	2000	1.4 X 1.5	2.9 X 2.7	64	stopped 21st	—	—
31	2000	0.7 X 0.7	0.8 X 0.8	78	stopped 78th	—	—
32	2000	1.3 X 1.5 0.8 X 0.6 48th	1.2 X 2.0	78	stopped 49th	—	—
33	2000	0.6 X 0.8 0.7 X 0.7 48th	1.6 X 1.7 1.1 X 0.9	78	stopped 35th	—	—
34	2000	2.4 X 1.9	4.9 X 3.5	30	grew	+	+
35	2000	1.3 X 1.7 0.5 X 0.5 14th	4.1 X 3.2 0.6 X 0.5	24	Both grew	+++	+

Table 2 (Cont.)

Amygdaline-Treated.

Weight (gm.)	Final size of tumor (cm.)	Duration of experiment (days)	Tumor growth	Lung metastasis		Termination
				Gross exam.	Microscopic exam.	
1.4	0.2 X 0.5	41	Stopped 41 d	—	—	Sac.
1.3	3.6 X 2.7	30	Both grew	—	—	Died.
1.6 15d	1.8 X 1.9					
1.1	0.8 X 0.9	115.	Stopped 115 d	—	—	Sac.
.7	1.0 X 1.6	112	Stopped 91 d	—	—	Sac.
1.7	3.4 X 3.5	51	Stopped 14 d	—	—	Sac.
1.7	1.5 X 2.5	55	Stopped 34 d	—	—	Sac.
0.7	3.1 X 3.1	38	Grew	+	+	Sac.
1.0	1.4 X 2.3	78	Stopped 42 d	—	+	Sac.
1.5	2.9 X 2.7	64	Stopped 21 d	—	—	Died
.7	0.8 X 0.8	78	Stopped 78 d	—	—	Sac.
.5 + 15d	1.2 X 2.0	78	Stopped 49 d	—	—	Sac.
+ 15d	1.6 X 1.7 1.1 X 0.9	78	Stopped 35 d	—	—	Sac.
1	4.9 X 3.5	30	Grew	+	+	Sac.
+ 14d	4.1 X 3.2 0.6 X 0.5	24	Both grew	++	+	Sac.

A Summary of the Effect of Amygdalin Upon Spontaneous Mammary Tumors in Mice

Kanematsu Sugiura: September 12, 1972 - June 13, 1973

Dr. Sugiura has performed three sets of major experiments to determine the effects of amygdalin (i.p.) in carboxy methyl cellulose (CMC) upon mice with spontaneous mammary tumors (adenocarcinomas). The mice strain was CD_8F_1. The results of these experiments have been combined and are shown in the Table below, along with pertinent procedural data:

TABLE I

Results of Amygdalin (i.p.) Treatment after Six Weeks

Tumors (varied from 2.8 x 2.1 cm - 0.9 x 0.6 cm)	Controls-CMC alone (28 mice, 28 tumors at start; 23 mice at end)	Amygdalin (1-2g/kg/day in CMC-30 mic 36 tumors at start;23 mice
Growing	27	28
Stopped Growing	1 (3.5%)	8 (22.2%)
Regressing	0	0
Regressed	0	0
New Tumors	11 (37%)	8 (22.2%)
Lung metastases present	18 (78.2%)	4 (17.4%)
Lung metastases absent	5 (21.8%)	19 (82.6%)

The results clearly show that amygdalin significantly inhibits the appearance of lung metastases in mice bearing spontaneous mammary tumors and increases significantly the inhibition of the growth of the primary tumors over the appearance of inhibition in the untreated animals. Laetrile also seemed to prevent slightly the appearance of new tumors but the significance level of this data is questionable.

The three experiments from which this data is pooled differed from each other in certain important ways. In one case the animals were younger and therefore exhibited smaller tumors. These animals were as well given 1g/kg/day. The results of this experiment (roughly contributing one-third of the data) indicated that smaller tumors were more readily inhibited (50%) by amygdalin but that lung metastases were present in greater than average frequency (30%) probably due to the lower dose. The other two experiments employed 2g/kg/day, older animals whose tumors were larger, and which displayed far fewer lung metastases (7%). The rate of appearance of new tumors in amygdalin-treated animals remained constant in the three experiments but varied in the control group. Young control mice show a far greater incidence of new tumors (77%) than old mice (21%).

The mice used in this study were the F1 of a cross between BALB/c (M+V) and DBA/8 mice. Eighty per cent of these mice produce spontaneous mammary tumors by the time they reach ten months of age. In spite of the fact that these mice are so prone to tumor development, amygdalin showed some interference

with the typical tumorgenic process of this strain. The agent has a decided effect against the formation of lung metastases and upon the appearance of new tumors. In some cases, inhibtion of established tumor growth was observed.

Dr. Daniel Martin, Department of Surgery Research at the Brooklyn-Queen Catholic Medical Center, has been employing this strain in examining the efficacy of various chemotherapeutic and immunotherapeutic protocols upon the post-surgical recurrence of malignancy. As Dr. Martin has already demonstrated, this strain lends itself perfectly to such an experiment and affords a close and valuable emulation of the clinical situation in human mammary cancer. As a possible extention of this sort of work, amygdalin might be used in this way to determine its effect upon recurrent disease.

Some preliminary data about Swiss Webster mice is shown in Table II. A total of five mice were used. As seen, three of these mice which had small mammary tumors and were treated as usual with amygdalin showed tumor regression and in two of these, tumors could no longer be detected. In mice with larger tumors, regression may be less easy to obtain but <u>inhibition</u> of tumor growth seems so far to be the rule in this strain. Dr. Sugiura has never observed complete regression of these tumors in all his cosmic experience with other chemotherapeutic agents. Also as seen in Table II, addition of B-glucosidase does not afford low doses of amygdalin any anti tumor effects. The results clearly state

that amygdalin must be further studied. The improvement of health and appearance of the treated animals in comparison to controls is always a common observation.

Dr. Sugiura is presently attempting to see if amygdalin will prevent the initial appearance of mammary adenocarcinoma in young CD_8F_1 mice.

Summary

Amygdalin in i.p. doses of 1000-2000 mg/kg/day causes significant inhibition of spontaneous mammary tumors in the highly inbred CD_8F_1 mice is significant inhibition of the formation of lung metastases and possibly prevents, to an uncertain degree, the formation of new tumors, regardless of the age of the mice. Greater inhibition of tumor growth was seen in smaller spontaneous tumors of this strain.

In Swiss Webster albino females with both large and small spontaneous mammary tumors, amygdalin caused regression in 4/5 animals studied and complete regression in 2/5. The complete regressions occurred only in small tumors on non-inbred mice.

All treated animals maintained better health and appearance than the controls.

Summary - Continued

Dr. Sugiura is presently involved in determining whether amygdalin will prevent the occurrence of spontaneous tumors in 60 CD_8F_1 mice. The results will be reported when available.

TABLE II

Animal(s)	Tumor	Dose of Amygdalin	+B-glucosidase	Inhibition	Regression	Lung Metastases
5 CD_8F_1	Spontaneous Mammary	50	yes	none	none	——
1 Swiss Webster	Spontaneous Mammary (1.6 x 1.3 cm)	37.5 (40 days)	yes	none	none	none
1 AKR	Spontaneous Osteogenic Sarcoma (1.6 x 1.7 cm)	2000 (10 days)	no	yes	slightly	——
2 Swiss Webster	Large Spontaneous Mammary (1.5 x 1.5 cm)	1000 (21 days)	no	1/2	1/2	none
3 Swiss Webster	Small Spontaneous Mammary (0.7 x 0.7 cm)	1000 (20 days)	no	3/3	2/3 (tumors undetectable)	——

Addenda (to Sugiura report) June 13, 1973

Table III is an updated extension of the data of Table II. The additional information pertains to experiments, some yet in progress, in which five Swiss mice were (are) being injected with 2000 mg/kg/day i.p. over extended periods. The data further points to the fact that tumors larger than about 1.0 x 1.0 cm are less likely to be inhibited by amygdalin ones about this size or smaller.

Dr. Sugiura reiterated that these animals are difficult or impossible to cure in all of his experience. This is why the two animals which have showed complete regression are so significant. So far, these two mice remain tumor free, in spite of discontinuance of treatment.

Unfortunately, no data are available about the comparative members of actual lung metastases because their size and the size of the average mouse lung makes this difficult. Apparently, gross examination reveals that the number and size of lung metastases/animal, in those animals which displayed them, were no different between the two groups.

No gross difference in the primary tumors could be observed between the treated and control groups in the CD_8F_1 experiments. Histology was not performed because no pathologist was available and at any rate, it was felt by Dr. Sugiura that the size of the tumors in some cases would have made making their sections precarious.

As yet, no tumors have appeared in the control or laetrile-treated batch of 60 CD_8F_1 female mice born in December, 1972. Spontaneous Tumors are expected to appear in these animals this month.

This report consists of observations on the effects of prolonged treatment with laetrile (SK1691) on the growth of spontaneous mammary tumors (adenocarcinomas) in Swiss albino mice. These animals received 1000 or 2000 mg/kg/day of laetrile daily intraperitoneally.

The results obtained are summarized in Table 3. It shows that repeated intraperitoneal injections of laetrile had no effect on large tumors (more than 1.5 cm diameter). However, it caused a complete regression of small tumors (less than 1.0 cm diameter). One of the six treated animals had lung metastases.

K. Sugiura
June 13, 1973

Table

Effect of Amygdalin on Spontaneous Mammary Tumors in Swiss

	Date Tested	Original tumor size (cm).	Dose mg/kg/day	Duration of injections (days)	Duration of Experiment days)
Control	4/3/73	0.9 x 1.6	CMC	35	49*
	4/3/73	0.9 x 1.1			
Control	6/19/73	0.8 x 0.8	Saline	49	49**
Control	6/19/73	1.3 x 1.3	Saline	49	49**
Control	6/19/73	1.6 x 1.4	Saline	49	49**,‡
	1/16/73	1.6 x 1.3	37.5 + 50 mg/kg β-glucosidase	36	40*
	2/19/73	1.6 x 1.6	2000	10	10*
	3/7/73	1.5 x 1.6	1000	18	19*
	3/7/73	1.5 x 1.5	1000	56	127**,‡
	3/13/73	0.7 x 0.6	1000	12	13*
	3/13/73	0.7 x 0.7	1000	34	35*,‡
	3/13/73	0.7 x 0.4	1000	56	122**
	4/18/73	1.5 x 1.3	2000	32	33*
	4/18/73	1.9 x 1.5	2000	34	42*
	5/31/73	0.8 x 0.7	2000	43	70**,‡
	5/31/73	1.0 x 0.7	2000	43	70**,‡
	5/31/73	1.6 x 2.1	2000	20	21*

* Death of animals
** Sacrificed
‡ Growth of tumor stopped for entire cour:
□ Absence of tumor at autopsy
△ Absence of tumor at 14th day

Albino Mice (Taconic Farms)

Tumors						
Growing	Stopped Growing	Regressing	Regressed	New Tumors	Lung Metastases	
X				0	+	*1
X						
X				0	-	
X				0	-	
	X			0	-	
X				0	-	
		X		0	-	*2
X				0	-	
	X			0	-	
			X□	0	-	
	X			0	-	*3
			X△	0	-	
X				0	-	
X				1	+	
	X			0	-	
	X			0	-	
X				0	-	

REMARKS

***1 Also metastases in pleural cavity**

***2 Fibrosarcoma, not a mammary tumor. Also had a nodule at mediastinum.**

***3 Tumor contained only pus.**

SLOAN-KETTERING/MEXICO

CORRESPONDENCE

MEMORIAL SLOAN-KETTERING CANCER CENTER
1275 YORK AVENUE, NEW YORK, NEW YORK 10021
(212) 879-3000

Office of President and Director
Sloan-Kettering Institute

January 24, 1975

Dr. Mario Soto de Leon
Centro Hospitalario "20 de Noviembre"
Av. Coyoacan y Felix Cuevas
Col. del Valle
Mexico 12, D.F.

Dear Dr. Soto:

It was indeed a pleasure to have you and Dr. Sanen visit our Institute and share with us your clinical experience with Amygdalin in cancer patients. I was pleased to hear from Dr. Sanen that our proposed collaborative controlled trials have the approval of your hospital. We are looking forward to a fruitful exchange of information.

My best wishes,

Sincerely yours,

Lloyd J. Old

Lloyd J. Old, M.D.
Vice-President and
Associate Director

ANATOMY OF A COVERUP

ANATOMY OF A COVERUP

The controversy over the substance Laetrile as a cancer-fighter had been raging since at least 1950 before the advent of a turning point in 1972.

Until that time, Laetrile (amygdalin, Vitamin B17) had seemed doomed: rubbing up against the powerful pharmaceutical-medical-governmental establishment, it had been found wanting and classed as quackery, despite the fact that Laetrile already had thousands of testimonials to its benefit, had reached full legal status in 23 other countries and was the subject of solid scientific research.

The controversy had simmered on-again, off-again until July, 1972, when the Committee for Freedom of Choice was formed following the arrest of a California doctor on "cancer quackery" statute violations involving the use of Laetrile in cancer treatment.

The formation of the Committee for Freedom of Choice caused the turnaround in the Laetrile controversy:

First, hundreds and then thousands of irate citizens grouped themselves under the Committee for Freedom of Choice in Cancer Therapy in defense of doctors and against the legislation which, they believed, denied them both freedom of choice in therapy and also intruded into

the privacy of the doctor-patient relationship.

Secondly, the plight of other embattled medics wishing to be true to their Hippocratic oaths, and the advent of a strong grassroots backlash against the Gestapo-like powers of the "establishment" in medical matters, brought the entire controversy over Laetrile back to the surface again.

The dam began to break: by 1973, scores of U.S. doctors were admitting either interest in or use of Laetrile. Doctors were winning their court cases. Thousands of new testimonials to the efficacy of Laetrile were being logged. An originally hostile press was beginning to take renewed interest in "the apricot-pit cancer cure." Pro-Laetrile and pro-natural health organizations were flourishing.

A grassroots movement, the Test Laetrile Now Committee, was underway gathering signatures to then President and Mrs. Richard Nixon urging the full scale testing of the substance on humans, despite the fact that, extraofficially, it had been "tested" thousands of times in the U.S.A.

At the same time, it was announced that the Memorial Sloan-Kettering Cancer Center in New York, perhaps the most prestigious cancer research facility in the world, had undertaken the scientific testing of the compound, reportedly at the behest of Benro Schmidt, a New York investment banker tabbed by Nixon to head the President's Cancer Panel—the board of directors, so to speak, of the "War on Cancer."

Schmidt was asked later what had prompted him to approach Sloan-Kettering for the test program. His response:

"I have had more mail since I've been chairman on the subject of Laetrile than on any other single subject—virtually equal to all the mail on all subjects put together. There is a very considerable traffic in Laetrile....My only interest in Laetrile is that we find out for an absolute certainty what it does or does not do."

The first view of what was going on at Sloan-Kettering in tests of amygdalin on selected strains of mice came out at a Committee for Freedom of Choice press conference: the report "leaked" from the New York institution on a series of tests conducted by veteran scientist, Kanematsu Sugiura, indicating initially positive results.

The report spoke of results gleaned over a 10-month period during which doses of the substance caused "significant inhibition of spontaneous tumors" as well as "significant inhibition of the formation of lung metastases," and it was noted that Laetrile "possibly prevents, to an uncertain degree, the formation of new tumors."

Sloan-Kettering was justifiably irked that a "leaked" report had gotten out. Within months, the institute, in the first of a series of statements on the Laetrile affair, announced that a second series of tests had been unable to confirm Dr. Sugiura's original tests, but that research was continuing.

A battle of statements and press releases then ensued. Laetrile champions were certain that history cannot be rewritten and that the early tests could not simply be brushed aside. The statements of officialdom—The Food and Drug Administration, the National Cancer

Institute, the American Medical Association and the American Cancer Society—continued to be to the effect that Laetrile simply had never been demonstrated as an effective anti-cancer agent despite considerable testing.

In the wings, however, lurked Dr. Dean Burk, one of the founders of NCI and, until his retirement in 1974, head of that organization's cytochemistry division, a well credentialed savant eminently qualified to discuss his subject matter. Burk's routine assessments of NCI-sponsored and otherwise officially sanctioned tests on Laetrile were simply that the government was lying.

"Once any of the FDA-NCI-AMA-ACS hierarchy so much as concedes that Laetrile antitumor efficacy was indeed even once observed in NCI experimentation, a permanent crack in the bureaucratic armor has taken place that can widen indefinitely by further appropriate experimentation," he said, while accusing medical orthodoxy and officialdom of "obfuscations, red herrings, misrepresentations and outright lies."

Dr. Burk, who had run tests on the substance himself, consistently and convincingly argued that Laetrile test statistics on animals revealed the very reverse of what the "experts" claimed they revealed and hence made a case for Laetrile testing on humans.

Part and parcel of the problem was the well-intentioned amendment to the Food, Drug and Cosmetic Act in 1962 whereby any substance to be "cleared" for use on humans must be demonstrated both safe and effective before it may be licensed. This enormous new legal loophole allows

"wanted" drugs to be approved but keeps unwanted ones out. It also vastly increases the amount of red tape needed to license a new medication. This can be seen in the case of, say, Parke-Davis alone. In 1948 this well-known pharmaceutical firm had to submit 73 pages of evidence to secure the licensing of a drug. By 1968 the same company had to submit 72,200 pages of data, transported by truck, in an effort simply to have an anestetic licensed.[1]

In the meantime, Laetrile had been presented with a classic Catch-22 situation:

American medical authorities confessed skepticism of foreign work with the substance and expressed the desire for American doctors who had information on good results with Laetrile to step forward with their evidence. However, since 1963 Laetrile had been indirectly banned by provisions of the Food, Drug and Cosmetic Act from interstate shipment and sale and, in California, specifically banned by state law. Hence, doctors stepping forward with information were quite openly risking themselves legally and, when not legally, professionally by state boards of medical examiners which held Laetrile to be quackery.

On top of that, since the vast majority of patients in the U.S. who turned to Laetrile only did so after orthodox therapy—cutting, burning and poisoning—had given up on them, the results from Laetrile use here or anywhere else were open to question. If the results were

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[1]Walter S. Ross, "The Medicines We Need—But Can't Have," Reader's Digest, October, 1973.

good, "spontaneous remission" or "response to earlier, orthodox treatment" or, at least, a "sugar-pill effect," could be argued. If they were bad, then they could be written off as "another failure for Laetrile."

And also in the meantime, terminal cancer victims on whom "the best treatments available" had given up were left with the prospect of either dying or desperately clutching at a straw of hope and, if they had the money, literally fleeing to Mexico, West Germany or some other place where access to the simple extract of apricot kernels was available. To make the latter decision has of course meant that many cancer sufferers have been treated like common criminals for being provided abroad with a substance not "cleared" by FDA red tape.

This was the background to the Sloan-Kettering selected mouse tests whose first "leaked" report opened this new phase of the Laetrile War in 1973.

By the end of 1974, newspaperman and writer, Mike Culbert, learned that a third series of animal studies had indeed confirmed the first series and that the failure of the second series at S-K was apparently due to a difference in material (between Mexican and West German production). He had just authored Vitamin B17: Forbidden Weapon Against Cancer and wanted the facts straight: that a third series of tests had confirmed the first, that tests of the substance on humans, probably for analgesic effects, were right around the corner. This was confirmed to him in October, 1974, at Sloan-Kettering.

For months, Laetrile boosters waited for official word from Sloan-Kettering. Intermittent contacts with S-K brought only the standard responses that tests were continuing. The responses from FDA, AMA and ACS continued to be that Laetrile was worthless—"not a shred of efficacy," as the FDA commissioner put it.

A Sloan-Kettering vice president told Canadian national television in January 1975 that "we have seen results that seem to be significant" in the Laetrile tests. Another Sloan-Kettering officer was quoted as saying that there was no indication of efficacy, and then slightly amended the original quote. Confusion at the level of the press release seemed to be the order of the day. All the time, a thousand Americans per day continued to drop deal of cancer, whose national fatality statistics had reached record high level in the nation whose best orthodox science could hold out little more than a less-than-gambler's chance for a 7.5% 5-year survival chance in the case of most metastasized cancer through the painful, disfiguring and expensive cut-burn-and-poison approach.

In July, 1975, Sloan-Kettering, through in-depth articles in The New York Times, made the global and apparently final announcement:

"Four cancer research centers working under Federal grants have been unable to confirm assertions that the contraband drug Laetrile can cure cancer or inhibit malignant growths, according to previously undisclosed findings of animal studies."

Moreover, S-K personnel were quoted to the effect that the idea

even of testing Laetrile for analgesic benefits on humans had been discarded.

This was deeply interesting, for the Committee for Freedom of Choice in Cancer Therapy already had copies of correspondence between Sloan-Kettering and 20 de Noviembre Hospital in Mexico City in which plans for actual human (clinical) studies with Laetrile were being planned! (See appendix.)

An NCI official was quoted as saying that "the push behind Laetrile...is financial and political. If we did a clinical (human) trial, it would legitimize the drug and its use would increase a hundredfold."

This was the situation, then, as of August, 1975: S-K's claim that its earlier Laetrile tests had not been confirmed by outside studies (the first tests were referred to as "spurious" and "curious" in Times coverage). There was a hint that many different studies had been conducted—as indeed they had.

Then another "leak" occurred:

Mike Culbert was sent, in August, a copy of six series of Laetrile mouse tests conducted by the veteran Dr. Sugiura. A cover letter to him on Sloan-Kettering stationery, but anonymous, claimed the results mentioned within were being suppressed. A check with Dr. Sugiura confirmed that the tests were indeed legitimate but that he had not sent the letter (see appendices). The tests cover research from March 1, 1974, to February 8, 1975.

The tests are significant for several reasons:

First, they put the lie to the statements by officialdom that Laetrile tests have never uncovered a "shred of efficacy" in cancer treatment.

Second, they indicate that at least seven series of mouse tests with amygdalin (Laetrile, Vitamin B17) have indicated a "shred of efficacy."

Third, they strongly suggest that somebody somewhere is terribly interested in not publishing all the facts about Laetrile and animal studies. It is not the purpose of this preface to speculate about who or why, or even to point fingers at the famed Sloan-Kettering Institute itself. But the lay public has clearly not been told the whole truth about Laetrile.

We must bear in mind, rationally, that what Laetrile does or does not do in animals is by no means conclusive as to what it does or does not do in humans. The animals involved are specially bred and the tumor systems are massive in nature.

To be concisely, precisely "clean" in the matter of semantics, the Committee would agree that if the only indication for the validity of a cancer drug is the measurement of a tumor, following the drug's administration, then—again, in a very strict semantical sense—Laetrile can be said to have at least partially failed in the referred-to tests.

But that is by no means the whole—or even the real—story. As Dr. Ernst T. Krebs, Jr. explains in the accompanying study of what the mouse tests show:

• Laetrile attacks only cancer tissue. It is "poisonous" only to cancer, unlike the "legal" and "orthodox" chemotherapeutic and/or radiation agents which are toxic to the entire metabolism.

• The bigger the tumor, the less the percentage of actual cancer cells per se there are. Laetrile's action, theoretically, is limited only to malignant cells. The "index of tumefaction"—measurement of a lump or bump, in layman's terms—may very well be measuring the effect of the total poisoning of the tumor, cancer and normal cells alike. Hence, in a person treated with "orthodox" modalities, a decrease in a lump or bump may be noted (as it may be noted in Laetrile administration, too), but that index says little about cancer as a systemic or metabolic disease.

• What the reports show, in (now) all seven sets (of the tests which have been "leaked" to the Committee) is that amygdalin administration OBVIOUSLY BLOCKED THE SPREAD OF MALIGNANCY!

The case made by Krebs and a growing phalanx of Laetrile researchers around the world is this:

Amygdalin (Vitamin B17, Laetrile) prevents cancer, first and foremost. In the event there is clinical cancer, it is the best available tool for fighting cancer because it helps block existing cancer and often effectively stops the spread (metastasis) of the disease. No claims of "cure"—but rather of "control" are made for the substance. No claims that Laetrile can restore damaged tissue are made. No "miracle" is offered. Even so, the number of total recoveries with Laetrile-based therapy is increasing—and Laetrile's capacity as an

analgesic is being accepted by hundreds of doctors.

No credible case can be made for stating that there is no efficacy indicated by Laetrile in mouse tests.

Much more importantly, however, we believe there is no credible reason for not going ahead with officially sanctioned amygdalin trials on humans.

The enclosed reports—the seven studies by Dr. Sugiura and the Krebs commentary on them—eloquently make the case for the legal vindication of Laetrile, if in fact it needs any.

Cancer is the number two natural killer in the United States, snuffing out more than 365,000 lives per year. The history of the "war on cancer" shows we are losing that war. In the meantime, a substance which offers efficacy, both in prevention and treatment, has been getting the bureaucratic runaround.

For God's sake, if there is genuine interest in winning the cancer war, let's get on with it.

The issue really is "freedom of choice". WHY are Americans being denied access to an admittedly non-toxic substance?

And most importantly—is it really necessary to wait for human studies? If human tests take as long as animal studies did—and experience indicates they take more time—then we may face the prospect of 5 more years of tests, during which time two million Americans will have died.

No, there is only one rational moral procedure—Freedom of Choice—Amygdalin (Laetrile) should be legal and available NOW!!

COMMITTEE

PRESS RELEASE

THE COMMITTEE FOR FREEDOM OF CHOICE IN CANCER THERAPY, INC.
146 MAIN STREET • SUITE 408 • LOS ALTOS, CALIFORNIA 94022 • (415) 948-9475

FOR IMMEDIATE RELEASE

September 5, 1975

LOS ALTOS, CALIFORNIA—Apparently suppressed reports of tests of the controversial substance Laetrile on mouse cancer reveal Laetrile's effectiveness—despite statements to the contrary by the research institution in which the tests were carried out.

This was the claim made today by the Committee for Freedom of Choice in Cancer Therapy, Inc., as it released hitherto unpublished reports from the Memorial Sloan-Kettering Cancer Center in New York.

Robert W. Bradford, committee president, said that the six mouse cancer tests conducted by Dr. Kanematsu Sugiura at Sloan-Kettering from 1973 to 1975 had been "leaked" to the organization by "persons unknown" at the center.

But Dr. Sugiura confirmed that the work—which reveals that Laetrile effectively blocked the spread of cancer in specially bred mice without destroying "primary" tumors themselves—is his.

A note on Sloan-Kettering letterhead stationery sent last month with copies of the Sugiura research to Mike Culbert, a former California newspaper editor and now editor of Committee for Freedom of Choice publications, stated that "due to political pressure these (mouse test) results are being suppressed."

Dr. Sugiura said he had not written the note.

In August, spokesmen at Sloan-Kettering announced that repeated tests, including independent outside efforts, had failed to confirm an earlier 1973 Sugiura test which had indicated the same pattern: Laetrile's effectiveness at halting the spread of cancer in specially bred mice.

Laetrile, an extract of the chemical amygdalin from apricot kernels and whose natural form occurs in over 1,200 plants, has been indirectly banned from interstate shipment by the Food and Drug Administration for over a decade and specifically banned from use in cancer treatment by California law.

The center of a long-standing controversy, the substance is legal in 23 other countries, the nearest being Mexico, which thousands of American cancer patients visit annually seeking Laetrile treatment. Despite many thousands of testimonial claims made for the substance's efficacy, American medical orthodoxy has long claimed that there is no objective evidence of Laetrile's efficacy either in treating or preventing cancer.

Along with the allegedly suppressed six Sloan-Kettering studies, Bradford also released a detailed commentary on the same by San Francisco biochemist, Ernst T. Krebs, Jr., the scientist who developed and named Laetrile and who has fought for its vindication as a cancer-fighter since 1949.

Dr. Krebs, who also discovered and anmed Vitamin B15, noted:

"Those who recognize as overwhelmingly important and decisive the criterion of the total inhibition of metastases from a primary tumefaction

see in Sugiura's findings a 70 percent total inhibition of such metastases in Laetrile-treated mice, as compared to controls, an experiment that at present not only proves the antineoplastic action of Laetrile, but proves it with a total success rate of at least 70 percent."

The controversial biochemist, who has argued that Laetrile is actually Vitamin B17 and that cancer is a dietary-deficiency disease, argued that current medical guidelines which define anti-cancer activity through measuring the effect of cancer drugs on the size of tumors are misleading.

They are misleading, he said, because the general rule is that the larger the tumor the less percentage there is of actual cancer tissue in it.

Claims made for Laetrile are that the substance only attacks cancer cells and halts their spread. The "legal" though admittedly poisonous anti-cancer drugs now in use attack all tissues. The attack frequently leads to a reduction in the size of a tumor—and also to a reduction in the overall health and life expectancy of the cancer patient, Krebs added.

It is the blocking of the spread of cancer—metastasis—and the subsequent increase in the feeling of well-being wherein lies Laetrile's effectiveness, Krebs noted, pointing out that all the Sugiura tests referred to just such results with the specially bred mice.

Pro-Laetrile forces have been arguing for decades that there is no reason "clinical" (that is, human) tests for Laetrile are not carried out, leading to its full acceptance and legalization in the United States.

Bradford, whose "freedom of choice" group claims about 20,000 members, including 600 physicians, and more than 300 chapters nationwide, said:

"Here we have further overwhelming evidence of the efficacy of Laetrile—and, sadly, further evidence of its apparent suppression in this country. We wonder how many thousands of mice must be saved by Laetrile before the product is made legally available for humans."

Bradford, by profession an engineer, also released copies of correspondence between Sloan-Kettering and Dr. Mario A. Soto de Leon, an oncologist of the 20 de Noviembre Hospital in Mexico City, which refers to a joint effort for planned human tests for Laetrile in Mexico. The tests never took place.

"If, as Sloan-Kettering keeps saying, no efficacy from Laetrile use was ever noted, then why were such human tests ever planned?" Bradford asked.

"We call on Sloan-Kettering to explain why animal studies indicating Laetrile efficacy are being suppressed, and why tests on humans, while planned, never took place," he added.

Culbert, to whom the Sugiura reports were released, is the author of Vitamin B17: Forbidden Weapon Against Cancer (Arlington House, 1974).

For more information, contact: The Committee for Freedom of Choice in Cancer Therapy, 146 Main Street, Suite 408, Los Altos, California 94022

Telephone: (415) 948-9475

Enclosures:
1. Sugiura Reports
2. Mexico Sloan-Kettering letters
3. Krebs report
4. The Choice