In Vivo Antitumor Activity of the Bitter Melon (Momordica charantia)¹

Catherine Jilka, Beth Strifler, G. William Fortner, Esther F. Hays, and Dolores J. Takemoto²

Department of Biochemistry [C. J., B. S., D. J. T.] and Division of Biology [G. W. F.], Kansas State University, Manhattan, Kansas 66506, and Laboratory of Biomolecular and Environmental Sciences, University of California, Los Angeles, California 90024 [E. F. H.]

ABSTRACT

The in vivo antitumor activity of a crude extract from the bitter melon (Momordica charantia) was determined. The extract inhibited tumor formation in CBA/H mice which had been given i.p. injections of 1.0 × 10⁶ CBA/D1 tumor cells (77% of the untreated mice with tumors versus 33% of the treated mice with tumors after 6 weeks). The extract also inhibited tumor formation in DBA/2 mice which had been given i.p. injections of either 1 × 10⁵ P388 tumor cells (0% of untreated mice survived after 30 days versus 40% survival of the treated mice) or 1 × 10⁶ L1210 tumor cells (0% survival of untreated mice versus 100% of treated mice after 30 days). The in vivo antitumor effect required both the prior exposure of tumor cells to the extract (2 hr) in vitro and i.p., briefery injections of the extract into the mice. The optimum dose for tumor inhibition (8 µg protein, biweekly, i.p.) was not toxic to mice for at least 45 days of treatment. This same treatment caused a marked enhancement of C3H mouse thymic cell response to concanavalin A in vitro. When compared to the untreated control mice, the bitter melon-injected animals exhibited a 4-fold-higher incorporation of tritiated thymidine into trichloroacetic acid-precipitable material after 4 hr of exposure to 50 µg of concanavalin A. Nylon wool-purified spleen cells from these same bitter melon-treated mice exhibited an enhanced mixed lymphocyte reaction when exposed to irradiated P388 stimulator cells (186% of the untreated control mice). These data indicate that in vivo enhancement of immune functions may contribute to the antitumor effects of the bitter melon extract.

INTRODUCTION

The bitter melon plant (Momordica charantia) contains several seed lectins which, while not highly toxic to animals in situ, will inhibit protein synthesis in vitro (13). Lin et al. (13) reported that 2 such lectins with molecular weights corresponding to 32,000 and 24,000 could be purified. These lectins inhibited Ehrlich ascites cell growth at relatively high concentrations (100 µg/ml or greater). The 2 purified lectins are called momordin (M, 24,000 protein) and agglutinin (M, 32,000 protein). The former inhibited protein synthesis (12–14). Barbieri et al. (1) reported that an additional M, 115,000 seed lectin inhibited protein synthesis in a rabbit reticuloocyte lysate system.

These lectins cause no apparent harm to animals when injected i.p. at 1 mg/100 g body weight (1). It is hypothesized that, like modeccin and similar toxins, these lectins cannot enter cells.

We (22, 26) and others (3) have observed similar activities in an aqueous extract from the bitter melon fruit. In these cases, however, cytostatic and cytotoxic effects were apparent on intact cells in culture. Vesely et al. (27) and Claffin et al. (3) reported that an aqueous extract of the ripe fruit contained both a guanylate cyclase enzyme inhibitor and a cytostatic factor which blocked rat splenic lymphocytes at the G₂-M phase of the cell cycle (3). We have also found that similar extracts prevent both Con A-stimulated thymidine incorporation into human peripheral blood lymphocyte DNA and a subsequent induction of a specific cyclic AMP phosphodiesterase (25).

This crude extract acts as a competitive inhibitor of guanylate cyclase activity in vitro and lowers cellular cyclic GMP levels in vivo (23). Inhibition is greater for the enzyme activity from leukemia lymphocytes when compared to normal human lymphocytes (245 µg/ml for normal human lymphocytes versus 170 µg/ml for leukemia lymphocytes) (23).

The most intriguing observation is the marked preferential cytotoxic effect of the extract for human leukemia cells (22). The LD₅₀ is greater than 5000 µg/ml for normal human peripheral blood lymphocytes versus 300 to 400 µg/ml for lymphocytes from patients with chronic or acute leukemia (8, 22, 23).

We have purified recently several of these cytostatic and cytotoxic factors (24). One such factor has a molecular weight corresponding to 50,000 to 70,000. This protein retains both guanylate cyclase-inhibitory activity and a preferential cytostatic effect on leukemic lymphocytes (22).

Our earlier observation that the crude extract contained multiple factors (26) has led recently to the purification of several additional components (24). One of these components also exhibits antiviral activity against vesicular stomatitis virus.

Finally, the observation that the crude extract from the bitter melon fruit reduced the occurrence of rat prostate adenocarcinoma in vivo (5) led us to attempt similar studies with several murine lymphoma systems. Although of a preliminary nature, these studies indicate clearly that the crude extract inhibits in vivo lymphoma formation in mice. Furthermore, this same extract appears to enhance the immune functions of these animals.

MATERIALS AND METHODS

The DBA/2 and C3H/HeN (MTV) mice (6 weeks to 2 months old, female) were obtained from the NIH. CBA/H mice were propagated at the University of California (Los Angeles). Con A was from Sigma Chemical Co. (St. Louis, Mo.).

Bitter melons (M. charantia, also called balsam pear) are grown locally and prepared initially as described (22). The whole ripe fruits (10 to 20 lb) are homogenized in cold PBS, filtered through cheesecloth, and then centrifuged at 16,300 × g (Sorvall GSA) for 20 min. The resulting supernatant is precipitated to 50% saturated ammonium sulfate, and the resulting pellet (from the above centrifugation step) is taken up in PBS and dialyzed against PBS overnight to remove residual ammonium sulfate. All procedures are conducted at 4°C unless otherwise stated. This crude aqueous extract is stored at −70°C in 50-ml batches and used as needed.

¹This research was supported by NIH Grants CA 27500 and CA 24196 and by a special fellowship from the Leukemia Society of America. This is Publication 83-156-1 from the Kansas Agricultural Experiment Station.
²To whom requests for reprints should be addressed.

Received April 25, 1983; accepted July 29, 1983.

NOVEMBER 1983
the source of crude bitter melon preparation. This extract is stable for up to 1 year at 

The *in vitro* dose-response curves are determined in 24-well microtiter plates (Costar, Cambridge, Mass.). Each well contained 1 x 10^6 cells, bitter melon extract or PBS, and medium to 1 ml final volume. The plates are incubated for 24 hr at 37° in an atmosphere of 95% air; 5% CO₂. Cell counts and viability are determined by hemocytometer counting and trypan blue dye exclusion, respectively.

Toxicity studies were conducted on 2-month-old C3H/HeN (MTV⁺) mice (male and female). Filter-sterilized (0.2-µm Acrodiscs; Gelman Instrument Co., Ann Arbor, Mich.) crude bitter melon extract was injected either i.p. or s.c. at doses of 8, 11, 16, 27, 31, and 50 µg protein per ml. Control animals received similar injections of sterile PBS. Animals were weighed and examined for gross pathological alterations.

*In vivo* antilymphoma activity was determined using 2-month-old CBA/ H mice. The animals were given injections of a CBA/DI T-cell lymphoma line established from a thymus of a CBA/H mouse with lymphomas induced by Gross murine leukemia virus. When injected i.p. at 10⁶ cells/mouse, the cells cause lymphomas and death within 30 days postinoculation in most of the mice.

All cell lines were maintained in Roswell Park Memorial Institute Tissue Culture Medium 1640 (Grand Island Biological Co., Grand Island, N. Y.), containing 10% calf serum (Dutchland Chemicals, Denver, Pa.), L-glutamine, and a penicillin:streptomycin mixture. These suspension cultures are grown in an atmosphere of 5% CO₂; 95% air.

Antilymphoma activity was measured in 6-week-old female DBA/2 mice given injections i.p. of either 1 x 10⁶ P388 cells or 1 x 10⁶ L1210 cells (both from American Type Culture Collection, Rockville, Md.).

Polyclonal lectin activation was measured in 6-week-old C3H/HeN (MTV⁺) mice which were given injections i.p. with 8 µg of protein of the bitter melon extract, biweekly, for 30 days. Control animals received PBS injections. Thymic lymphocytes were removed under sterile conditions, depleted of adherent cells, and purified on glass wool columns (11). Cells were cultured in 24-well microtiter plates at 2 x 10⁷ cells/ml as described above. Cells were harvested at various times after exposure to Con A.

For the final 3 hr of incubation, cultures were pulsed with 2 µCi each of tritiated thymidine ([methyl-3H]thymidine; 60 to 90 Ci/mmol; ICN Chemical Radioisotopes Div., Irvine, Calif.) and then harvested onto Whatman GF/A filters (Whatman, Inc., Clifton, N. J.). Filters are sequentially washed with PBS (3 times), 10% trichloroacetic acid, and 70% ethanol and then counted by liquid scintillation counting.

MLRs are measured in the same 6-week-old C3H/HeN (MTV⁺) mice which were used for polyclonal lectin activation studies. Mouse spleen cells are harvested, purified under sterile conditions, and cultured as described above at 1.5 x 10⁶ cells/ml for 3 days. Cells are stimulated with various numbers of irradiated P388 stimulator cells (Model 40 γ-cell small-animal irradiator; Atomic Energy of Canada, Ltd.; 2000 rads). For the last 8 hr of incubation, cultures are pulsed with 2 µCi of tritiated thymidine and then processed on GF/A filters as described above.

**RESULTS**

The LD₅₀ doses for the crude bitter melon extract were determined both i.p. and s.c. The LD₅₀ was 27 µg of protein per ml when injected s.c. and 16 µg of protein per ml when injected i.p. When mice were given injections of the LD₅₀ dose, death occurred within 3 to 5 days. When repeated lower doses were given (11 and 16 µg of protein per ml; Table 1), animal weight gain was stunted. However, animals appeared alert and fed normally. Weight returned to normal following termination of the injections (data not shown). Two mice from each group were sacrificed after 30 days and examined. No gross lesions were reported in any organ. However, a mild hepatic hyperplasia, some depletion of body fat, and a lower WBC were found, when compared to PBS-treated control animals (data not shown).

As indicated in Table 2, the crude extract is cytotoxic to the CBA/DI cells in culture. This toxicity is dose dependent and requires 24 hr before it becomes apparent. However, cells exposed to the crude extract for 30 min, washed, and then incubated for 24 hr will also exhibit a decreased viability (22). Thus, the constant presence of the crude extract is not required.

Although the crude extract was cytotoxic to CBA/DI cells *in vitro* (Table 2) after 24 hr of incubation, this treatment alone will not reduce the incidence of tumors (Table 3). No reduction in lymphoma formation was found, even when 10⁶ CBA/DI cells were incubated with 100 µg of bitter melon. It appears that injection of bitter melon directly into the mice is also necessary for antilymphoma activity. Thus, the antitumor activity of this extract may not be entirely a result of a direct effect on the CBA/ DI cells.

As indicated in Table 4, the injection of bitter melon i.p. into the mice does not cause an overall decrease in tumor incidence. However, a slight delay in the death of the animals is observed (5 of 6 mice by 53 days in the control groups versus 7 of 8 mice by 75 days in the group receiving injections beginning on Day 3).

The greatest antilymphoma effects were noted both when the CBA/DI cells were pretreated with the bitter melon extract and when animals were given injections biweekly (Table 5).

If animals were given 8 µg of bitter melon extract biweekly following the injection of pretreated lymphoma cells, the incidence of lymphoma formation was dramatically decreased (controls, 77% with lymphomas versus 30 to 33% of treated group).
C. Jilka et al.

Table 3
Effect of in vitro pretreatment of CBA/DI cells with crude bitter melon extract on subsequent lymphoma formation in mice

<table>
<thead>
<tr>
<th>Dose of bitter melon (µg protein/ml medium)</th>
<th>Animals with tumors* (no. dead/no. in group)</th>
<th>% dead at 60 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>9/10 (33-45)</td>
<td>90</td>
</tr>
<tr>
<td>20</td>
<td>10/10 (31-38)</td>
<td>100</td>
</tr>
<tr>
<td>50</td>
<td>9/10 (31-38)</td>
<td>90</td>
</tr>
<tr>
<td>100</td>
<td>8/10 (31-41)</td>
<td>80</td>
</tr>
<tr>
<td>Control</td>
<td>6/7 (31-38)</td>
<td>85</td>
</tr>
</tbody>
</table>

* CBA/DI cells (1 x 10⁶) were incubated with varying doses of crude bitter melon extract for 2 hr at 37°C. These cells were then washed once in PBS and inoculated, i.p., into mice. Cells were greater than 95% viable at the time of inoculation. No crude bitter melon was injected into the mice directly. Control cells were incubated in medium for 2 hr prior to i.p. injection. Numbers in parentheses, range of days after cells were given.

Table 4
Effects of multiple i.p. administration of the crude bitter melon extract on the lymphoma incidence in CBA/DI-inoculated mice

<table>
<thead>
<tr>
<th>Dose of bitter melon (µg protein/mouse)</th>
<th>Schedule*</th>
<th>Animals with tumors* (no. dead/no. in group)</th>
<th>% with tumors in 60 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (control)</td>
<td>No bitter melon treatment</td>
<td>6 (24-53)</td>
<td>84</td>
</tr>
<tr>
<td>8</td>
<td>Biweekly 8 µg injection: 3 days after cells given</td>
<td>7 (34-75)</td>
<td>87</td>
</tr>
<tr>
<td>8</td>
<td>8 µg biweekly: begin 6 days after cells given</td>
<td>9 (39-93)</td>
<td>55</td>
</tr>
<tr>
<td>8</td>
<td>8 µg biweekly: begin 9 days after cells given</td>
<td>10 (7-24-76)</td>
<td>70</td>
</tr>
</tbody>
</table>

* The crude bitter melon factor was given i.p. beginning 3, 6, or 9 days after the inoculation with the CBA/DI cells for 6 weeks.

Table 5
Effect of multiple i.p. administration of the crude bitter melon extract on the lymphoma incidence of the mice after inoculation with bitter melon-treated CBA/DI lymphoma cells

<table>
<thead>
<tr>
<th>Dose of bitter melon (µg protein/ml in vitro)</th>
<th>Schedule* of in vitro injections</th>
<th>Animals with tumors* (no. dead/no. in group)</th>
<th>% with tumors in 60 days</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 µg; biweekly: begin 3 days after cells given for 6 wk, total</td>
<td>18 (14-25-53)</td>
<td>77</td>
<td></td>
</tr>
<tr>
<td>8 µg; biweekly: begin 9 days after cells given for 3 mos., total</td>
<td>9 (3-62-73)</td>
<td>33</td>
<td></td>
</tr>
<tr>
<td>8 µg; biweekly: begin 3 days after cells given for 3 mos., total</td>
<td>10 (3-75-123)</td>
<td>30</td>
<td></td>
</tr>
</tbody>
</table>

* The crude bitter melon factor was given i.p. in biweekly doses of 8 µg of protein per mouse at 3 days postinoculation of CBA/DI cells. The CBA/DI cells were pretreated as described in Table 3 with 8 µg of bitter melon per ml.

Table 6
Effect of multiple i.p. administration of the bitter melon extract on tumor incidence in DBA/2 mice after injection with pretreated L1210 or P388 tumor cells

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Animals/group</th>
<th>Survival time (% alive after 30 days)</th>
</tr>
</thead>
<tbody>
<tr>
<td>L1210</td>
<td>Control group*</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Bitter melon-treated group*</td>
<td>5</td>
</tr>
<tr>
<td>P388</td>
<td>Control group*</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Bitter melon-treated group*</td>
<td>5</td>
</tr>
</tbody>
</table>

* DBA/2 mice (females, 6 weeks old) were each given an injection i.p. of 1 x 10⁶ P388 or L1210 cells. Cells were pretreated, prior to injection, with either PBS (control group) or with 8 µg of protein of the bitter melon extract (bitter melon-treated group) for 3 hr at 37°C. These treated cells were washed 3 times in sterile PBS and injected into the mice. The animals in the bitter melon-treated group were given injections biweekly with 8 µg of bitter melon for 30 days.

Furthermore, after 73 days of bitter melon treatment, these animals did not get lymphomas if treatment was stopped (observed for up to 6 months). At this point, we assume that all of the CBA/DI tumor cells have been killed.

Table 6 illustrates that the bitter melon extract also prevents tumor formation in DBA/2 mice given injections of either P388 or L1210 tumor cells. The tumor inhibition observed in these animals also required exposure of both animals and tumor cells to the bitter melon extract.

Since the antilymphoma effect was only apparent when both tumor cells and host animals were exposed to the bitter melon extract, we wished to determine if in vivo treatment with this extract enhanced the immune responses of the mice. Because the ability to reject a tumor in vivo is considered partially a result of a T-cell response against the tumor, we measured 2 T-cell responses, the ability of T-cells to proliferate when exposed to Con A and that when exposed to foreign tumor cells.

Chart 1 illustrates the results of exposing nylon wool-purified mouse thymic T-cells to Con A. These cells were taken from C3H/HeN (MTV+) mice which had been given injections i.p. with either PBS or bitter melon extract for 30 days. After exposure of these cells in vitro to Con A (50 µg/ml), the cells from the bitter melon-treated animals incorporated more tritiated thymidine at an earlier time period than did those T-cells obtained from PBS-treated mice. Since the bitter melon extract was not added directly to the in vitro incubation, we assume that these differences in response to Con A must be a result of in vivo exposure to the extract. This enhanced response to Con A was observed as (a) greater incorporation of tritiated thymidine into cells, (b) earlier time of peak responsiveness, and (c) responsiveness at a lower concentration of Con A (data not shown). In vivo treatment is not directly mitogenic to the mouse thymic cells. Rather, another signal (Con A, in this case) is needed. We are uncertain as to the mechanism by which the extract primes these cells in vivo. Small doses of the extract, added in vivo to normal thymocytes, with Con A, have no effect on subsequent mitogen-induced proliferation. However, at higher doses (around 1 µg of protein), the extract inhibits Con A-induced proliferation of normal mouse thymocytes. No effect is noted on MLR-induced proliferation of normal mouse spleen cells (data not shown).

Table 7 illustrates the results of an MLR using nylon wool-purified spleen cells obtained from the same animals as described in Chart 1. The cells isolated from the bitter melon-injected animals exhibited a greater MLR response to the irradiated P388 stimulator cells than did those cells purified from the PBS-treated control group (about 180% of the control group). This enhanced reaction was seen when varying the effector:stimulator ratio from 1:1 to 1:100. The optimum ratio was the same for each group, however. Since the bitter melon extract was not added to the in vitro incubation, we assume that these differences in response to Con A must be a result of in vivo exposure to the extract. This enhanced response to Con A was observed as (a) greater incorporation of tritiated thymidine into cells, (b) earlier time of peak responsiveness, and (c) responsiveness at a lower concentration of Con A (data not shown). In vivo treatment is not directly mitogenic to the mouse thymic cells. Rather, another signal (Con A, in this case) is needed. We are uncertain as to the mechanism by which the extract primes these cells in vivo. Small doses of the extract, added in vivo to normal thymocytes, with Con A, have no effect on subsequent mitogen-induced proliferation. However, at higher doses (around 1 µg of protein), the extract inhibits Con A-induced proliferation of normal mouse thymocytes. No effect is noted on MLR-induced proliferation of normal mouse spleen cells (data not shown).
vitro incubation, we assume that these differences are due to in
vitro effects of the extract on the mouse spleen cells.

DISCUSSION

There is a tremendous legacy of folklore involving the use of
plant preparations in medicine. Hartwell (8) has reported that
over 3000 species have been used in cancer treatment alone.
Among those utilized most recently are maytansine and vincris-
tine, both of plant origin (21, 28).

Until recently, very little was known about specific anticancer
proteins from plants. Although plant proteins which are toxic to
animal cells are widely distributed among different species (7),
their mechanisms of action are not known in detail.

However, considerable progress has been made on the mech-
amanism of action of plant toxins, usually of seed origin. Among
these toxins, abrin and ricin have been studied most extensively
(12, 14). These seed toxins are from Abrus precatorius and from
Ricinus communis, respectively. While both abrin and ricin are
extremely toxic to normal cells, they have been reported to be
more toxic towards malignant cells.

The toxicity, in vivo, appears to depend on the presence of
both toxin subunits; the B chain is needed for cellular binding,
while the A chain shuts off protein synthesis (16). The latter acts
enzymatically by preventing the ribosomal-linked GTP hydrolysis
catalyzed by Elongation Factor 2 at the 60S ribosomal subunit
(20).

Initially, these seed toxins appeared to be too toxic for use as
anticancer agents. Recently, however, through the specific cou-
pling of abrin or ricin to monoclonal antibodies directed against
tumor cell surface antigens, a more "selective delivery" has been
achieved (9). For example, Thy 1.1-specific monoclonal antibod-
ies covalently linked to ricin will selectively kill only Thy 1.1-
positive cells in culture (9).

Other plant seed toxins appear to act by the same mechanism
(7). However, some do not bind to cells and therefore only inhibit
protein synthesis in cell-free systems (7). Among this group are
modocin (6, 19) and momordica lectins (1).

Recently, plant lectins have been found to inhibit tumor for-
mation in vivo (4). Although a direct effect is observed in the
tumor cell, enhancement of host-cell immune functions is also
observed. Grifonia simplicifolia ganglioside I lectin, for example,
prevents in vivo formation of Ehrlich ascites tumors in mice when
injected i.p. (4). It appears that direct interaction of tumor cells
with the lectin is required. Pretreatment of Ehrlich ascites cells
in vitro for 1 hr prevented subsequent tumor formation by these
same cells when injected into the animal.

However, the lectin-mediated prevention of tumor formation
also involved activation of pertussode macrophages which med-
iate tumor cell lysis (15). In vitro incubation of macrophages
caused these cells to develop a ganglioside I-binding cell surface
glycoprotein. Appearance of this glycoprotein correlated with the
appearance of activated macrophages. It has been proposed
that treatment of the tumor cells and macrophages facilitates
recognition by both cell types.

Although this is an attractive hypothesis, it does not fully
explain the effects which we have observed in our system.

We have observed a markedly enhanced reaction of mouse
thymic and spleen cells to Con A and in a MLR, respectively.
Since both the tumor cells and bitter melon extract are adminis-
tered i.p., this implies that there may be a generalized enhance-
ment of host animal immune functions after treatment with the
extract.

Enhancement of immune functions could occur through the
action of lectins which may be present in the crude bitter melon
extract. However, in our system, stimulation of immune functions
does not appear to be sufficient for tumor inhibition. Rather,
interaction of some component in the bitter melon extract directly


cancerres.aacrjournals.org Downloaded from on July 9, 2017. © 1983 American Association for Cancer Research.
with the tumor cells is also required. At this point, we cannot determine if an immune enhancement contributes to the antitumor effect of the bitter melon extract. Further studies showing a reduction of antitumor activity of the extract in immune-deficient mice would answer this question.

We feel that there may be several components in this extract which are acting via different mechanisms.

We have purified several antileukemia factors previously from this extract (24, 26). One such factor acts as a competitive inhibitor of human lymphocytic guanylate cyclase in vitro and lowers cellular cyclic GMP levels of human leukemia cell lines in culture (23).

The 50%-inhibitory dose for purified soluble guanylate cyclase inhibition by the bitter melon factor is around 7 μg of protein for the leukemic cell enzyme and around 100 μg of protein for the enzyme from normal human lymphocytes (23).

Another intriguing observation is the marked preferential cytotoxic (at higher doses) or cytostatic (at lower doses) effect of the factor for human leukemia cells (22, 26). This preferential effect could be due to the presence of the more sensitive guanylate cyclase found in the leukemia cells (22, 26).

Since cyclic GMP is elevated in these leukemic cells (22, 26), the inhibition of guanylate cyclase could serve to return the cyclic AMP-cyclic GMP ratio to normal, resulting in a decreased rate of cell proliferation and, perhaps, cell death.

It has been demonstrated recently that in vitro or in vivo manipulation of human lymphocytic and monocytic cyclic AMP or cyclic GMP levels alters dramatically both cell growth and specific cell differentiation of both normal and neoplastic cells (10, 11, 17, 18).

Further purification of these factors from the bitter melon extract should determine which are necessary for optimum in vivo antitumor activity and what their mechanisms of action are.

ACKNOWLEDGMENTS

We gratefully acknowledge the excellent technical assistance of Fran Jilka and Nadine Morganatier.

REFERENCES

In Vivo Antitumor Activity of the Bitter Melon (Momordica charantia)

Catherine Jilka, Beth Strifler, G. William Fortner, et al.


Updated version
Access the most recent version of this article at:
http://cancerres.aacrjournals.org/content/43/11/5151

E-mail alerts
Sign up to receive free email-alerts related to this article or journal.

Reprints and Subscriptions
To order reprints of this article or to subscribe to the journal, contact the AACR Publications Department at pubs@aacr.org.

Permissions
To request permission to re-use all or part of this article, contact the AACR Publications Department at permissions@aacr.org.