Anticancer Activity of Selected *Colocasia gigantea* Fractions

Apichai Pornprasertpol MEng*,
Amornpun Sereemaspun MD, PhD**, Kanidta Sooklert MSc**,
Chutimon Satirapipatkul MSc*, Suchada Sukrong PhD***

* Department of Chemical Engineering, Faculty of Engineering, Chulalongkorn University, Bangkok, Thailand
** Nanobiomedicine Laboratory, Department of Anatomy, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand
*** Department of Pharmacognosy and Pharmaceutical Botany, Faculty of Pharmaceutical Sciences, Chulalongkorn University, Bangkok, Thailand

The objective of this study is to investigate the anticancer potential of the extract of *Colocasia gigantea* (*C. gigantea*), a plant member of the Araceae family. In the present study, we investigated the cytotoxic activity of *C. gigantea* extract on cervical cancer (Hela) and human white blood cells (WBC) in vitro. The authors then identified the bioactive ingredients that demonstrated cytotoxicity on tested cells and evaluated those bioactive ingredients using the bioassay-guided fractionation method. The results showed that not all parts of *C. gigantea* promote cytotoxic activity. The dichloromethane leaf fraction showed significant cell proliferation effect on Hela cells, but not on WBCs. Only the n-hexane tuber fraction (Fr. 1T) exhibited significant cytotoxicity on Hela cells (IC₅₀ = 585 μg/ml) and encouraged WBC cell proliferation. From GC-Mass spectrometry, 4,22-Stigmastadiene-3-one, Diazoprogesterone, 9-Octadecenoic acid (Z)-, hexyl ester, and Oleic Acid were the components of Fr. 1T that demonstrated cytotoxic potential. In conclusion, *C. gigantea*‘s Fr. 1T shows potential for cervical cancer treatment.

**Keywords:** *Colocasia gigantea* (Araceae), Cervical cancer, Cytotoxicity, Bioactive ingredients

Full text. e-Journal: http://www.jmatonline.com

Cancer is a disease of uncontrollable cell fission in malignant tumors that invade normal human body systems(1,2). Many factors are known to increase the risk of contracting cancer, such as environmental pollutants, radiation, stress, obesity, and diet(2). Cancer is a primary health crisis problem for people in many parts of the world(1-3). Thailand is one of the many countries that has been affected by cancer. National Cancer Institute Thailand, a cancer research and care center under the Thailand Ministry of Public Health reported 3,314 new cancer patients in Thailand in 2009(4). Among all cancers, cervical cancer is the most common carcinoma found in Thailand(4).

Many studies and articles from all over the world have reported the anticancer activity of phytochemical ingredients(5,6). Plants are a source of natural bioactive compounds. Various compounds are cytotoxic and therefore have the potential for anticancer activity(7-11). When contained in nutrients and foodstuffs, the cytotoxicity of phytochemicals must target specific cells, such as cancer cells, and must not be harmful to consumers(1).

*Colocasia gigantea* Hook. f. was selected for this study. *C. gigantea* belongs to the Araceae family, similar to *Colocasia esculenta* (L.) Schott (Taro). *C. gigantea* grows commonly in Thailand and other Southeast Asian countries(12). In the Pacific islands, the tubers are cooked and eaten as a starch(12). In India and Bangladesh, the tubers are used as a main ingredient in curries and stews(13). In Thailand, *C. gigantea* is considered as a minor food crop and is mainly utilized as a stem vegetable. *C. gigantea*‘s stem is often used for making homemade Thai food called “Bon curry”. In Thai traditional medicine, *C. gigantea* tuber is heated over a fire. It is used to reduce “internal heat” (fever) and also for the treatment of drowsiness. Fresh tuber has been shown to ameliorate stomach problems, combat infection, and accelerate the healing...
of wounds. In the northern region of Thailand, fresh or
dried tuber is used for the treatment of phlegm by mixing
it with honey[14].

The objective of the present study was to
explore C. gigantea as an anticancer agent against
cervical cancer cells. Our method of investigation
centered on the use of Hela in vitro and bioassay-
guided fractionation techniques to identify bioactive
ingredients in the active fractions of C. gigantea.

Material and Method

**Extraction**

Tuber and leaf components of C. gigantea
were obtained from the Department of Anatomy, Faculty
of Medicine, Chulalongkorn University. The tuber and
leaf samples were washed with water, cut, and dried by
dehydrator at 40°C for 5 days. They were then ground
into small pieces with a grinder. C. gigantea extract
samples were obtained by macerating the C. gigantea
in n-hexane (n-C₆H₁₄) for 3 days at room temperature;
this step was performed 3 times. The mixture was then
filtered using a vacuum filter. The filtrate was
concentrated to remove n-C₆H₁₄ under reduced
pressure by rotary vacuum evaporator to obtain a crude
n-C₆H₁₄ fraction. Marc was then extracted using
dichloromethane (CH₂Cl₂) and then again using
methanol (CH₃OH), with increasing polarity. For both
dichloromethane and methanol, the same extraction
process used for n-hexane (n-C₆H₁₄) was used. These
fractions were evaluated for cytotoxic activity, with the
most active fractions identified for further fractionation.
The C. gigantea extraction process is illustrated in
Fig. 1.

**Fractionation**

Column chromatography was used for isolating compounds which stationary phase was silica
gel 60 (230-400 mesh ASTM). Thin layer
chromatography (TLC, aluminum sheet silica gel 60 F₂₅₄)
was used for the detection of compound fractions. UV
light at wavelengths of 254 and 365 nm and 5%
anisaldehyde in sulfuric acid heated to 110°C were used
for the detection of compound fractions on TLC sheets.

**Identification of fractions**

Modification of Mitova et al 2003[15], GC-Mass
spectroscopy (Leco Corporation, St. Joseph, MI, USA)
was used for fraction identification. The column HP5-
MS (30 m x 0.25 mm, 0.25 μm film thickness) was used.
The injector port was set at 300°C. The injection volume
was 0.2 μl and injections were carried out under split/
splitless: split/split ratio: 1:20. Helium was used as a
carrier gas with a constant flow of 1 ml/min. The
temperature was programmed from 40°C (2 min) to 300°C
at a rate of 6°C min⁻¹ with a 10 min hold. The fractions in
the test solution were identified by comparing the
spectra with known compounds stored in the internal
library.

**Cell lines and culture**

Cervical cancer cells (Hela) were obtained from
the Department of Anatomy, Faculty of Medicine,
Chulalongkorn University. Human white blood cells
(WBC) were collected from the blood of one of the
research authors and this sample was used as the
primary cell line in the experiment. Hela cells were
maintained in Dulbecco’s Modified Eagle’s Medium
(DMEM). WBCs were maintained in Roswell Park
Memorial Institute Medium (RPMI) at 37°C in an
incubator containing 5% CO₂. Both DMEM and RPMI
were supplemented with 10% (v/v) fetal bovine serum
(FBS), 1% penicillin/streptomycin, and 3.7 g/L sodium
bicarbonate (Na₂CO₃).

**In vitro assay for cytotoxicity**

Hela cells were washed two times with
phosphate buffered saline (PBS) and were then
collected by trypsinization. Hela cells and WBC cells were stained with trypan blue. Cell counts were adjusted to 1 x 10^3 cell/well and seeded in 96-well plates containing DMEM medium for Hela and RPMI medium for WBC cells and then incubated for 24 hours in an incubator. After 24 hours of incubation, the Hela and WBC cells were treated with various concentrations of *C. gigantea* fractions and then incubated for an additional 48 hours. *C. gigantea* fraction-free plates containing DMEM for Hela and RPMI for WBC were used as a negative control. Doxorubicin (Adriamycin), an effective anticancer drug, was used as a positive control. PrestoBlue™ Cell Viability Reagent was used as a cell viability indicator after treatment (48-hour incubation). 10 μl of PrestoBlue™ Cell Viability Reagent was added to each well and then incubated for 30 minutes. Cytotoxicity was determined by measurement of the fluorescence of the converted PrestoBlue™ Cell Viability Reagent at a wavelength of 590 nm in a microplate reader (BioTek Synergy HT, Winooski, VT, USA). Percent (%) of cell viability was calculated from:

\[
\text{Cell viability} (\%) = \frac{\text{Treated cells}}{\text{Negative control}} \times 100
\]

**Statistical analysis**

All experiments were conducted as three independent replicates. The experimental data are shown in terms of both mean and standard deviation. The data were analyzed using Microsoft Excel software (Microsoft Software Inc., Redmond WA, USA) and GraphPad Prism 5 (GraphPad Software Inc., San Diego, CA, USA).

**Results**

**Screening of crude n-hexane, dichloromethane, and methanol fractions for cytotoxicity**

The objective of the initial experiment was to screen the crude n-C\textsubscript{6}H\textsubscript{14}, CH\textsubscript{2}Cl\textsubscript{2}, and CH\textsubscript{3}OH fractions of tuber and leaf parts of *C. gigantea* for cytotoxicity. The concentrations of each fraction were 62.5, 125, 250, 500, and 1,000 μg/ml. As shown in Table 1, only fraction 1T from the tuber part of *C. gigantea* exhibited cytotoxicity on Hela cells. The half maximal inhibitory concentration value (IC\textsubscript{50}) was 585 μg/ml for Hela cells. Fig. 2 describes how increasing concentrations of fraction 1T resulted in a dramatic decrease in Hela cell viability. The IC\textsubscript{50} value of doxorubicin on Hela cells was 7.03 μM, which is equivalent to 3.82 μg/ml. Fraction 1T from the tuber part not only expressed cytotoxicity on Hela cells, but also significantly stimulated WBC cell viability.

<table>
<thead>
<tr>
<th>Fractions</th>
<th>IC\textsubscript{50} (μg/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hela</td>
</tr>
<tr>
<td>1T</td>
<td>585</td>
</tr>
<tr>
<td>2T</td>
<td>-</td>
</tr>
<tr>
<td>3T</td>
<td>-</td>
</tr>
<tr>
<td>1L</td>
<td>-</td>
</tr>
<tr>
<td>2L</td>
<td>-</td>
</tr>
<tr>
<td>3L</td>
<td>-</td>
</tr>
<tr>
<td>Doxorubicin</td>
<td>3.82</td>
</tr>
</tbody>
</table>

Fraction cytotoxicity is considered statistically significant if >1,000 μg/ml and doxorubicin cytotoxicity is considered statistically significant if >5.43 μg/ml.

**Fig. 2** Screening of crude n-C\textsubscript{6}H\textsubscript{14}, CH\textsubscript{2}Cl\textsubscript{2}, and CH\textsubscript{3}OH fractions for cytotoxicity in vitro: (A) Growth inhibition of leaf part fractions on Hela cells, (B) growth inhibition of tuber part fractions on Hela cells, (C) growth inhibition of doxorubicin on Hela cells, (D) growth inhibition of leaf part fractions on WBC cells, (E) growth inhibition of tuber part fractions on WBC cells, (F) growth inhibition of doxorubicin on WBC cells (the triple asterisk (***)) indicates a significant difference from the control *p*<0.001, one-way ANOVA, Dunnett’s test) Results are mean values ±SD of independent experiments performed in triplicate.
Inexplicably, fraction 2L from the leaf part stimulated Hela cell proliferation very significantly (Fig. 2). Hela cell proliferation varied according to fraction 2L concentration, with the highest level of cell proliferation occurring at 250 μg/ml. The IC₅₀ values of all fractions and doxorubicin on WBC cells were more than 1,000 μg/ml and 10 μM (equivalent to 5.43 μg/ml), respectively. From the experimental results, fraction 1T was selected for further investigation.

Bioassay-guided fractionation of the fraction 1T

Fraction 1T was loaded into column chromatography, which was packed with silica gel and eluted sequentially with n-C₆H₁₄ (100), followed by CH₂Cl₂: C₄H₈O₂ (100:0, 90:10, 80:20, 70:30, 60:40, 50:50, and 0:100), to yield four fractions (4T, 5T, 6T and 7T). Each fraction was once again subjected to cytotoxicity testing to observe the most active fraction. As described in Table 2, fraction 5T (CH₂Cl₂: C₄H₈O₂; 80:20) was associated with the lowest IC₅₀ value (IC₅₀ = 93.0 μg/ml). Fig. 3 further illustrates that IC₅₀ values for all fractions exceeded 500 μg/ml with regard to WBC cells. Moreover, fraction 5T not only delivered the lowest IC₅₀ value with regard to Hela cells, but also stimulated significant WBC cell proliferation. Increasing concentrations of fraction 5T dramatically decreased Hela cell viability. Therefore, fraction 5T was chosen for further fractionation. Fraction 5T was then subjected to column chromatography on silica gel eluted with n-C₆H₁₄: CH₂Cl₂: C₄H₈O₂ (30:65:5) to yield four fractions (8T, 9T, 10T and 11T). Once again, a descriptive outline of the fractionation process is illustrated in Fig. 1. Additionally, Fig. 4 pictorially describes Hela cell morphology resulting from treatment with fraction 5T.

The bioactive ingredients in fractions 8T, 9T, 10T and 11T, which were fractionated from fraction 5T, were analyzed by GC-Mass spectroscopy and the analyzed results are shown in Table 3. Analysis of the chromatograms showed that the most commonly occurring and the highest peak height of expected bioactive compounds found in fractions 8T, 9T, 10T and 11T belonged to 4,22-Stigmastadiene-3-one.

Table 2. Cytotoxicity of 4 fractions fractionated from fraction 1T on Hela and WBC cells in vitro

<table>
<thead>
<tr>
<th>Cell</th>
<th>IC₅₀ (μg/ml)</th>
<th>Fraction 1T</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>4T</td>
<td>5T</td>
</tr>
<tr>
<td>Hela</td>
<td>104</td>
<td>93.0</td>
</tr>
<tr>
<td>WBC</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Fraction cytotoxicity is considered statistically significant if > 500 μg/ml and Doxorubicin cytotoxicity is considered statistically significant if > 5.43 μg/ml.

Fig. 3 Cytotoxicity of 4T, 5T, 6T and 7T fractions in vitro: (A) The cytotoxicity of each fraction on Hela cells; (B) The cytotoxicity of each fraction on WBC cells (the triple asterisk (***) indicates a significant difference from the control p<0.001, one-way ANOVA, Dunett’s test). Results are mean values ±SD of independent experiments performed in triplicate.
Fig. 4  Hela cell morphology after being treated fraction 5T: (A) control; (B-F) Hela cells treated with fraction 5T concentrations of 32.25, 61.5, 125, 250, and 500 μg/ml, respectively.

Discussion

*C. esculenta* has been reported to contain several biologically active ingredients and has been used as a food and a traditional medicinal plant(14). Although, there are many reports that highlight the biological activities of the phytochemicals in *C. esculenta*, there are few reports on *C. gigantea*’s biological activities and its bioactive ingredients. This study is the first to report the cytotoxic activity and the suspected bioactive ingredients of some *C. gigantea* fractions on Hela cells. The results showed that not all parts of *C. gigantea* demonstrated cytotoxicity on Hela cells. In fact and paradoxically, some *C. gigantea* leaf part fractions promoted significant Hela cell proliferation. Similarly, Wei et al (2011)(16) reported that extract from the leaf part of *C. esculenta* encouraged human breast adenocarcinoma (MCF-7) proliferation. Accordingly, cancer patients should perhaps avoid reasonable precautions against the consumption of *C. gigantea* leaf parts as a food ingredient. *C. gigantea* fraction 1T, which comes from the tuber portion of the plant, demonstrates two different pathways for Hela cell inhibition. First, it directly restrains Hela cell proliferation by the effects of its bioactive ingredients. Second, fraction 1T encourages the immune system by acting as a mitogen. Ohno et al (1994)(17) reported that mitogen activated lymphocytes can kill numerous types of colon cancer cells in humans and in rodents. To demonstrate that the cytotoxicity of the bioactive ingredients in this case is specific to Hela cells, but is safe for human white blood cells, fraction 1T from the tuber part of *C. gigantea* stimulated WBC cell proliferation significantly. The experimental results on WBC cells in the present study were similar to the results reported by Brown et al (2005)(5), in which normal mouse splenocyte cells were treated with *C. esculenta* extract. Brown et al suggested that *C. esculenta* contains an endogenous mitogen and a mannose-binding lectin, similar to *Phasleus vulgaris*, that activates lymphocytes(18,19). Some lectins can induce lymphocyte proliferation through interleukin-2 production. Lymphocytes incubated 1-2 days with a high dose of interleukin-2 induced normal lymphocytes to become lymphokine-activated killer cells (LAK). LAK cells have a non-specific tumoricidal activity that kills various types of cancer cells(17).

The main idea of bioactive ingredients in anti-cancer property *in vitro* is that it acts on non-nutritive plant compounds(5). Bioassay-guided fractionation was the method used for the identification of active ingredients. Column chromatography was used for the fractionation of bioactive ingredients. TLC and GC-Mass spectroscopy were used to observe various components contained within each fraction.

4,22-Stigmastadiene-3-one and Diazoprogesterone can be classified as a phytosterol. There have been many reports regarding the anticancer activities of phytosterol(20,21). Phytosterol can act against carcinogenesis by various mechanisms. The authors expected that phytosterol in *C. gigantea* might be a bioactive ingredient that plays an important role as an anticancer compound. In 2004, Awad et al(22) reported on mechanisms of phytosterol acting on cancer cells. Phytosterol suppressed cancer development by reducing the production of carcinogens. In 2005, Vivancos and Moreno(23) reported that phytosterol increased the activities of antioxidant enzymes, superoxide dismutase, and glutathione peroxidase in cultured macrophages. They suggested that phytosterol prevented cells from damage by reactive oxygen species. Phytosterol also induces apoptosis in cancer cells, an important mechanism in the inhibition of carcinogenesis(20). Park et al (2007)(24), Moon et al (2007)(25), and Rubis et al (2008)(26) published the...
apoptosis mechanism of β-sitosterol and showed that it increased the activity of caspase-3 and, at the same time, deactivated the Bel-2 pathway. Furthermore, Moon et al (2007) explained the activation of caspase-3 showing that phytosterol could be mediated by extracellular signals that were complemented by mitochondrial pathways. Moreover, phytosterol also inhibited angiogenesis and metastasis in cancer cell proliferation. Choi et al (2007) found basic fibroblast growth factor (bFGF)-induced angiogenesis in endothelial cells isolated from human umbilical veins and successfully reduced proliferation of the cells with campesterol treatment. The anticancer property of Oleic Acid was proposed by Menendez et al (2005). They observed that Oleic Acid supported trastuzumab (Herceptin, an anticancer drug) when used in cancer cell cultures. They suggested that Oleic Acid up-regulates polyomavirus enhancer activator 3 (PEA3), which suppressed the expression of human epidermal growth factor receptor 2 (HER2/neu), a cause of breast cancers. Therefore, high levels of PEA3 would result in lower levels of HER2/neu, thereby lowering the risk of cancer onset as a result of HER2/neu over-expression.

Valko et al (2007) and Sakthivel et al (2010) reported that Diazoprogesterone, 9-Octadecenoic acid (Z)-, hexyl ester, and Oleic Acid not only displayed anticancer activity, but also had antimicrobial, hypercholesterolaemic, and anti-ulcerogenic effects.

**Conclusion**

The potential anticancer properties of *C. gigantea* tuber parts had the combined effect of...
Table 4. The chemical ingredients of fractions 10T and 11T that were isolated from fraction 5T and analyzed by GC-Mass spectroscopy

<table>
<thead>
<tr>
<th>No.</th>
<th>Rate (sec)</th>
<th>Molecular Formula</th>
<th>%</th>
<th>Name</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Formula</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fraction 10T</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>150.7</td>
<td>C₆H₁₂O</td>
<td>2.66</td>
<td>Furan, tetrahydro-2,5-dimethyl-</td>
</tr>
<tr>
<td>2</td>
<td>226.45</td>
<td>C₅H₁₀O₂</td>
<td>4.44</td>
<td>1-Propen-2-ol, acetate</td>
</tr>
</tbody>
</table>
| 3   | 239.55     | C₃₂H₄₁NO₃        | 10.36 | 3‘H-Cycloprop(1,2) cholesta-1,4,6-trien-3-one, 1′-carboethoxy-1′-cyano-1a,2a-dihydro-
| | |                   |       | Hexanal                                                               |
| 4   | 240.45     | C₆H₁₂O            | 16.27 | Hexanal                                                               |
| 5   | 573.45     | C₆H₁₂O₂           | 0.65  | Pentanoic acid, 2-methyl-                                              |
| 6   | 700.65     | C₇H₁₄O₃           | 0.76  | Oxalic acid, allyl heptyl ester                                        |
| 7   | 728.75     | C₃₂H₄₅NO₃        | 1.69  | Octanoic acid, methyl ester                                           |
| 8   | 932.95     | C₁₂H₂₀O₃          | 0.94  | 4-Pentenal, 2-ethyl-                                                 |
| 9   | 1026       | C₉H₁₆O₂           | 1.42  | Methyl 8-octoctanoate                                                |
| 10  | 1040.35    | C₁₀H₂₀O₂          | 1.01  | 2-n-Hexylocyclopentane                                               |
| 11  | 1116       | C₁₀H₂₀O₂          | 4.78  | 4-Octenoic acid, methyl ester, (Z)-                                  |
| 12  | 1160.9     | C₁₀H₂₀O₂          | 9.82  | Nonanoic acid, 9-oxo-, methyl ester                                  |
| 13  | 1627.7     | C₁₆H₃₂O₂          | 1.71  | 2-Heptanone, 5-methyl-                                               |
| 14  | 1712.2     | C₁₆H₃₂O₂          | 0.96  | 2-Methylheptanoic acid                                               |
| 15  | 1869.95    | C₁₃H₂₂O₃          | 2.69  | Décanoic acid, propyl ester                                          |
| 16  | 2023.7     | C₁₂H₂₄O₂          | 18.08 | 9-Undecenoic acid (Z)-, hexyl ester                                  |
| 17  | 2047       | C₁₂H₂₄NO          | 0.77  | Dimethylamine, N-(neopenelyloxy)-                                    |
| 18  | 2051.7     | C₁₂H₂₄O₂          | 2.92  | Nonanoyl chloride                                                    |
| 19  | 2095.35    | C₁₀H₁₆O₂          | 1.44  | Tetrahydrofuran-2-one, 3-[2-pentenyl]-4-methyl-                      |
| Fraction 11T |          |                   |       |                                                                     |
| 1   | 151.15     | C₁₀H₂₀O₂          | 1.40  | Furan, tetrahydro-2,5-dimethyl-                                        |
| 2   | 536.55     | C₁₆H₃₂O₂          | 13.02 | Pentanoic acid                                                        |
| 3   | 1115.75    | C₁₆H₃₂O₂          | 2.69  | Hexanoic acid, methyl ester                                           |
| 4   | 2022.7     | C₁₈H₃₄O₃          | 23.58 | Oleic Acid                                                            |
| 5   | 2050.4     | C₁₁H₂₆O₂          | 2.43  | Decanoic acid, 2-oxo-, methyl ester                                  |
| 6   | 2240.15    | C₂₃H₄₈O₄S₂        | 17.86 | 2a,4a-Epoxyethylphenanthrene-7-methanol, 1,1-dimethyl-2-methoxy-8-(1,3-dithiin-2-ylidine) methyl-1,2,3,4,4a,4b,5,6,7,8,8a,9-dodecahydro-, acetate |

inhibiting cervical cancer Hela cell proliferation and stimulating human white blood cell growth. These experimental results may stimulate future research and in vivo mechanism studies on cell death. The results demonstrate that a fraction extracted from C. gigantea tuber parts (using n-hexane (n-C₆H₁₄) as a solvent) contains powerful bioactive ingredients with anticancer properties. With further study, these findings may contribute to the development of new-targeted cancer chemotherapy agents.

Acknowledgement

The authors gratefully acknowledge members of Nanobiomedical Laboratory, Faculty of Medicine Chulalongkorn University, for their technical assistance. The present study was supported in part by the Thailand Research Fund (TRF-MRG 5180151), Chulalongkorn University Centenary Academic Development Project (Under the Center of Innovative Nanotechnology, Chulalongkorn University), and Ratchadaphiseksomphot Endowment Fund of Chulalongkorn University (RES560530230-AM).

Potential conflicts of interest

None.

References

2. Anand P, Kunnumakkara AB, Sundaram C,


อุปหัตถานะเร็วของส่วนพืช สุทธิภรณ์จาก COLOCASIA GIGANTEA

นางวิจัยมีข้อการศึกษาที่จะตรวจสอบคุณค่าในการคลองตันเร็วของสารสกัดจากตนบอนคุณ (Colocasia gigantea) ซึ่งอยู่ในวงศ์ Araceae ในการค้นหาได้สารของอุทุมควัฒเป็นพืชตัวเองของสารสกัดจากตนบอนคุณได้เชื่อมต่ำเร็ปคฤษละ (Hela) และเซลล์เนื้อเยื่อ

ผลการทดลองแสดงให้เห็นว่าสารสกัดจากตนบอนคุณที่มีคุณสมบัติที่เกิดความเป็นพืชตัวเองด้วย bioassay-guided fractionation ผลการทดลองแสดงให้เห็นว่าสารสกัดจากตนบอนคุณที่มีคุณสมบัติในเรื่องของการปรับคุณสมบัติของเซลล์เนื้อเยื่อในข้อคือคุณมีที่มีความมุ่งหมายในการทดลองแบบทางวิทยาการเร็วตัวใดของเซลล์เนื้อเยื่อในเกณฑ์ของคุณให้กับเซลล์เนื้อเยื่อในตัวตัวละ люди (Fr.1T) นี้ความเป็นพืชตัวเองจะเร็ปคุณละ (IC50 = 585 ไมโครกรัมต่อลิตร) ที่มีผลการเร็วตัวใดของเซลล์เนื้อเยื่อ ผลการวิเคราะห์ย่อยเทคนิค GC-MS พบว่าสารสกัด Fr.1T ประกอบไปด้วย 4,22-Stigmastadiene-3-one, Diazoprogesterone, 9-Octadecenoic acid (Z), hexyl ester และ Oleic acid ซึ่งคาดว่าจะมีคุณสมบัติเป็นพืชตัวเองเนื่องจากสูตรสารสกัด Fr.1T จากตนบอนคุณมีคุณสมบัติที่จะใช้ปรับคุณสมบัติได้