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Review

Anticancer effect of herbal and marine products: A systematic review

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ABSTRACT

The majority of the world's nations have faced the second-highest cancer mortality rate. The main causes of cancer include an unbalanced diet, genetic factors, and a few specific environmental substances. Recently, a variety of substances have been used to treat cancer, and some are still being studied. It has long been known that the mid of the twentieth century that plant and marine species create a wide range of chemically and physiologically diverse metabolites with a variety of biological effects, including anticancer, anti-inflammatory, antioxidant, antibacterial, antifouling and so on. The focus of this study is on newly found compounds from plant and marine sources that have potent anticancer effects.

Contents

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1. Introduction

Cancer is a disorder in which cells in a particular area of the body multiply and develop uncontrolled. The malignant cells have the capacity to penetrate and damage nearby healthy tissue, including organs (Weinberg, 1996). In 2019, there were 23.6 million new instances of cancer each year and 10 million people die worldwide, suggesting rises of 26% and 21% over the previous ten years, respectively (Kocarnik et al., 2022). According to estimates, there will be 1.9 million new cancer diagnoses and 609,360 cancer related deaths are observed in the United States in the time of 2022 (Beger et al., 2008). The growth of cancer registries around the globe has sparked an interest in discovering novel drugs that seem to be toxic against cancer cells but harmless to healthy cells. The anticancer medications that were traditionally used were relatively toxic to both normal body cells and tumor cells in the area of the body where the cancer had first appeared. Right now, both terrestrial plants and marine environments are being used in the search for new anticancer medications (Greenwell and Rahman, 2015). For generations, people have employed plants to treat illnesses. Many plants are consumed around the world for their health advantages as a form of traditional folk remedies. A wide range of anticancer drugs derived from plant materials are purified, and then they are tested in clinical trials on cells (including several cancer cells lines) and experimental animals (Greenwell and Rahman, 2015). In very recent time, the number of recently discovered natural substances has increased dramatically. The use of plants as sources of highly biologically active materials has been around for centuries in traditional medicine (Fridlender et al., 2015). One way to obtain these substances is by extracting them from plant materials. An alternative approach is to use biotechnological tools to produce anticancer compounds derived from plants. Some of the naturally occurring substances found plants and aquatic animals that have antitumor properties include alkaloids, diterpenoquinone, diterpenes, purine-based compounds, peptides,l actonic sesquiterpene, cyclic depsipeptide, macrocyclic polyethers, proteins etc. (Lichota and Gwozdzinski, 2018). Additionally, there is a lot of potential in marine environments to find novel organisms that can help with cancer treatment and prevention. Late in the 19th century, marine first appeared. After 1980, the field of biotechnology emerged as one that gave the study of the oceans direction, focusing on uses like drug development (Newman and Cragg, 2016). There is growing interest in utilizing the diversity and complexity of marine natural product scaffolds due to their tremendous potential for rational drug discovery (Nobili et al., 2009). New anticancer medications are required due to the rise in the prevalence of various types of cancer (Lichota and Gwozdzinski, 2018). This study's objective was to identify compounds with anti-cancer properties that were derived from plant and marine sources.

2. Materials and methods

A search was conducted (till May 2022) in the following databases: PubMed, Science Direct, MedLine, and Google Scholar using the keywords 'plant derivatives' and 'anticancer activity/effect'. There were no language restrictions. The articles were reviewed for information on plant derivatives, marine source, cancer pathophysiology, anticancer activities, test results, and potential mechanisms of action.

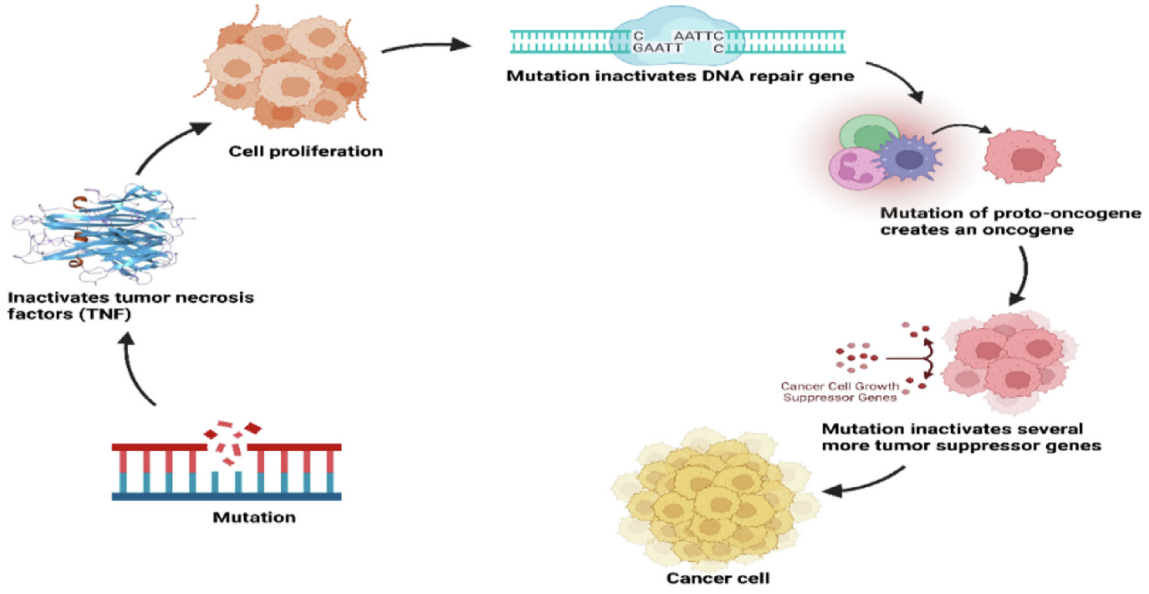


Fig. 1. Mutations play a role in the development of cancer. Every mutation modifies how a cell behaves.

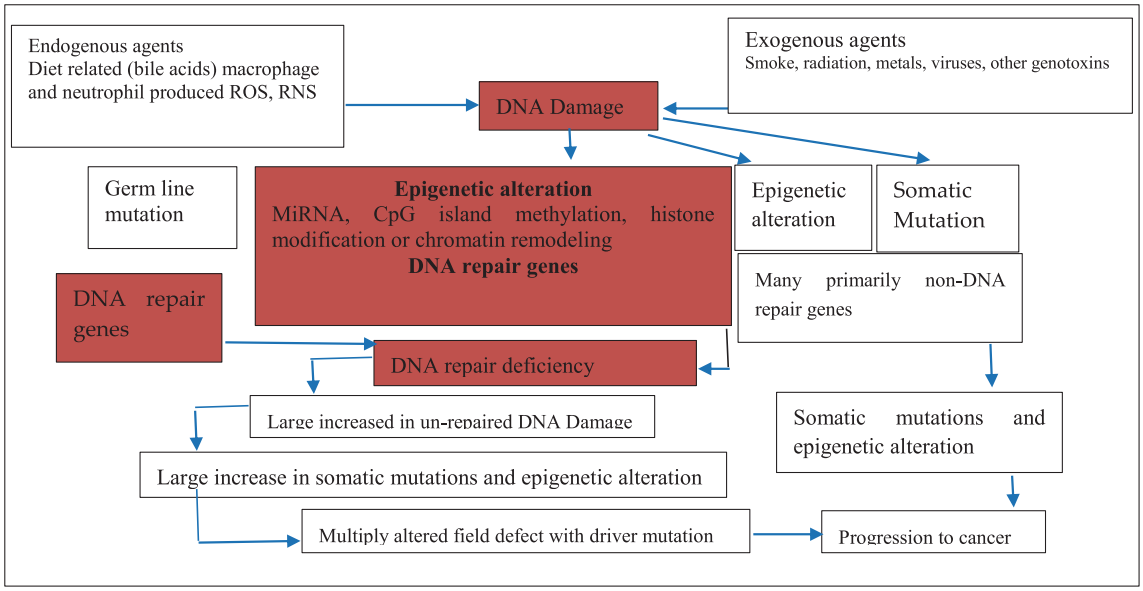


Fig. 2. The primary significance of DNA damage and epigenetic changes in DNA repair genes in the development of cancer.

3. Results

3.1. Cancer pathophysiology

Cancer is well-known disease that are occurred by the regulation of tissue growth. A normal cell must change its genes to become a cancer cell, which regulates cell development and differentiation. Genetic alterations can take place at a variety of different scales, from the addition or deletion of whole chromosomes to a single DNA nucleotide mutation. These modifications have an impact on two large types of genes. Oncogenes can be either normal genes that are overexpressed or mutated genes that exhibit unique features. In either instance, the expression of these genes promotes cancer cell malignancy. Tumor suppressing genes are those that impede cancer cell division, survival, or other qualities. Tumor suppressing genes are frequently silenced by cancer-promoting genetic mutations. The way of the development of cancer cells are displayed in Fig. 1.

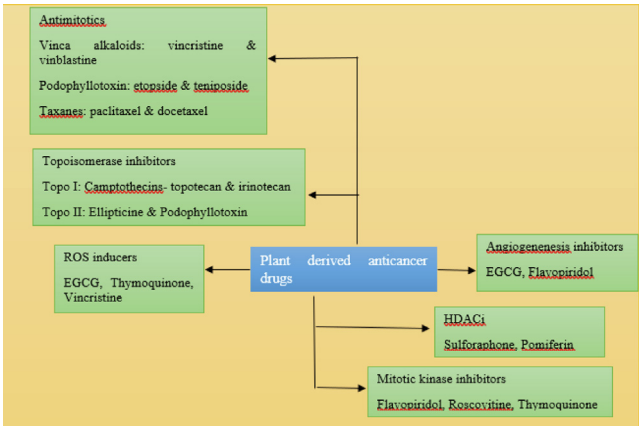


Fig. 3. Plant-based anticancer medicines in specific groupings. Some medicines can provide therapeutic and/or chemoprotective actions via various routes. EGCG is well-known for its anti-ROS effect; it may also suppress DNA methylation and angiogenesis. Thymoquinone is both a ROS inducer and a mitotic kinase inhibitor.

The traditional understanding of cancer is that it is a collection of diseases caused by progressive genetic abnormalities such as tumor-suppressor gene mutations, oncogene mutations, and chromosomal abnormalities (Baylin and Ohm, 2006). Epigenetic alterations are those that affect the genome in a way that is relevant to function but do not alter the nucleotide sequence. Changes in DNA methylation (hypermethylation and hypomethylation), histone modification, and chromosomal layout are only a few examples of such modifications (arise through the negative protein expression like HMGA2 or HMGA1) (Kanwal and Gupta, 2012). While epigenetic abnormalities are common in malignancies, epigenetic modifications in DNA repair genes, which result in lower production of DNA repair proteins, may be especially important. Such changes are expected to begin early in cancer growth and are a plausible cause of the genomic instability seen in malignancies (Bernstein et al., 2013). The main role of DNA damage and epigenetic modifications in DNA repair genes in the development of cancer is illustrated in Fig. 2.

3.2. Plant derived compounds

Plant-derived compounds have shown to be a rich source of different types of novel medicinal molecules applied against several type of human disease. Many anticancer drugs have been isolated from plants, including *Catharanthus roseus, Cuphea hyssopifolia, Podophyllum species, Coptis chinensis, Taxus brevifolia, Camptotheca acuminate, Betula alba, Streptococcus peucetius, Cephalotaxus species, Erythroxylum pervillei, Evodiae fructus, Curcuma longa, Ipomoeca batatas, Centaurea schischkinii, Eugenia jambos L., Alnus rubra, Punica granatum L, Phyllanthus niruri L., Hydrastis Canadensis, Sanguinaria canadensis, Stephania tertrandra* and others. Scientists are still investigating the bioavailability of anti-cancer substances in heretofore unrecognized plant species. Fig. 3 depicts various plant-derived anticancer medications and their main modes of action.

3.3. Marine source compounds

Based on the previous, numerous research organizations throughout the world have recently focused on the separation

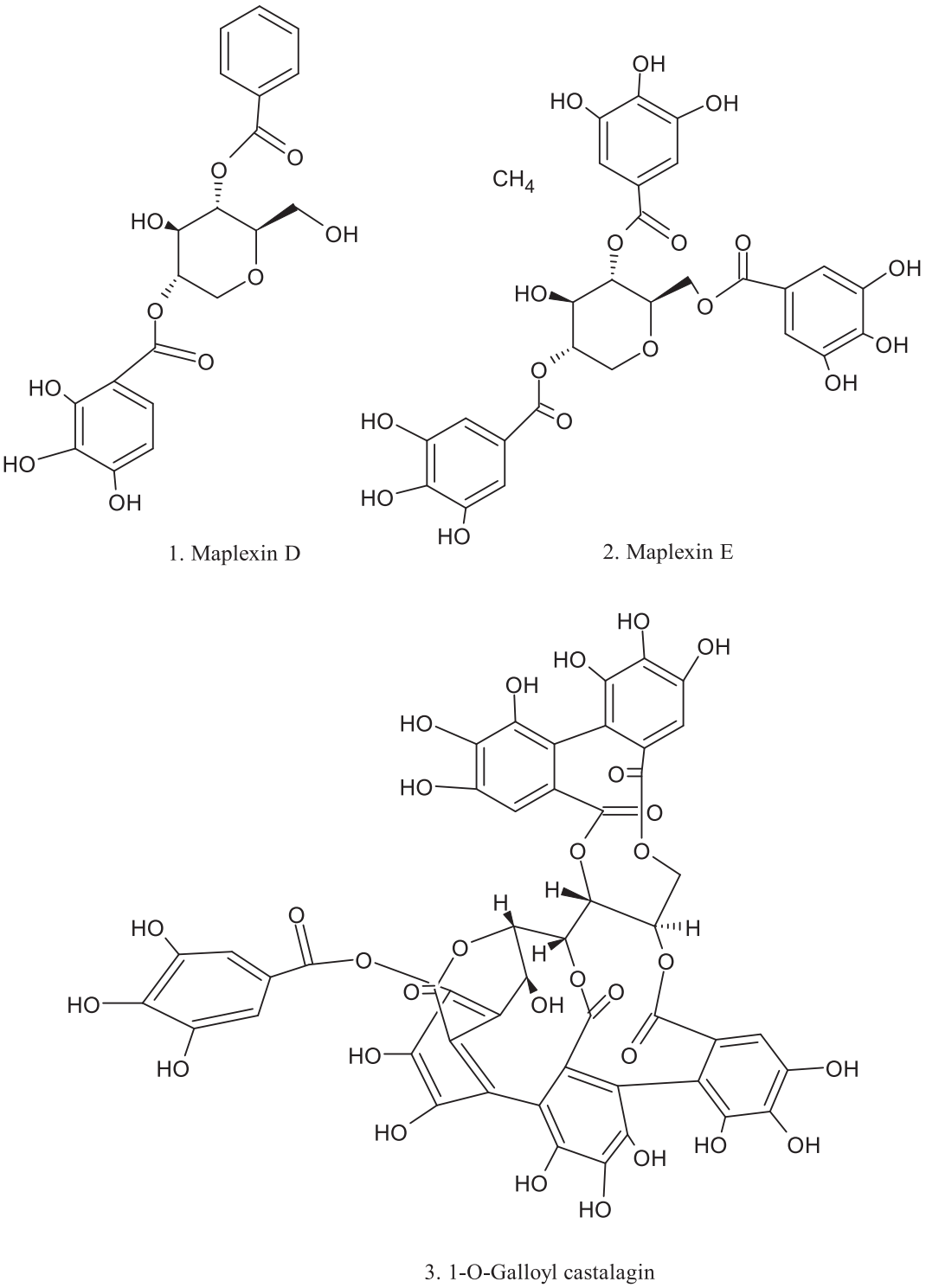
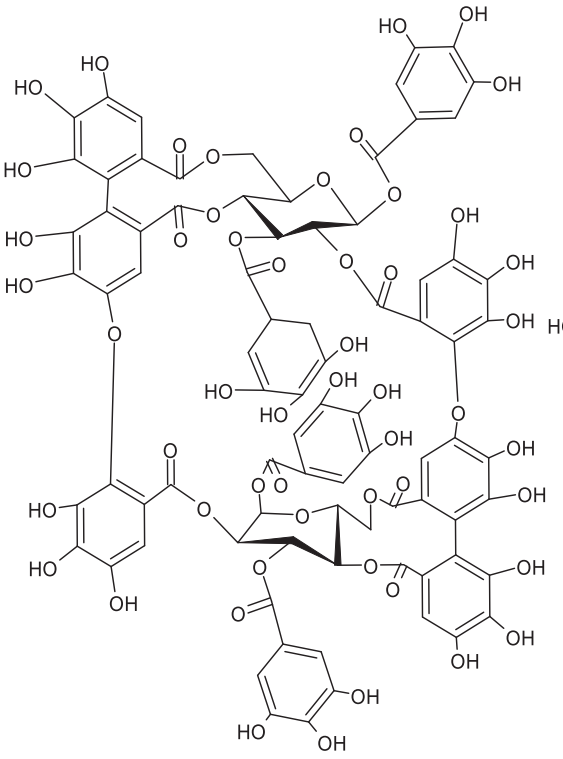
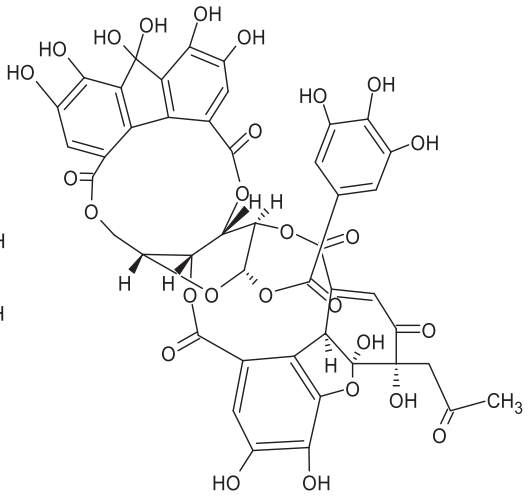


Fig. 4. Chemical structure of plant derived compounds.

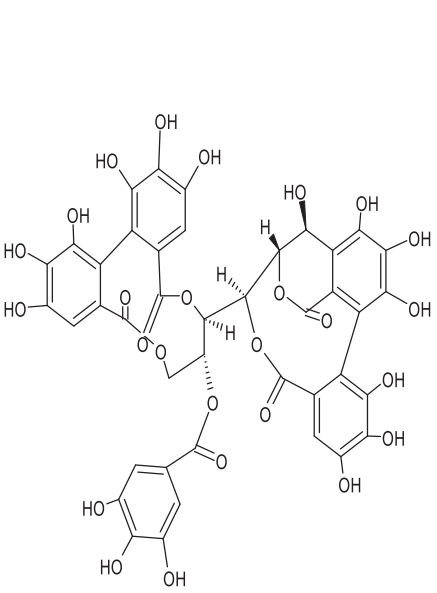
and characterization of biologically active components from marine source due to there several application (Fig. 5). The marine environment has developed into a significant source of molecules that have strong anticancer properties and display unusual chemical characteristics and mechanisms of action. Thirty-four of forty compounds in the pipeline for marine pharmaceuticals indicate "cancer therapy," and twelve of the seventeen marine-derived medications approved by regulatory bodies are used to treat cancer



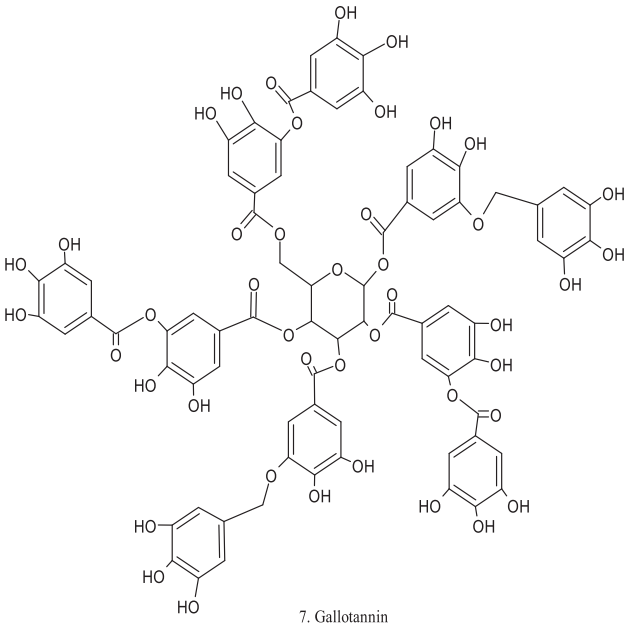
4. Cuphiin D1



5. Ellagitannin



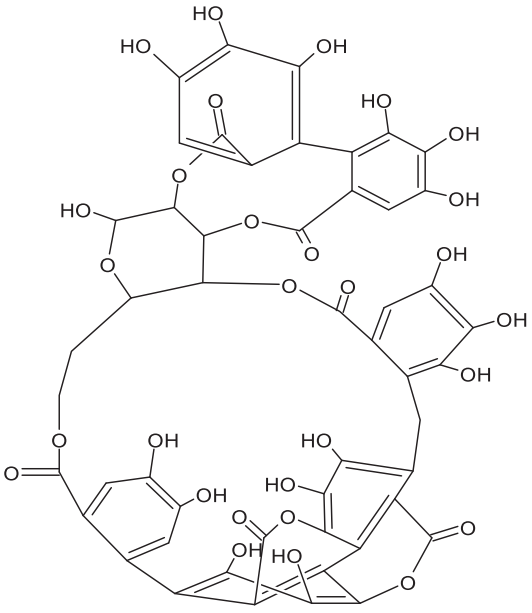
6. Casuarinin



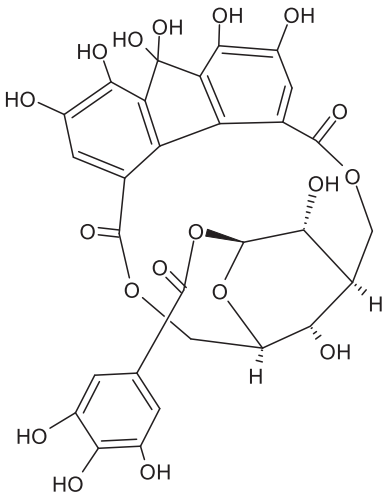
7. Gallotannin

Fig. 4 (*continued*)

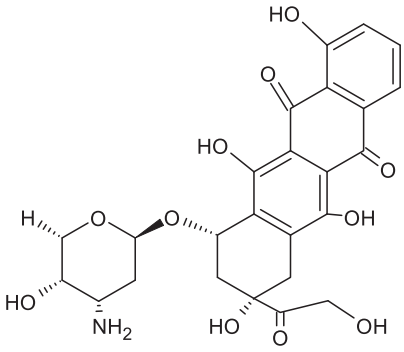
(Mayer et al., 2012). Sea is one of the most abundant habitats, teeming with variety of creatures, where their compounds are stand out because of their distinctive qualities. The development of cancer medicines derived from marine sources is extremely important in the fight against cancer. More than 60% of anti-tumor medications come from natural sources, including pharmaceuticals and compounds that are now being tested in clinical studies. This study is targeted to find out the anticancer activity of marine source compounds.



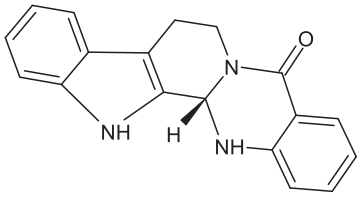
8. Punicalagin



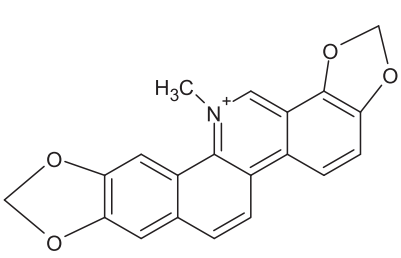
9. Corilagin



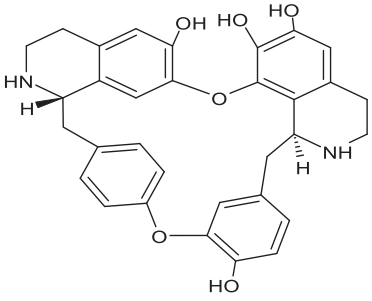
10. Doxorubicin



11. Evodiamine



12. Sanguinarine

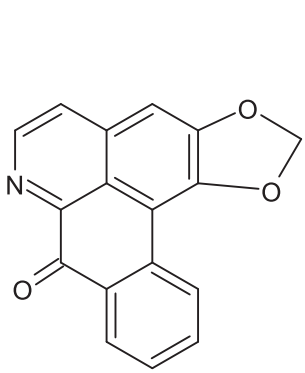


13. Tetrandrine

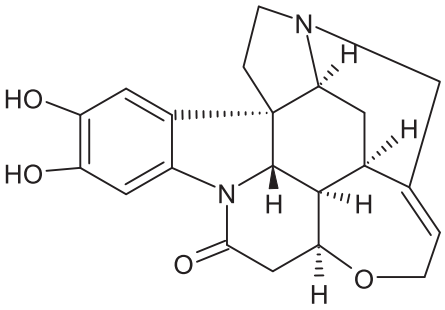
Fig. 4 (*continued*)

4. Discussion

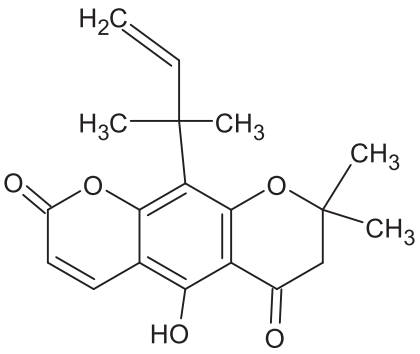
Several research has examined the anticancer potential of compounds derived from plants and marine source. Some of these substances demonstrate efficient anti-cancer activity in one or more cancer types. Based on their activities several compounds have been listed in Table 1/Fig. 4 and Table 2/Fig. 6. For biomedical uses, natural substances are effective therapeutic and chemopreventive agents as well as useful tools for evaluating molecular targets (Orlikova et al., 2014). Numerous studies have shown that phytochemicals found in natural products can prevent the initiation, promotion, and progression of carcinogenesis, and some of their medicinal compounds have the potential to be highly effective chemopreventive and chemotherapeutic approaches against can-



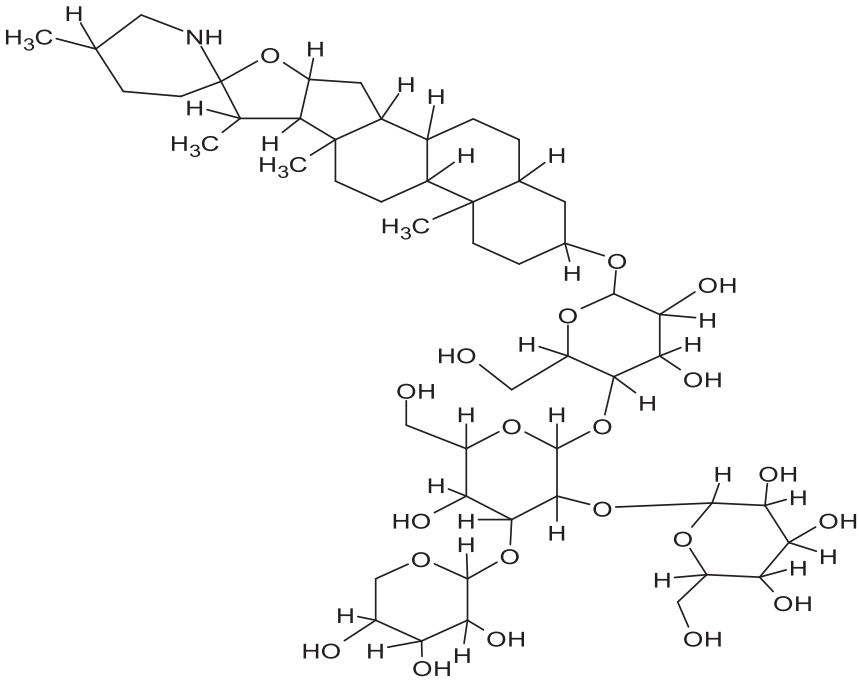
14. Liriodenine



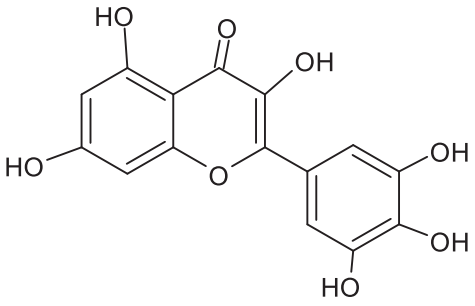
15. Brucine



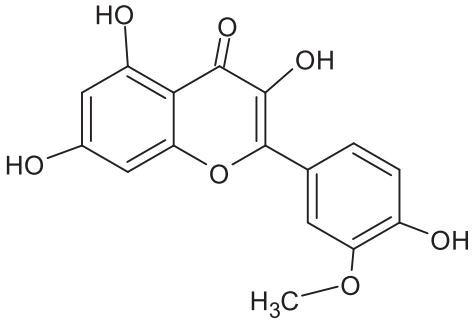
16. Clausenidin



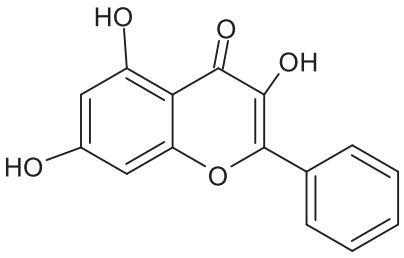
17. alpha-Tomatine



18. Myricetin



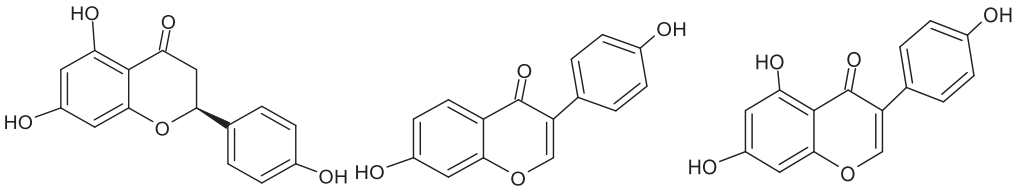
19. Isorhamnetin



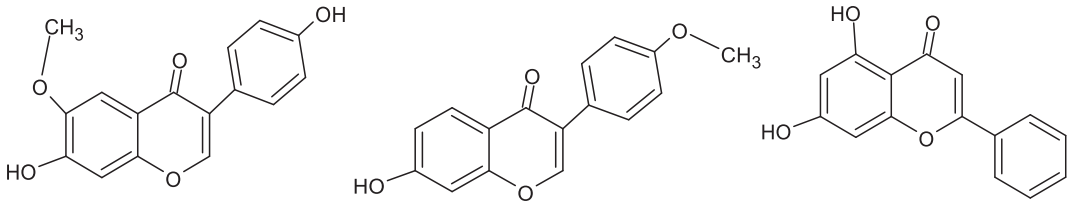
20. Galangin

Fig. 4 (*continued*)

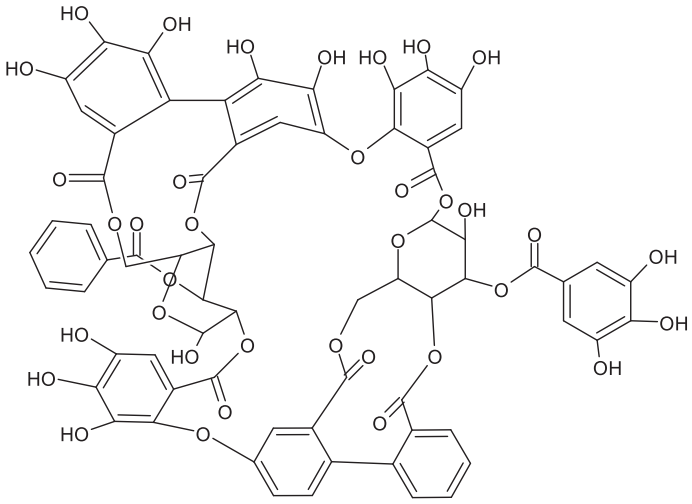
cer (Gupta et al., 2010). Plants produce a large number of bioactive metabolites, and because of their therapeutic benefits, they are highly sought-after in the field of pharmacology. They play a crucial role in the formation of sophisticated traditional medicine particularly that used to treat cancer diseases (Moghadamtousi et al., 2013). However, marine floras, which make up over 90% of the ocean's biomass, include bacteria, actinobacteria, cyanobacteria, fungus, microalgae, seaweeds, mangroves, and other halophytes. They provide a lot of opportunity for the development of novel anticancer medicines (Sithranga Boopathy and Kathiresan, 2010).



21. Naringenin 22. Daidzein 23. Genistein



24. Glycitein 25. Formononetin 26. Chrysin



27. Oenothein B

Fig. 4 (*continued*)

Numerous substances derived from plants have cytotoxic properties with a wide range of mechanisms of action, including DNA damage, the inhibition of topoisomerases I and II, the induction of apoptosis, and the inhibition of tumor cell growth. Studies have demonstrated that plant-derived compounds combined with chemotherapy drugs have a significant potential to kill tumor cells without harming healthy cells like lymphocytes and fibroblasts (Lichota and Gwozdzinski, 2018). Marine-derived bioactive molecules have been found to be effective against a variety of tumor cells, including those that cause bone, blood, lung, mammary, melanoma, prostate, bladder, and renal cancers in addition to the recognized mechanisms of action mediated by necrosis, apoptosis, and lysis of tumor cells.

5. Conclusions

It has been found that a number of plant and marine natural products have anticancer action in vitro on a variety of tumor cell lines, including those originating from kidney, lung, prostate, bladder, melanoma, osteosarcoma, breast, and lymphoid malignancies.

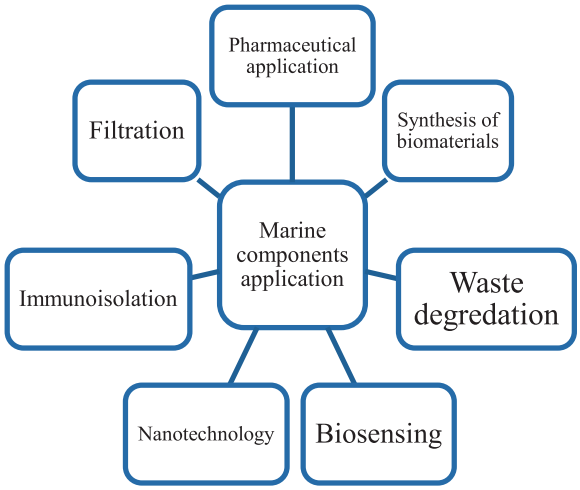


Fig. 5. Several applications of marine source components.

Table 1
Shortly structured anticancer activity of plant derived compounds.

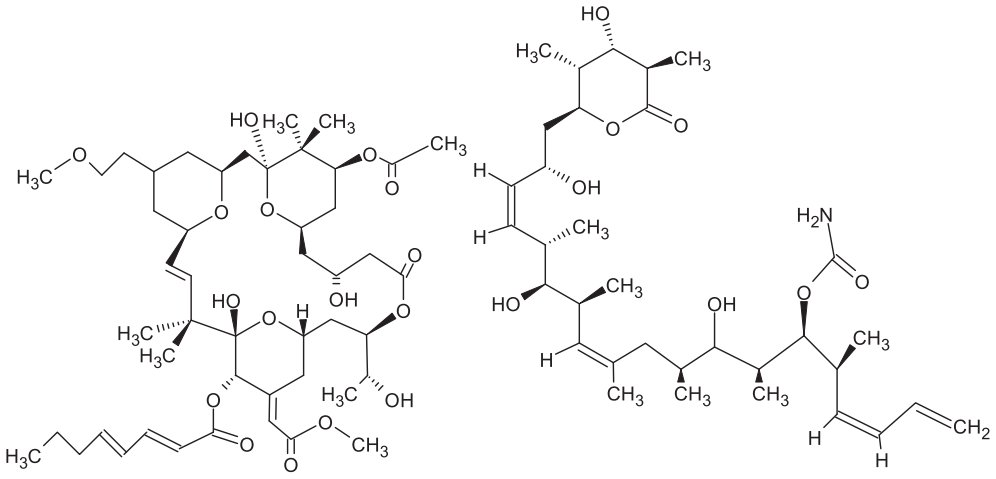
Compounds	Plant Source	Test Medium	Dose/Concentration	Mechanism of action	References
Maplexins C-D and Maplexins E-1	*A. rubrum L.*	HCT-116 and MCF-7 cells	IC50 = 59.8–67.9 and 95.5–108.5 μM vs 73.7–165.2 and 115.5–182.5 μM	inhibit cancer cell growth	(González-Sarrías et al., 2012)
Cuphiin D1	*Cuphea hyssopifolia*	HL-60 cells	IC50 = 16 μM	decrease cell population and inhibit Bcl-2 expression	(Wang et al., 2000)
Punicalagin (PUNI) and Ellagic acid (EA)	Pomegranate	Caco-2 and CCD-112CoN cells	PUNI 1; 10; 100 μM/l, EA 1; 10; 30 μM/l	apoptosis induction	(Larrosa et al., 2006)
1-O-galloyl castalagin and casuarinin	*Eugenia jambos L.*	HL-60	10.8 and 12.5 μM	induced apoptosis	(Yang et al., 2000)
Gallotannin	*Alnus rubra*	Colon cancer cells from humans (T-84)	10 μg/mL	Induced apoptosis	(Gali-Muhtasib et al., 2001)
Corilagin	*Phyllanthus niruri L.*	ovarian cancer cells	160 μM	increased apoptosis	(Jia et al., 2013)
Doxorubicin (DXR) + Tannic acid (TA)	–	MDA-MB-231 cells	DXR (2.5 mg/Kg, once weekly), TA (10 mg/Kg)	shows maximum tumor volume reduction	(Tikoo et al., 2011)
Oenothein B, woodfordin C and woodfordin D	–	Human squamous cell carcinoma (HSC-2) and salivary gland tumor (HSG)	CC_{50} = 0.060 μM CC_{50} = 0.026 μM CC_{50} = 0.026 μM	Accelerated apoptosis	(Sakagami et al., 2000)
8-cetylberberine	*Coptis chinensis and Hydrastis Canadensis*	A549 and MRC-5 cells	In vivo: 10 mg/Kg In vitro: 2 μg/mL	inhibit tumor growth decreased the survival rate	(Xiao et al., 2018)
Evodiamine	*Evodiae fructus*	MCF-7 and MDA-MB-231cells	–	prevents cells proliferating	(Wang et al., 2013)
Sanguinarine	*Sanguinaria canadensis*	HeLa and Siha human cervical cancer cells	2.43 μM/L (IC50) in HeLa cells and 3.07 μM/L in SiHa cells	induction of apoptosis	(Xu et al., 2012)
Tetrandrine (TET)	*Stephania tertrandra*	143B cells	1, 2 and 4 μM	inhibits the proliferation	(Tian et al., 2017)
Liriodenine	*natural plant species*	A549	20 μM and 50 μM	suppressed proliferation	(Chang et al., 2004)
Brucine	*Strychnos nux-vomica L.*	MDA-MB-231	1–2 mM	apoptosis induction	(Xu et al., 2019)
Cathachunine	*Catharanthus roseus*	human leukemia cells		anti-proliferation and pro-apoptosis abilities	(Wang et al., 2016)
Clausenidin	*Clausena excavata Burm. f*	HepG2	30, 40 and 50 μg/mL	induces apoptosis	(Waziri et al., 2016)
α-tomatine	*Solanum lycopersicum*	CT-26 colon cancer cells	at 3.5 μM	increased caspase-independent apoptosis	(Kim et al., 2015)
Myricetin	*berries, herbs and walnuts*	HCT-15 cells	50 and 100 μM	induces apoptosis	(Kim et al., 2014)
Isorhamnetin	*Hippophae rhamnoides L*		0,10,20,40 and 80 μM /L	reduced cell proliferation	(Li et al., 2014)
Baicalein	*Scutellaria baicalensis*	cell lung cancer (NSCLS)	0.5% CMC-Na solution, 40 mg/Kg	inhibits tumor growth	(Zhao et al., 2019)
Naringenin	*Fruits*	A549 cell	0–300 μM	alteration cell proliferation	(Chang et al., 2017)
Daidzein	*in nuts, fruits, soybeans, andsoy-based products*	JAR and JEG-3	100 μM	induce apoptosis	(Zheng et al., 2018)
Genistein (GEN)	*soy isoflavones*	HT-29 cells	200 μM /L	induces apoptosi	(Zhou et al., 2017)
Glycitein	Soybean	SKBR-3 cells	10, 30, 60, 100 mg/mL	damaged the cell membranes	(Zhang et al., 2015)
Formononetin	*Pongamia pinnata, Astragalus membranaceus, Ononis angustissima and Trifolium pratense*	FaDu cell	50 μM	decelerated tumor growth	(Oh et al., 2020)
Chrysin	–	CT26 cells	80 μg/mL	induction of apoptosis	(Bahadori et al., 2016)
Galangin	*Alpinia galangal*	*MCF-7 and T47D*	20 μM	inducing apoptosis	(Song et al., 2017)

Table 2
Shortly narrate the anticancer activity of compounds found from marine source.

Class	Natural compound	Chemistry	Test system	Test dose/ concentration	Proposed mechanism	Reference
			In Vitro1.Renca renal adenocarcinoma, 2. B16 melanoma3. M5076 reticulum cell sarcoma, the L10A B-cell lymphoma	100 ng/mL	Antiproliferative responses against cancer cell	
Marine Bacteria	Bryostatins	Macrolide	In vivo1. mice bearing 8–10-mm s.c. masses of L10A lymphoma ($5–10 \times 10^9$) 2. Six human B-cell lymphoma cell lines	1 μg/injection/day	B-cell lymphoma growth inhibition	(Hornung et al., 1992)
	Taxol/ discodermolide	–	SKOV-3	25 mg/kg i.p. and 5 mg/kg i.v.	induces tumor regressions	(Huang et al., 2006)
	Cryptophycins	Depsipeptide	Murine *in vivo* xenograft models mice model	0.1 mL/10 g body weight of the animals	active antitumor agents against the rat 13,762 mammary carcinoma	(Menon et al., 2000)
	Indanone from *Lyngbya majuscula*	Polyketide	Human hepatocellular carcinoma cell line. Hep3B human liver tumor cells	–	VEGF expression inhibition	(Nagle et al., 2000)
	Lyngbyabellin A (*Lyngbya majuscula*)	Desipeptide	Human nasopharyngeal and colon carcinoma cell line	1.0.03 Ìg/mL2.0.50 Ìg/mL	Disruption of cellular microfilaments	(Luesch et al., 2000)
	Apratoxin A from *Lyngbya boulloni*	Polyketide	Cervical cancerCell line (HeLa)	2.2 nM	Blocking the progression of G1 phase → Cell cycle inhibition → Cytotoxicity	(Ma et al., 2006)
Marine Corals	Cembrane (*Alcyonacea, Nephtheidae*)	–	Three cancer cell linesSF-268 (CNS), MCF-7 (breast), and H460 (lung)	100 μM	Three primary tumor cell lines were exposed to non-selective anticancer activities	(Januar et al., 2010)
	Eleutherobin analogues	Diterpene glycoside	Human breast carcinoma cell line	1–100000 nM	–	(Cinel et al., 2000)
	Sterols	Steroids	Dalton's lymphoma ascites cells (DLA)	10 μg/mL, 20 μg/mL, 50 μg/mL, 100 μg/mL, and 200 μg/mL	exhibited remarkable apoptosis agonist activity	(Byju et al., 2014)
Marine Algae	Sterol fraction (cholesterol, β-sitosterol, and campesterol)	–	4 T1 cell	10 and 25 mg/Kg	induced apoptosis	Kazłowska et al., 2013)
	Fucoidan from *Sargassum mcclurei*		DLD-1 cells	1–200 μg/mL	colony formation inhibition	(Duc Thinh et al., 2013)
	Dioxinodehydroeckol Isolated from *Ecklonia Cava*	Phloroglucinol derivatives	MCF-7 and MDA-MB-231 human breast cancer cell line	1, 5, 10, 50 and 100 μM	inhibit the proliferation	(Kong et al., 2009)
	Elatol isolated from algae *Laurencia microcladia.*	Sesquiterpene	Western blot analysis, C57Bl6 mice bearing B16F10 cells	0.1–100 μM	induces apoptosis	(Campos et al., 2012)
	Fucoxanthin	Carotenoids	CMT-U27	10, and 20 μM	induced apoptosis	(Jang et al., 2021)
	Sargassum oligocystum extract	–	In-vitro testK562 and Daudi human cancer cell lines	0–500 μg/mL. Most effective concentration 500 μg/mL and 400 μg/mL	Inhibited G0/G1 stage SGC-7901 from entering to S stage	(Ji et al., 2004)
	Violaxanthin from *Dunaliella tertiolecta*	–	Breast adenocarcinoma (MCF-7)	40 μg/mL (to observe cytostatic activity)	Cancer cell proliferation is inhibited→ ↑Apoptosis	(Pasquet et al., 2011)
	Phloroglucinol from Brown seaweed	–	Colorectal cancer Cell lines (HCT116 & HT29)	300 μM	Induce DNA damage → Cytotoxicity→ ↓ Cell death	Lopes-Costa et al., 2017)
		–	Human leukemia (HL-60) cells	μM	↑Caspase 3 & 7 → ↓Bcl-2→ ↑Apoptosis → Cytotoxicity	Ganesan et al., 2011)

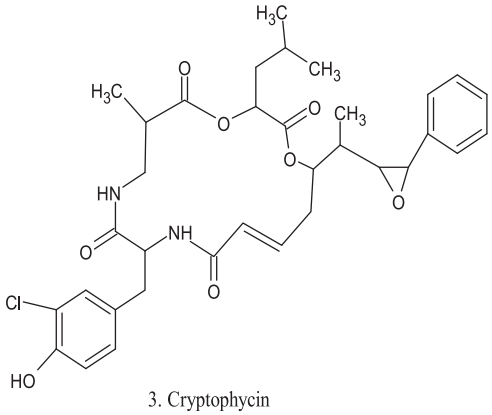
Table 2 (*continued*)

Class	Natural compound	Chemistry	Test system	Test dose/ concentration	Proposed mechanism	Reference
Marine Tunicate	Didemnin B	Depsipeptide	Rabbit reticulocyte lysate and human adenocarcinoma cell line	–		(Ahuja et al., 2000)
			10 patients	5.6 mg/m^2	competitive inhibition enzyme	(Benvenuto et al., 1992)
		Alkaloid	Human and murine leukemia cell lines	μM	Apoptosis induction; no impact on topoisomerases I and II	Dassonneville et al., 2000)
			Human colon carcinoma cell line	10–50 nM	Inhibition of transcription of the human P glycoprotein gene (MDR1)	Jin et al., 2000)
	Trabectedin (ET-743) isolated from *Ecteinascidia turbinata*	Alkylating agent	52 patients with solid tumors (mostly colorectalcancers and sarcomas)	0.05–1.8 mg/m^2	impact on a number of transcriptional regulators, cell proliferation, and the nucleotide excision repair system	(Ganjoo and Patel, 2009)
Clam	Spisulosine			––10 μM		Cuadros et al., 2000)
		–	Colon and breast, cancers cell lines	–		Vasko et al., 2010)
Sponge	Fascaplysin	Alkaloid	Cell lines from human colon cancer, osteogenic sarcoma, and normal fibroblasts	0.35 μM	Inhibition of Cyclindependent Kinase 4	(Soni et al., 2000)
	Aragusterol A	Steroid	Human and murine cancer cell panel and *in vivo* assays	0.01–1.6 μM	$_1$/S cell cycle phase	(Fukuoka et al., 2000)
	Discodermolide	Polyketide	Human and murine tumor cell lines	0–1000 nM	stabilize microtubules and inhibit cells	(Martello et al., 2000)
Sea squirts	Ecteinascidin/ Trabectedin from *Ecteinascidia turbinata*	Alkaloids	A549 cell	0.6 ng/mL ng/mL	Cytotoxicity against tumour cell line in vitro.	(Ghielmini et al., 1998)
Diatom	Monoacylglycerides	–	Haematological cancer cell line (U-937)	μg/mL	↑caspase3/7 activation→	(Miceli et al., 2019)
	(MAGs) from *Skeletonema marinoi*	-	Colon cancer cell line (HCT-116) MePR-2B normal cells		↑Apoptosis → Cytotoxic activity	
	Polyunsaturated aldehydes (PUAs2-trans,4-*trans*-decadienal(DD)) from *Skeletonema marinoi*	–	A549 cells Colon adenocarcinoma metastaticascites-deriving (COLO205) Normal lung/brunch epithelial (BEAS-2B)	2,5 & 10 μM	↑Apoptosis → Cytotoxic effect→↑ on cell death	(Sansone et al., 2014)
	Polyunsaturated aldehydes (PUAs) from *Thalassiosira rotula, Skeletonema costatum, Pseudo-nitzschia delicatissima*	–	Colon adenocarcinoma (Caco-2) cells	(11 ± 17) μg/mL	Arrest cell proliferation→↑Apoptosis	(Miralto et al., 1999)
	Chrysolaminaran from *Synedra acus*	–	Human colon cancer cells (HT-29) Colon cell line (DLD-1)	54.5 μg/mL 47.7 μg/mL	Inhibition of cancer cell proliferation → Cytotoxic activity	(Kusaikin et al., 2010)
	Nonyl 8-acetoxy-6-methyloctanoate (NAMO, fatty alcohol ester) from *Phaeodactylum tricornutum*	–	Human promyelocytic leukemia (HL-60) Human lung carcinoma (A549) Mouse melanoma (B16F10)	22.3 μg/mL 50 μg/mL –	Cell cycle arrest sub-G1 phase→ ↓damage DNA →↑Apoptosis → Cytotoxicity –	(Samarakoon et al., 2014)
	Monogalactosyl diacylglycerols from *Phaeodactylum tricornutum*	–	Wild-type W2 Wild-type D3	64 μM 1 μM	↑Caspase 3/7 → ↑Apoptosis → Cytotoxicity	(Andrianasolo et al., 2008)
	Fucoxanthin from *Phaeodactylum tricornutum*	Xanthophyll	Caco-2 (derived from a human colon adenocarcinoma),HepG2, and HeLa (derived from cervical cancer cells)	1 μM	↑Caspase 3/7 → ↑Apoptosis → Cytotoxicity	(Neumann et al., 2019)
	from *Navicula incerta*	Phytosterol	Liver hepatocellular carcinoma (HepG2)	8.25 μg/mL	↑caspase-8, 9 →↓damage DNA → ↑Apoptosis → Cytotoxicity	(Kim et al., 2014)

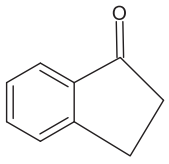


1. Bryostatin

2. Discodermolide

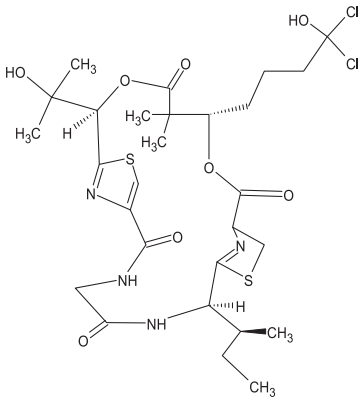


3. Cryptophycin

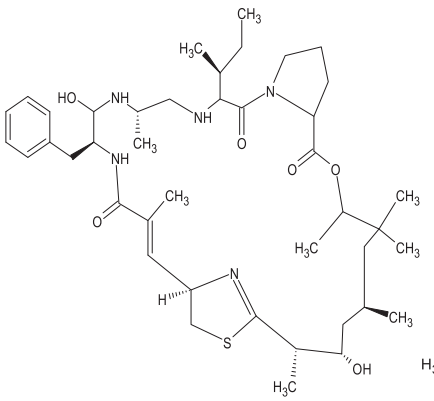


4. 1-Indanone

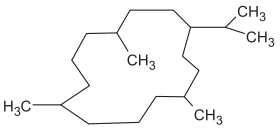
Fig. 6. Chemical structure of marine source compounds.



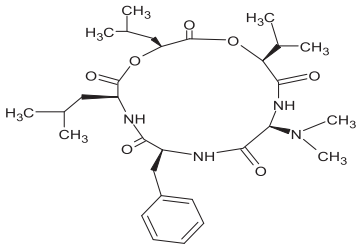
5. Lyngbyabellin A



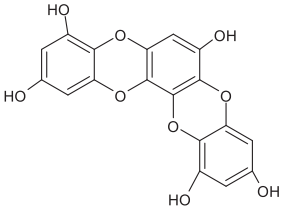
6. Apratoxin A



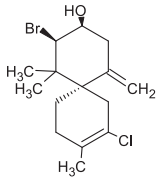
7. Cembrane



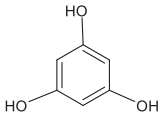
8. Sansalvamide



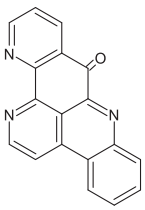
9. Dioxinodehydroeckol



10. Elatol



11. Phloroglucinol



12, Ascididemin

Fig. 6 (*continued*)

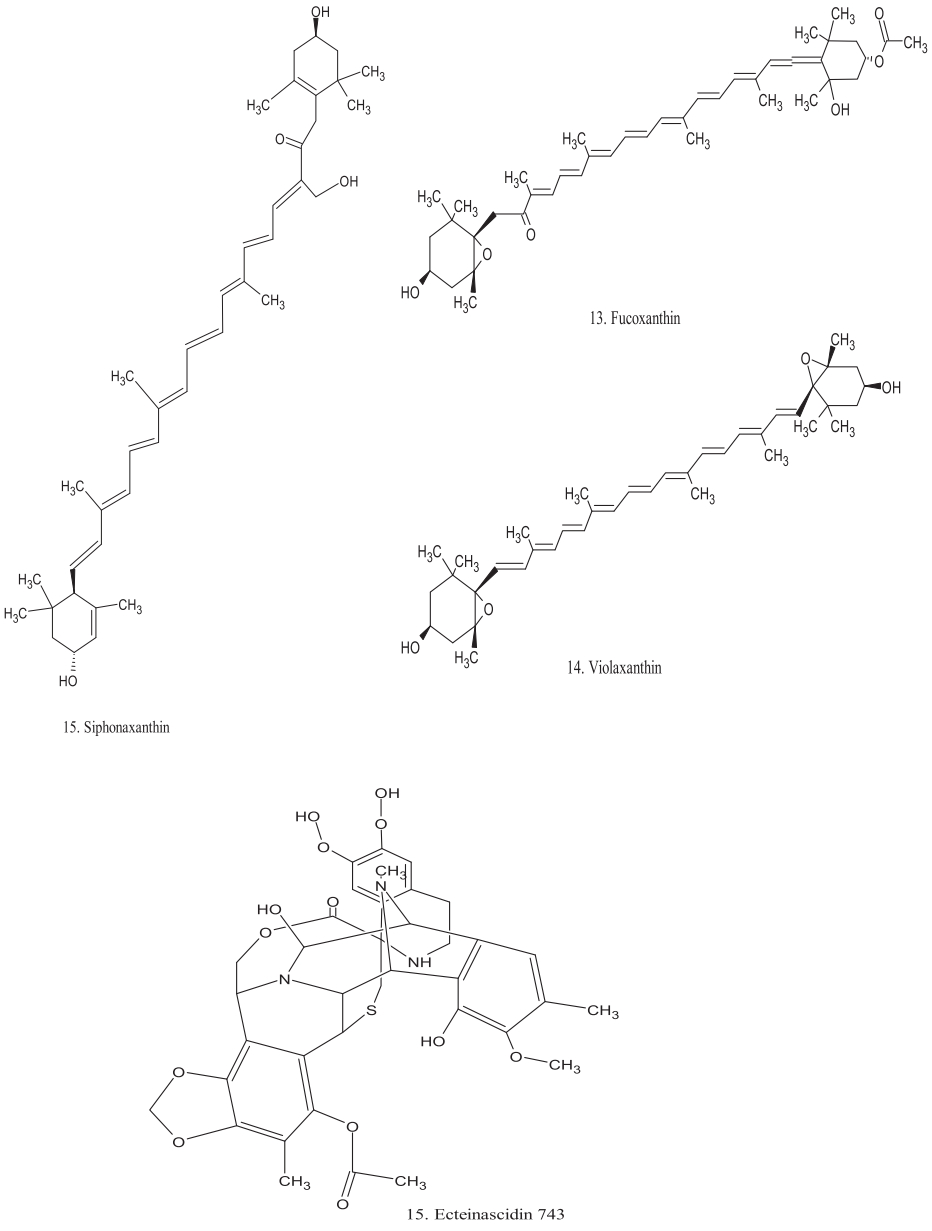


Fig. 6 (*continued*)

Furthermore, the majority of data on just how plant as well as marine products inhibit tumorigenesis both in vitro and *in vivo* point to the possibility that this is accomplished by inducing apoptosis, necrosis, and lysis in the tumor cells. WHO estimates that more than 80% of people in underdeveloped nations rely on traditional medicines for their most basic medical requirements. A healthy diet rich in fruits and vegetables can help stave against the progression of cancer. As chemoprotective medicines against different forms of cancer, several natural compounds are available. Fruits, vegetables, extracts from plants, herbs, microorganisms, and marine life all contain these chemoprotective compounds. The preventive effect against cancer may be attributed to a variety of natural product ingredients. In this work, we attempted to examine the anticancer properties of a number of organic compounds that were isolated from plant and marine sources.

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Data availability statement

The data will be available after request to the corresponding authors.

CRediT authorship contribution statement

Md. Mizanur Rahaman: Conceptualization. **Polrat Wilairatana:** Concepualization, Project Administration. **Mehedi Hasan Bappi:** Methodology. **Tawhida Islam:** Methodology. **Md. Nayem**

Mia: Software. **Henrique Douglas Melo Coutinho:** Project administration. **Abolghasem Siyadatpanah:** Validation. **Muhammad Torequl Islam:** Conceptualization, Supervision.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jksus.2023.102919.

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