

Understanding Cancer for non-technical pupils

Norliyana Amran

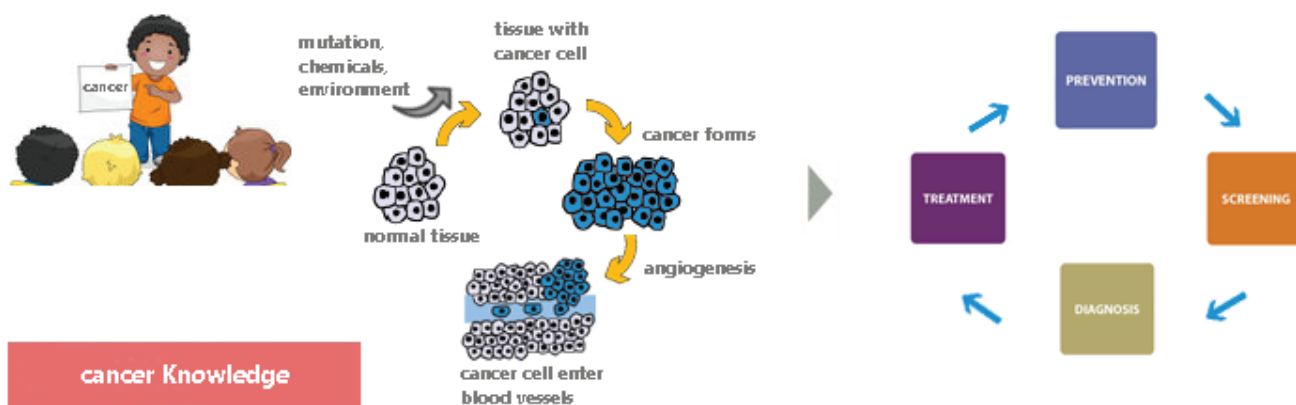
EMAN Research and Testing Laboratory, Department of Pharmaceutical Chemistry, School of Pharmaceutical Sciences, Universiti Sains Malaysia, Penang, Malaysia

Please cite this article:

Norliyana Amran. (2017). Understanding Cancer for non-technical pupils , 1(1), pages 039-040.

Significance | a brief summary on cancer and its treatment will provide a realistic insightful knowledge for non-technical persons.

Graphical Abstract



*Correspondence: EMAN Research and Testing Laboratory, Department of Pharmacology, School of Pharmaceutical Sciences, Universiti Sains Malaysia, Penang, Malaysia. E-mail: liyana_amran@yahoo.com



Understanding Cancer for non-technical pupils

Norliyana Amran

Cancer is reported to have affected every fourth citizen of a developed country sometime during his or her lifetime. Approximately 400 new incidents emerge per 100,000 people annually (Parkin et al., 2005). In 2008, it was estimated that there were 12.7 million cancer cases and 7.6 million deaths (within 5 years of diagnosis) globally (Ferley et al., 2010). Further, this number will continue to increase year by year. But the question remains, what is cancer? There are many definitions of cancer and the easiest way to explain it is uncontrollable proliferation of cell. Cancer develops following a number of genetic mutations that cause functional alterations in oncogenes, tumor-suppressor genes and microRNA genes. It can affect any part of the body by means of uncontrolled and abnormal cell development (Vanhoecke et al., 2005).

Cancers have been treated in a few ways using conventional methods such as surgery, radiotherapy and chemotherapy (Kintzios & Barberaki, 2004). Surgery is performed if the cancerous tissues are still localized and have yet to metastasize to other parts of the body. However, this method usually develops scarring after surgery and may injure healthy tissues and organs adjacent to the cancerous tissues. In addition to that, some remains of neoplastic tissues will be present upon post-surgery (Kintzios & Barberaki, 2004). Radiation therapy is done by using ionizing wave that are directed to the cancerous cells. The damaging wave causes DNA fragmentation and subsequent apoptotic death of the cancerous cells. However, radiation treatment often leads to treatment failure due to the presence of radio-resistant cancer cells (Kintzios & Barberaki, 2004; Pesenti et al., 1992). Also, chemotherapy is a well-known cancer treatment that uses cytotoxic agents to either damage the DNA of cancerous cells or

disrupt the metabolic pathways that are essential for cell survival. Despite the advancement of medical research and technology, the complete cure for cancer remains elusive. Current cancer therapies, as explained previously, can bring about undesired physical and psychological distress to the patients. Therefore, continuing global efforts in the search for novel anticancer compounds that possess high therapeutic efficacy and less adverse reactions compared to the existing anti-cancer drugs in the market are necessary.

An ideal anticancer agent is expected to inhibit, delay or reverse the progression of cancer through its cytotoxicity and/or apoptosis-inducing properties (Naveen Kumar et al., 2012). The discovery and development of anticancer drugs, especially cytotoxic agents, differ significantly from the drug development process given for any other diseases. Identification of cytotoxic compounds has led the development of anticancer therapeutics for several decades. Cytotoxic drugs are primarily used as anticancer drugs because they are poisonous (toxic) to cancer cells and they kill or stop them from multiplying. These types of drugs have an effect of preventing the rapid growth and division of cancer cells (Narang and Desai, 2009).

Future efforts in the design of new therapies to improve survival and the quality of life of cancer patients must include strategies that specifically target cancer cells that are resistant to current chemo/radiotherapies. Defective apoptosis is one of the major factors in the development and progression of cancer. The ability of uncontrolled cells to override apoptosis plays an important role in their resistance to conventional therapeutic anticancer agents (Kasibhatla and Tseng, 2003). Apoptosis becomes a target mechanism in recent therapeutic strategies, since it involves tumor-selective killing without compromising the function of a normal cell. Apoptosis is just one form of cell death. Cells may also be eli-

Significance | a brief summary on cancer and its treatment will provide a realistic insightful knowledge for non-technical persons.

*Correspondence: EMAN Research and Testing Laboratory, Department of Pharmacology, School of Pharmaceutical Sciences, Universiti Sains Malaysia, Penang, Malaysia.
E-mail: liyana_amran@yahoo.com

Edited by Md Shamsuddin Sultan Khan, Hawkesbury Institute for the Environment, University of Western Sydney, Hawkesbury Campus, Bourke Street, Richmond, NSW AUSTRALIA 2753 and accepted by the Editorial Board May 8, 2017 (received for Sep 11, 2016)

Author Affiliation:

EMAN Research and Testing Laboratory, Department of Pharmaceutical Chemistry, School of Pharmaceutical Sciences, Universiti Sains Malaysia, Penang, Malaysia

Please cite this article:

Norliyana Amran. (2017). Understanding Cancer for non-technical pupils, 1(1), pages 039-040.

minated by a number of alternative mechanisms, including necrosis. However, when cells die from necrosis, this causes inflammation of the surrounding tissue, which can further distress or cause injury within the body. This critical difference between apoptosis and necrosis is the reason why apoptosis is the most desirable target mechanism for the induction of cell death in cancer cells and is being widely focused upon in up-to-date cancer-related studies.

Angiogenesis is the process by which new blood vessels form and it is a key factor of the spreading and growing of cancer. Anti-angiogenesis is being used as a target mechanism for the induction of cell death because of its cytotoxic and apoptosis-inducing properties. Phytochemicals were a first antiangiogenic agent to be isolated. In 1990, Ingber et al. reported on the anti-angiogenic properties of *Aspergillus fumigatus*-derived fumagillin. Since Folkman (1971) hypothesized that tumors are angiogenesis dependent, extensive research and development on angiogenesis are being carried out globally. To date, the combination of angiogenesis inhibitors in early chemotherapy treatment has shown a decline in the growth of cancerous cell (Browder et al, 2000).

Various species of medicinal plants have been reported to possess anti-cancer activities. In fact approximately 74% of anti-cancer drugs developed today originated from medicinal plants (Lopes et al., 2009). Secondary metabolites synthesized by plants play a major role in drug development against cancer. Secondary metabolites such as aldehydes, alkaloids, flavonoids, glycosides, lignans, lipids, nucleic acids, phenols, polysaccharides, proteins and terpenoids have been shown to be capable of stimulating the inhibition of tumor development directly (Kintzios & Barberaki, 2004). Herbal components and drug compounds can change the rate of activation or detoxification of carcinogens by changing the activities of detoxifying metabolic enzymes. Again, the balance between metabolic detoxification and activation reactions would depend on the chemical structure of the herbal components or the drug.

Acknowledgment

The author would like to express his gratitude to his editorial fellows.

Author Contribution

Norliyana A. made substantial contributions to the conception of the review.

Competing financial interests

The author(s) declare no competing financial interests.

References

- Browder T, Butterfield CE, Kraling BM, Marshall B, O'Reilly MS, Folkman J. (2000). Antiangiogenic scheduling of chemotherapy improves efficacy against experimental drug-resistant cancer. *Cancer Res.*, 60, 1878–1886
- Ferlay, J., Shin, H.R., Bray, F., Forman, D., Mathers C. and Parkin, D.M. (2010). Estimates of worldwide burden of cancer in 2008: GLOBOCAN 2008. *International*

Journal of Cancer, 127, 2893-2917.

Folkman, J. (1971). Tumor angiogenesis. Therapeutic implications. *N Engl J Med*, 285, 1182–1186

Ingber D, Fujita T, Kishimoto S, Sudo K, Kanamaru T, Brem H, Folkman J. (1990). Synthetic analogues of fumagillin that inhibit angiogenesis and suppress tumour growth. *Nature*, 348(6301), 555–557.

Kasibhatla, S. and Tseng, B. (2003). Why Target Apoptosis in Cancer Treatment? *Molecular Cancer Therapeutics*, 2, 573–580.

Kintzios, S.E. & Barberaki, M.G. (2004). *Plants that fight cancer*, CRC Press, Boca Raton.

Lopes, F., Rocha, A., Pirraco, A., Regasini, L., Silva, D., Bolzani, V., Azevedo, I., Carlos, I. and Soares, R. (2009). Anti-angiogenic effects of pterogynidine alkaloid isolated from *Alchornea glandulosa*. *BMC Complementary and Alternative Medicine*, 9(1), 1-11.

Naveen Kumar, D.R., Shikha, S., Cijo George, V., Suresh, P.K. and Ashok Kumar, R. (2012). Anticancer and anti-metastatic activities of *Rheum emodi* rhizome chloroform extracts. *Asian Journal of Pharmaceutical and Clinical Research*, 3, 189–194.

Narang, A.S. and Desai, D.S. (2009). Anticancer drug development. In *Pharmaceutical Perspectives of Cancer Therapeutics*, Springer: Berlin, German, pp. 49–92.

Parkin, D.M., Bray, F., Ferlay, J. and Pisani, P. (2005). Global cancer statistics, 2002. *CA: A Cancer Journal for Clinicians*, 55(2), 74-108.

Pesenti, E., Sola, F., Mongelli, N., Grandi, M. and Spreafico, F. (1992). Suramin prevents neovascularisation and tumour growth through blocking of basic fibroblast growth factor activity. *British Journal of Cancer*, 66, 367–372.

Vanhoecke, B.W., Depypere, H.T., De Beyter, A., Sharma, S.K., Parmar, V.S., De Keukeleire, D. and Bracke, M.E., (2005). New anti-invasive compounds: Results from the Indo-Belgian screening program. *Pure and Applied Chemistry*, 77(1) 65-74.

Submit your next manuscript to Angiotherapy published by EMAN Research.

- Convenient online submission
- Thorough peer review
- No space constraints or color figure charges
- Immediate publication on acceptance
- Inclusion in Australian National Library and Google Scholar
- Both Open (80-100% subsidized APC by ER) & non-open access option

Submit your manuscript at
angiotherapy.emanresearch.org

eman Research