

Cancer.

Inside:

On Treating Cancer: A
Journey Of Reflection

Untangling the Genetic
Mysteries of Cancer Using
Computational Tools

Cancer's Little Helpers:
cells within the tumour
microenvironment
and their roles in
tumourigenesis and
metastasis

Simulacrum

ON THE COVER



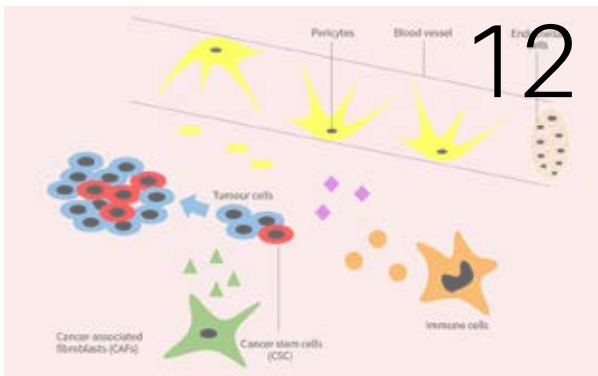
On Treating Cancer: A Journey Of Reflection

By Dr. Tan Chin Joo



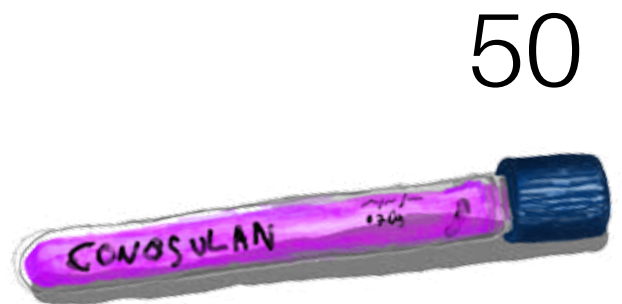
Untangling the Genetic Mysteries of Cancer Using Computational Tools

by Dr. Camelia Quek and Dr. Kelly Quek



Cancer's Little Helpers: cells within the tumour microenvironment and their roles in tumourigenesis and metastasis

By Ng Chong Lee and Dr. Oon Chern Ein



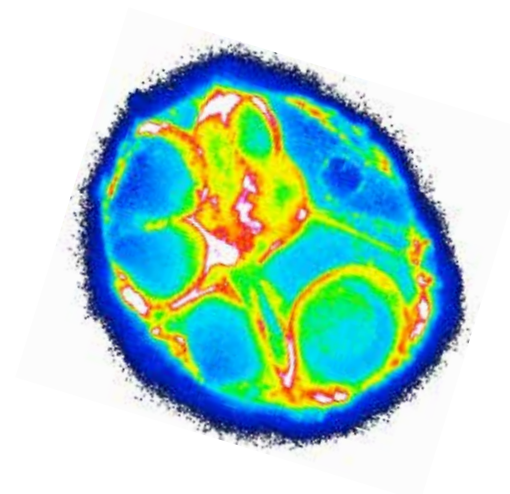
Simulacrum

by Lee Ee Leen

The cover photo depicts aggregated spheres of colorectal cancer cells that were artificially grown and cultured in the laboratory. These cancer cells are unable to self-organise into a proper tissue-like structure and are routinely used in research for drug testing and genetic manipulation. The images were captured using a laser-scanning microscope aimed at the mid-section of the sphere, and was then coloured for visualisation.

Contributed by:

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Welcome to the 14th issue of Scientific Malaysian!

EDITOR'S FOREWORD

The theme for this issue is Cancer—a disease so familiar, it hardly needs an introduction. While we have previously published several articles on cancer (The use of immunotherapy in cancer treatment by Litt-Yee Hiew and the potential of viruses to cure cancer by Suet Lin Chia in Issue 12; The challenges of personalised cancer detection by Dr. Chang Yang Yew in Issue 8), the disease is so multi-faceted and prevalent in our society, we felt it deserved a whole issue dedicated to it! Therefore, in this edition we bring you a series of articles that discuss the topic of cancer from different angles. We are also trying out a new columnar format to help you, readers, and potential writers, navigate our magazine.

When faced with such a tragedy as cancer, it's sometimes hard to think beyond ourselves. In our opening article in Interviews & Anecdotes, Dr. Tan Chin Joo shares her experiences on treating cancer patients (pg 1). Next, we have Lee Yeuan Ting and Dr. Oon Chern Ein introduce precision medicine as an approach which is widely believed to be the future of cancer therapy in our introductory column, In Brief (pg 3). In the next column, Tip of the Iceberg, we include two articles that discuss the underlying complexities of cancer, with first Dr. Teow Sin-Yeang stressing how a cancer is more than just its tumour (pg 8), and Ng Chong Lee and Dr. Oon Chern Ein delving further to describe the roles of various components within the tumour microenvironment in cancer progression and treatment (pg 12).

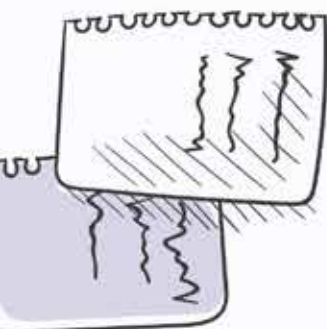
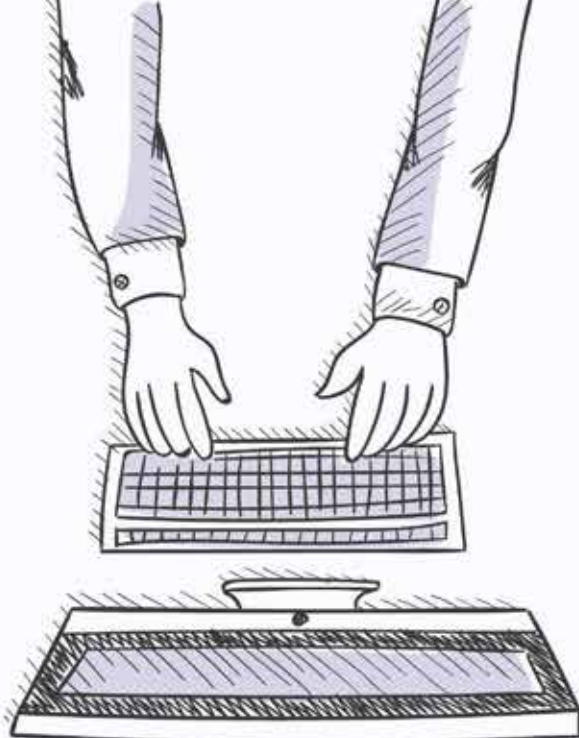
Then, dear reader, if you're left wondering when it all got so complicated, check out our Stats & Facts column on the historical timeline of cancer written by our own editorial team (pg 19). We follow on this momentum with an article in Cutting Edge by Dr. Camelia Quek and Dr. Kelly Quek on how tools developed through computational biology and bioinformatics may help us to characterise cancers and determine the best treatment strategies to be employed for each individual (pg 27). Next, we explore the issues between alternate and conventional treatments for cancer in an article by Enakshi Sivasudhan in Critical Thinking (pg 36).

In our second Interviews & Anecdotes article, Jennifer Eyahmalay offers us an anecdote to illustrate how easy it can be to develop bad habits that may put one at risk for developing cancer (pg 44). Finally, for a bit of light reading in Inkwell, Joshua Teh reviews the movie Me and Earl and the Dying Girl (pg 47), and Lee Ee Leen takes us into a dystopic future where cancer therapeutics are traded on the black market in Kuala Lumpur (pg 50).

As always, feel free to reach out to us at magazine@scientificmalaysian.com to express your thoughts on this issue or to suggest a theme for us to focus on in the future. We seek to inform and also to entertain, so any feedback or comments on our articles, or on the magazine in general are very welcome.

Thank you for your support in downloading and reading our magazine and I hope you enjoy this issue!

Juliana Ariffin
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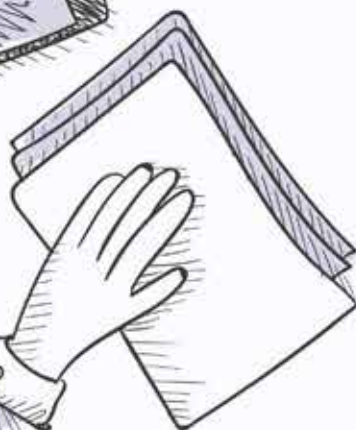


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On Treating Cancer: A Journey of Reflection

By Dr. Tan Chin Joo

When people ask me what kind of doctor I am, or in which discipline I practice, I simply answer “Oncology”. Those who understand the term often appear sympathetic. To others who stare at me blankly, I add “Cancer, chemotherapy, radiotherapy, pain control, best supportive care... that’s me”. Most of them are in awe and yet, will never be able to comprehend my work or how I can treat cancer patients, as it is a depressing and debilitating disease.

Cancer patients come in various forms. Some are well-aware of their diagnosis, and are keen to discuss treatments and disease prognoses in good faith. Some are emotionally labile, fearful and too upset to even begin any form of discussion at all. Then there are the sceptical sort—either the patients themselves or their accompanying family members would quote findings from journals, internet searches or even unverified opinions of non-medical experts, and expect doctors like me to try

harder to convince them to undergo treatment even though they are already leaning towards alternative therapy, which is not my area of expertise. In my years of practice, I often think I have seen it all but still I sometimes encounter new and unexpected scenarios when interacting with my patients.

Many years ago, I was trying to console a distraught patient with breast cancer that had fortunately been detected at an early stage, when she blatantly said, “How can you understand when you don’t have cancer?” Such a reaction was surely food for thought. Nevertheless, I referred her to a few cancer support groups and put her in contact with a cancer survivor who was working very closely with us. The next time I met her in an outpatient clinic, she appeared cheerful and had already completed her treatment. She continued to volunteer in one of the support groups to help others to overcome their negativity.

As doctors, we try to offer the best available treatment options to our patients. We aim to achieve a complete cure and to reduce the risk of recurrence and progression. If these approaches fail, we ensure comfort and offer symptomatic treatment to them until the end of their lives, with dignity and respect.

It isn’t always a losing battle when it comes to fighting cancer. We have many survivors who are in remission and under surveillance with follow up treatments. They lead normal lives post-treatment and are able to return to their daily routine, with most of them making extra effort to stay health-conscious. One





thing they all have in common is an increased appreciation for life and its value.

I have learned much from their journeys—I have witnessed raw and unrestrained emotions, the will to live, the bond of family and friends, and even the kindness of strangers. I have treated patients who were humble and grateful to be alive; as well as those who were rude and bitter at everyone and everything. Some were desperate for an extension of their time in this world, while an equal number showed signs of giving up the fight. Thanks to their experiences, I am constantly reminded that I may be a doctor, but I too, may fall sick one day. Should that day come, I know what kind of patient I want to be. 🌈

About the Author

Dr. Tan Chin Joo graduated from Moscow Medical Academy, Russia. She works in Penang General Hospital, up north of Peninsular Malaysia. She is the sub-investigator for several Phase 2/3 clinical trials in cooperation with her peers. Apart from her daily practice, she volunteers most of her free time at the dog shelter and is devoted to helping stray animals. She loves to travel and hopes to visit all the Wonders of the World in her lifetime.



Honing in on Cancer with Precision Medicine

At a Glance

Cancer is a disease characterised by the abnormal growth of cells that serve no purpose and which can disrupt normal bodily functions, leading to the destruction of normal healthy tissue and ultimately, death. Many options are currently available for cancer treatment including surgery, radiotherapy and chemotherapy. However, doctors and researchers are beginning to realise that not all patients respond or even benefit from these treatments. Thus, with the increasing wealth of genomic knowledge and the advancement of technology, scientists are beginning to explore new strategies to improve the diagnosis and treatment of cancer. At the forefront of these is a new approach called precision medicine, which is surfacing as the new future of cancer therapy. This article provides a brief overview on what precision medicine involves.

By Lee Yeuan Ting &
Dr. Oon Chern Ein*

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How Does Cancer Develop?

Cancer develops when one or more cells undergoes DNA damage that fails to be detected by the body. The unrepaired DNA damage may cause gene mutation that leads to malfunction in cellular activities affecting cell growth, division and death. In normal cells, cell

proliferation and cell death are controlled by genes that either favour or stop the cell division. However, mutation resulting from DNA damage allows the cancerous cells to gain survival advantage through increased proliferation by rendering the cells to disregard

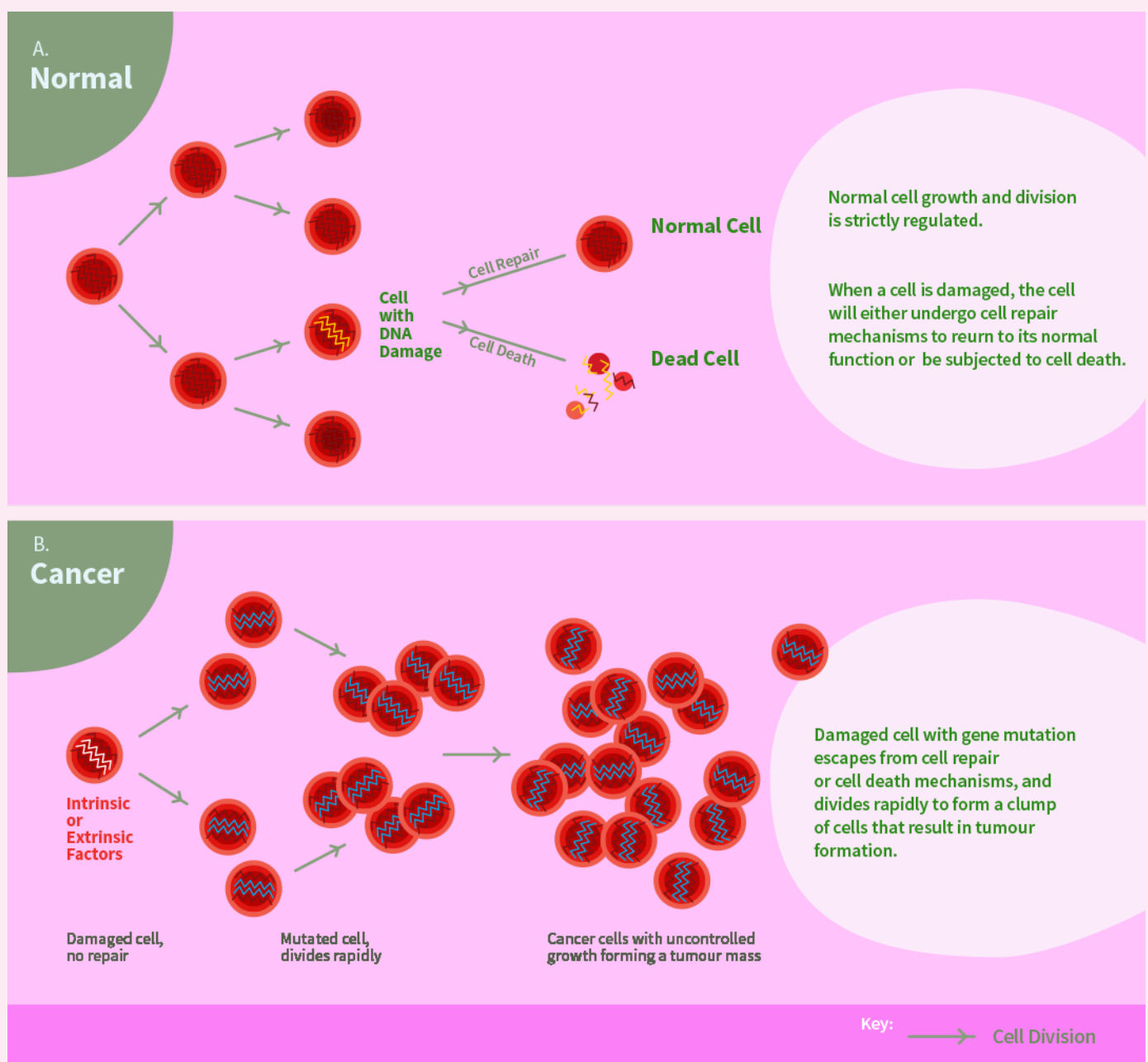


Figure 1: Cellular events associated with DNA damage in normal and cancerous cells. A) Cellular repair mechanisms in normal cells are highly regulated by the body to correct genetic abnormalities while B) cancerous cells are flawed in their repair mechanisms, and thus divide uncontrollably, leading to tumour formation.

signals that halt cell division, and by triggering the cells' ability to produce growth factors which promote cell survival, or both (Figure 1).

DNA damage can be caused by both intrinsic and extrinsic factors. Intrinsic factors refer to genes inherited from either parent that predisposes an individual to develop cancer, or a gene mutation that occurs spontaneously within the individual. In the case of inheritance, the probability for

an individual to develop cancer increases if there is a history of familial cancer. DNA damage can also be caused by extrinsic factors which encompasses environmental and individual lifestyle factors. According to the World Health Organisation (WHO), environmental factors such as ionising and non-ionising radiation, alcohol use, smoking tobacco and a sedentary lifestyle may all contribute to higher risk factors for developing cancer [1].

What is Precision Medicine?

Besides radiotherapy and surgery, chemotherapy is the most common strategy for cancer treatment. It involves the use of cytotoxic or cytostatic drugs to kill cancerous cells. However chemotherapeutic drugs are known to also affect normal cells as they indiscriminately kill rapidly dividing cells such as cells in the nails, blood, stomach lining and hair. The chemotherapeutic agents are mostly cytotoxic, leading to unwanted side effects in patients. Common side effects include fatigue, nausea, vomiting, hair loss, mouth and throat sores, diarrhoea, blood disorders and appetite loss [2]. In addition, these drugs are currently prescribed as "one size fits all", and thus are less effective for some patients who may not respond to these treatments. As such, precision medicine holds a big promise in cancer treatment.

Precision medicine is an emerging

approach for disease treatment and prevention. It targets different patients based on their unique circumstances by taking into account their genetic profile and environment and lifestyle factors. The genetic variations in each individual may affect how a patient responds to a certain drug. Single nucleotide polymorphism (SNP) refers to a single base change in the genome. Some SNPs may increase the susceptibility of some individuals to a certain cancer type. Environmental factors and individual lifestyle have also been shown to influence gene expression and how an individual responds to treatments. SNPs are useful to predict an individual's respond to certain drugs, taking into account environmental factors, and to assess their risk of developing particular diseases [3, 4]. By combining the information yielded by an individual's SNPs with precision medicine, medical

treatments may be precisely tailored to ensure that the most effective drugs and suitable doses are administered for each patients.

Current advances in biotechnology and the completion of the Human Genome Project, which involved the mapping of all the genes in the human genome, have paved the way for a more effective application of precision medicine.

Occurrences of cancer are mostly attributed to severe irreversible gene mutations that may vary in different patients according to cancer type, stage or location. Precision medicine can make use of these mutations through sequencing and analysis of the genome in the tumour sample

obtained from each patient (Figure 2). This information will allow doctors to customize treatments for every patient according to the genetic profile of their tumour, and to prescribe drugs that specifically target cancerous cells without harming other non-cancerous cells in the body.

In short, precision medicine involves studying how an individual's health is influenced by the interaction between environmental factors and genetic variations. Through these information, healthcare providers can better predict a patient's response to the drugs. This allows the doctor to give the right treatment to the right patient for better treatment outcomes. 🧬

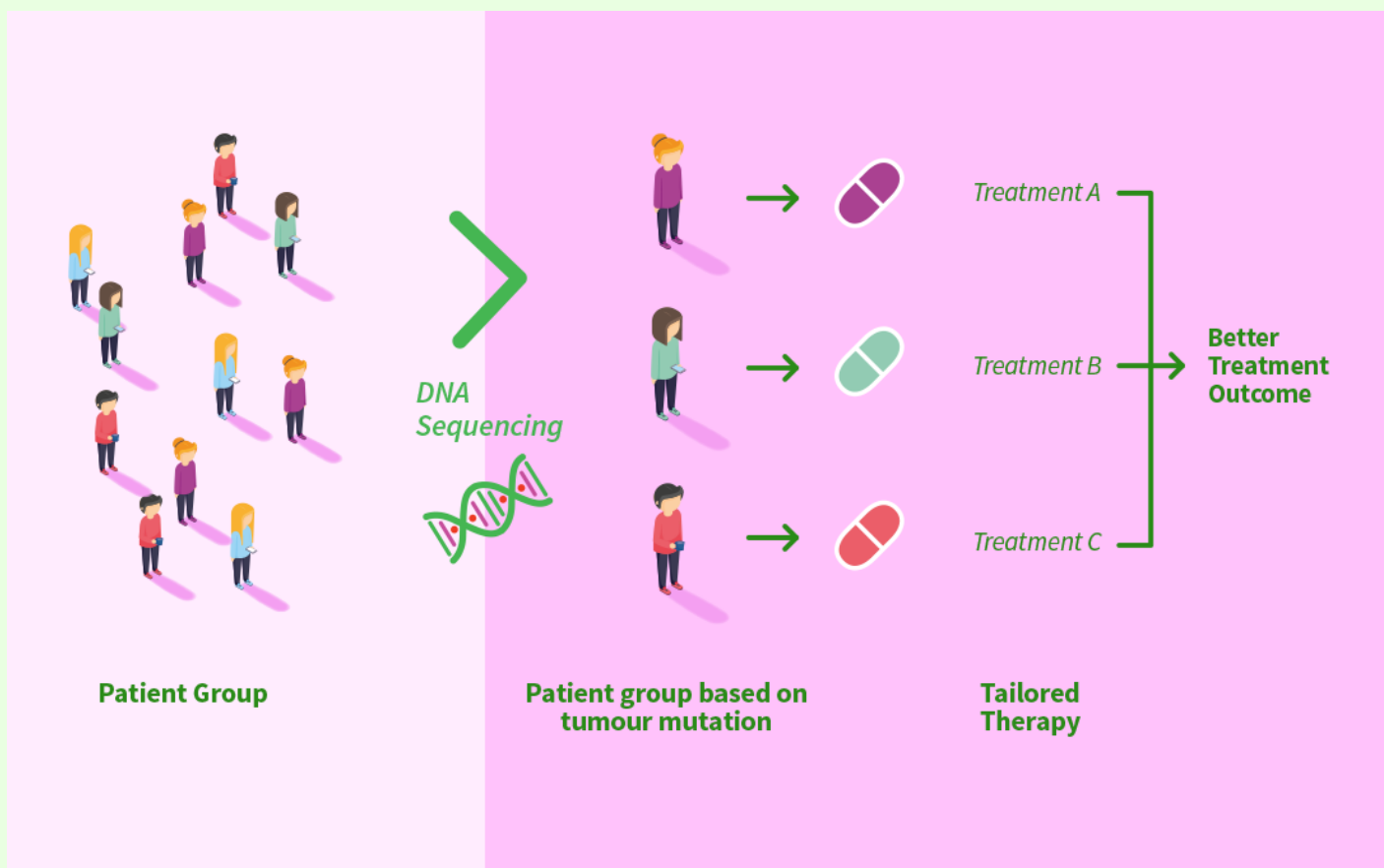


Figure 2: Schematic flow of precision medicine. Precision medicine allows patients to be treated by tailoring therapies according to their genetic make-up.

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Lee Yeuan Ting obtained her Bachelor of Science (1st Class Hons) in Molecular and Cell Biology at UPM and is now a MSc student at INFORMM, USM. Her masters research is about preclinical toxicology and mutagenicity study of a novel anti-cancer agent.

The Tumour and Its Surrounding 'Allies'

by Dr. Teow Sin-Yeang

At a Glance:

Cancer is one of the main killers in the world but it doesn't act alone. Recent advancements in cancer research have highlighted the role of non-cancerous cells surrounding the tumour (also known as the 'tumour microenvironment') that contribute to the progression of cancer. This has dramatically changed the classical approach of treating cancers. Targeting the 'tumour microenvironment' is likely to be more effective treatment than treating the

tumour cells alone. However, this is extremely challenging given that 'tumour microenvironments' vary from one cancer type to another, depending on genetics and environmental factors. This has given rise to precision therapy which is foreseen to be the future for cancer therapy. Due to the complexity of cancer, the advancement of technology will help in profiling the 'microenvironment' for personalised therapeutic intervention.

Cancer is the second leading cause of global death, with nearly 1 in 6 deaths caused by the disease [1].

The word 'cancer' is no stranger to us regardless of our educational background and level, mainly because of its high incidence rate and its notorious difficult-to-treat characteristics. According to the Malaysian National Cancer Registry Report (MNCR) 2007-2011, the most common cancer amongst Malaysians is breast cancer followed by colorectal and lung cancers [2].

From the first time we ask about what cancer is, we are 'meducated' that it is a disease caused by uncontrolled growth of a group of abnormal cells that results in a tissue mass known as a tumour. This statement is true for its appearance and general understanding, but is not enough in the context of treating it as a disease. Cancer is simply far more complicated to describe than as just 'a group of abnormal

cells'! This can be explained by the recurrence of cancer even after the complete removal of the tumour mass. It also explains why cancer is so difficult to treat.

Scientific research has helped us understand that there is more than one cell population involved in cancer. In most of the cancers, the tumour is often surrounded by multiple non-tumour cell types [3]. These cells (e.g. fibroblasts, tumour-infiltrating lymphocytes, cancer stem cells, and tumour-associated macrophages) closely interact with each other and are viewed as 'allies' or 'friends' to the tumour due to their tumour-promoting properties (Figure 1). A few examples of these cells and their normal and tumour-associated functions are illustrated in Table 1. The composition of these cells in the tumour varies depending on the disease staging and genetic factors.

In addition to these cells, non-cellular components such as extracellular matrix

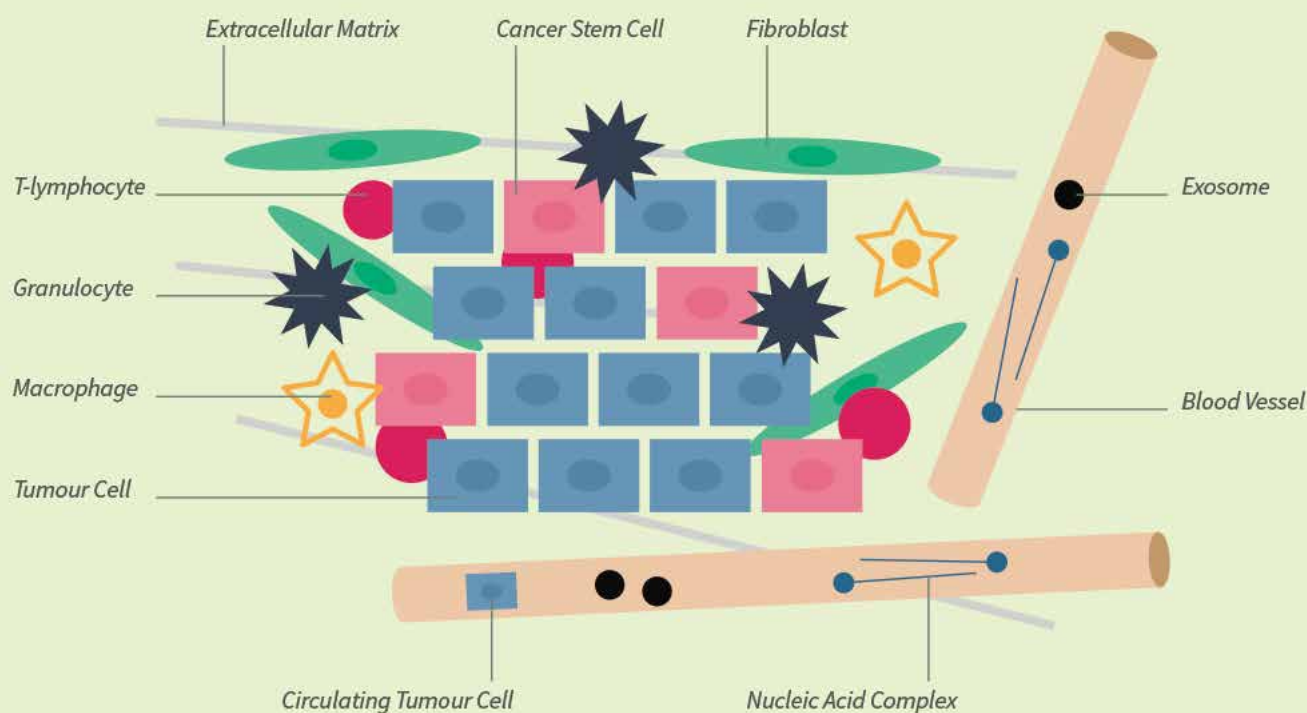


FIGURE 1: Cells such as fibroblasts, tumour-infiltrating lymphocytes, cancer stem cells, and tumour-associated macrophages are viewed as 'allies' or 'friends' to the tumour due to their tumour promoting properties.





Cell Type	Normal Function	Cancer-associated Function
Cancer associated fibroblasts 	Responsible for making extracellular matrix and collagen to maintain structural integrity within connective tissue and tissue repair during wound healing	Secretes growth factors that support tumour cell survival, proliferation and cancer progression (eg. metastasis)
Tumour-infiltrating lymphocytes 	Major component in immune system to specifically eliminate the antigen and provide memory for killing	Mediates response of cancer to therapy and often improve clinical outcome
Cancer stem cells 	Progenitor cells that develop and differentiate into more specialised cells	Contributes to tumour progression, cancer recurrence, metastasis and therapy resistance
Tumour-associated macrophages 	Clean unwanted particles by engulfing and destroying pathogens and apoptotic cells	Recruited to the tumour due to cancer-associated inflammation and they play roles in cancer progression and immune evasion

Table 1: The tumour associated functions of the tumour's 'allies'

(ECM), exosomes, nucleic acid complexes (DNA, messenger RNAs, and microRNAs) are also observed to be enriched in the tumour surroundings [3,4] [Figure 1]. These components play multifaceted and important roles in cell-cell interaction and communication, hence, contributing to the cancer pathogenesis.

Altogether, the cellular and non-cellular components collectively act as the 'tumour microenvironment' that supports tumour development and progression. In advanced stage cancers, the close contact between the tumour complexes and blood vessels further eases the transport of tumour cells (i.e. circulating tumour cells and cancer stem cells) or tumorigenic factors (i.e. exosomes and circulating miRNAs) to other sites or organs for the tumour to spread (termed as metastasis) [Figure 1].

So, now we know that cancer is not merely a


single group of uncontrolled growing cells, but that it includes many other types of cells and components. And yes, all these complexities contribute to difficulty and failure in treatment. For example, the surrounding cancer stem cells and some of the tumour-specific miRNA have been reported to contribute to chemo- and/or radio-therapy resistance [5]. As anticancer treatment strategies that specifically target tumour cells (e.g. targeting cancer antigen or cellular receptors) may no longer be effective, we may need an entirely new strategy which targets the tumour microenvironment instead (e.g. targeting a cancer-activating signalling pathway).

Furthermore, the composition of the tumour-associated cells varies depending on the cancer types and individuals with different immunological profile and genetic make-up. These further complicates the therapeutic strategies that may be used and this is where precision medicine or personalised therapy

comes into play [6]. Precision medicine is expected to reduce overall treatment time and cost. However, the complexity of the tumour microenvironment is always a challenging hurdle for precision medicine. The classical single-target cell-based approach is no longer useful in targeting a 'microenvironment' that constitutes of multiple cell types and other components.

To conclude, understanding the role of the 'tumour microenvironment' on cancer pathogenesis is only the tip of the iceberg. We are still far from 'curing' cancer. Continuous

efforts are required to decipher the exact molecular mechanisms involved in cancer pathogenesis in various stages, from disease establishment to progression.

Understanding these mechanisms will unquestionably improve current anti-cancer therapy. However, the future of cancer therapy will also require much more robust technology due to the complexity and variation of the disease. Hence, the advancement of technology must move forward along with our understanding of cancer. 

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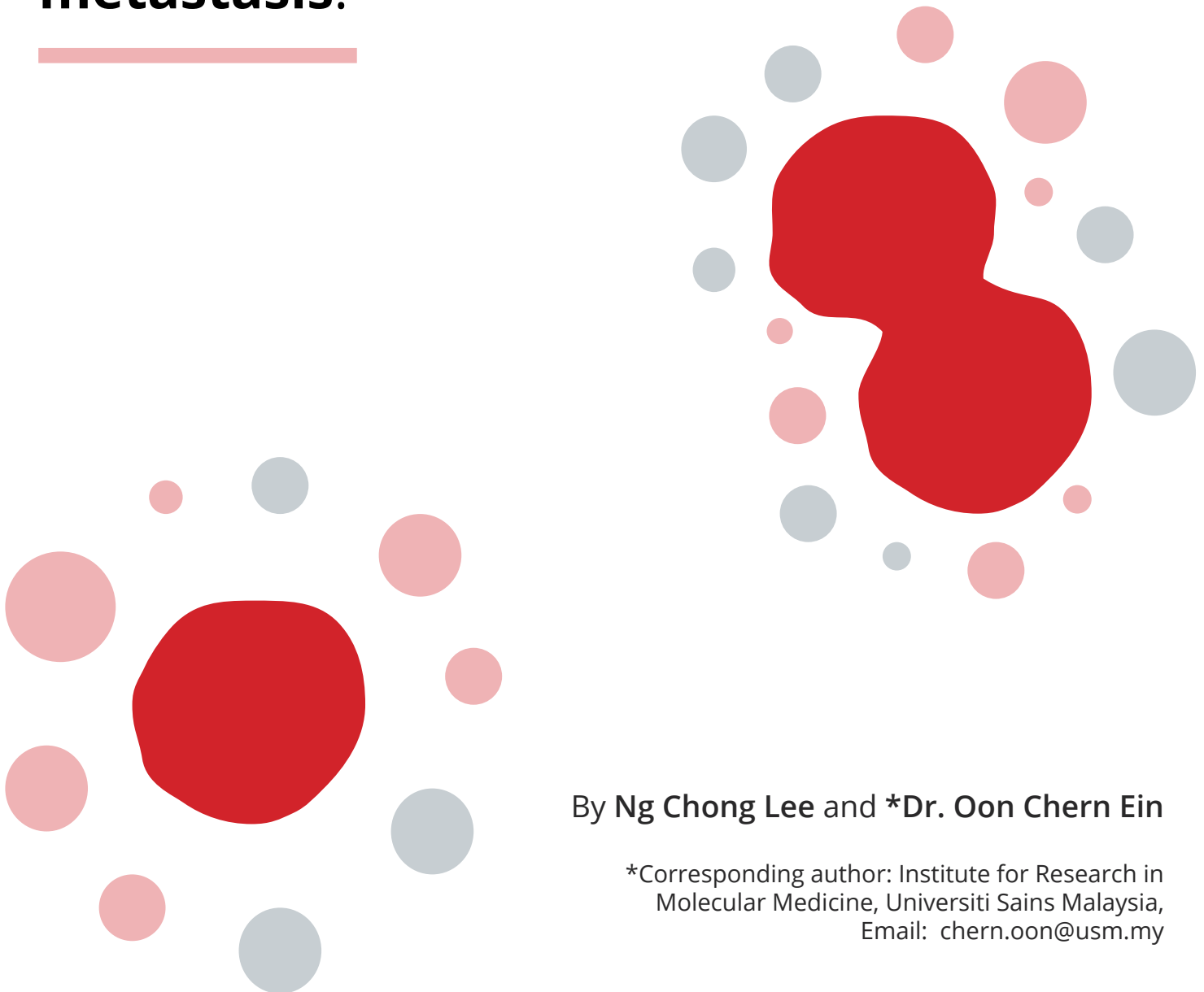
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Cancer's Little Helpers:

Cells within the tumour microenvironment
and their roles in **tumourigenesis** and
metastasis.



By Ng Chong Lee and *Dr. Oon Chern Ein

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At a Glance

Diverse cell populations in solid tumours have been shown to affect the outcome of cancer treatments. This is because each component within the tumour microenvironment interacts with the tumour as a whole. Thus, the role of the tumour microenvironment in cancer progression is gaining recognition. This article intends to provide an introduction to the different types of cells that make up the tumour microenvironment, and how they may aid in cancer progression and metastasis.

Cancer is a disease characterised by abnormal cell growth in the body, which could be lethal if not diagnosed and treated early. According to the Malaysian National Cancer Registry Report 2007-2011, there were 103,507 total new cancer cases reported in Malaysia from 2007-2011, with a lifetime risk for males and females of 1:10 and 1:9, respectively [1]. Although fatalities due to cancer have been increasing from year 2007-2011, there is still no complete cure for all cancers and, it bears a risk of recurrence and resistance to current drugs used for therapy. Thus, intense research is still being done to understand cancer in order to find a cure for it.

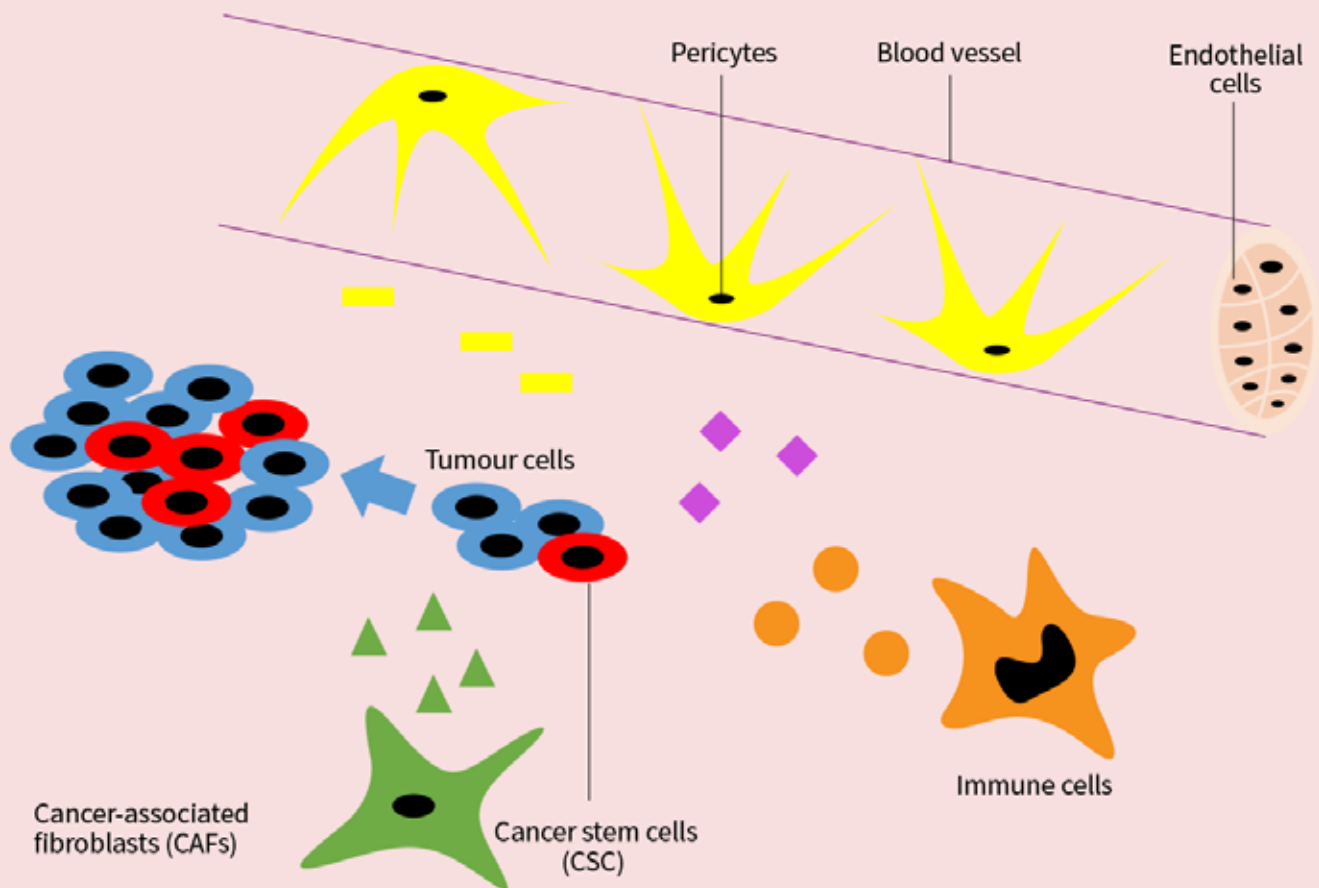
While efforts to treat cancer normally focus on the characteristics of the tumour itself—its size, location, and how invasive it is, the cell populations surrounding the cancerous cells have also been found to play important roles in influencing cancer growth and metastasis [2]. These cell populations and their secreted factors within the surrounding environment of the tumour cells are

described as the tumour microenvironment.

This microenvironment consists of various components including cancer-associated fibroblasts (CAFs), endothelial cells, pericytes, immune cells and cancer stem cells (CSC), among many others. Although these cells can be found in almost all body parts, they are found to be altered within the tumour microenvironment and may therefore be different from their normal forms in the way they interact with tumour cells. However, it is still under investigation whether these abnormal functions of the tumor microenvironment are the causes or consequences of cancer. As demonstrated in **Figure 1**, the tumour microenvironment can interact with cancerous cells and modulate the surrounding environment to favour cancer growth and the spread of cancerous cells to other sites (metastasis). Tumours often work together with their surrounding components as a unit to promote cancer growth, progression and metastasis, rather than only relying on the aberration in the tumour cells alone.

Figure 1:

Interaction of tumour cells with the tumor microenvironment



- Pericytes secreted factors
- ◆ Endothelial cells secreted factors
- Immune cells secreted factors
- ▲ CAFs secreted factors

Interaction of tumour cells with the tumour microenvironment. Different cell populations in the tumour microenvironment are able to communicate with tumour cells directly (cell-cell interactions) or indirectly (secreted factors) to promote tumour proliferation and metastasis.



Immune cells

Immune cells such as white blood cells are vital to our daily health by protecting us from infections like flu and fever. However, studies in recent decades have suggested that secreted inflammatory substances from white blood cells are also able to promote cancer growth [3]. Additionally, different subtypes of immune cells have been found to suppress the immune response toward tumour cells, thus fueling tumour growth instead of destroying abnormal tumour cells [3].



Endothelial cells

Similarly, endothelial cells which line the blood vessels in the body and help in nutrient and oxygen delivery to our organs can be hijacked and modified to aid tumour growth during cancer development. This occurs through the interaction of tumour cells with endothelial cells to encourage secretion of blood vessel-forming factors. This results in an abnormal increase in blood vessel formation in cancerous tissue, which then promotes nutrient and oxygen transport to the tumour cells, to enable proliferation of the tumour. Interestingly, the blood vessels surrounding the tumour cells are usually more permeable than normal blood vessels [2]. These blood vessels have been suggested to promote the spread of the tumour because it is easier for the tumour cells to enter these blood vessels and be transported to other sites within the body (tumour metastasis), as opposed to normal blood vessels that have fortified walls that do not allow entry of normal cells excluding immune cells.



Pericytes

Pericytes, which are contractile cells that wrap around the blood vessels, appear to have lower vasculature coverage within the tumour than is normal and are associated with increased cancer metastasis [5]. Under normal conditions, pericytes can provide structural support to blood vessels, to regulate the blood vessels' permeability and blood flow. However, low pericyte coverage on the blood vessels surrounding the tumour may contribute to permeability of tumour cells into the bloodstream and its delivery to other body sites.



Fibroblasts

While normal fibroblasts act as part of the connective tissues in the body and have important functions in wound healing processes, fibroblasts near to tumour cells, also known as cancer-associated fibroblasts (CAFs), are known to be quite different. These CAFs have been shown to release growth factors that promote cancer metastasis and resistance toward cancer therapy [4].



Cancer Stem Cells (CSCs)

CSCs are stem cells that exist inside a tumour. Under normal physiological conditions, stem cells are well regulated by different factors and their proliferation is controlled for appropriate formation of different organs and cell types. However, within a tumour, the growth of stem cells are not regulated, leading to abnormal growth. Thus, these stem cells are known as cancer stem cells. Similar to normal stem cells that possess the ability to regenerate blood, skin, nails and hair in our body, these CSCs are able to re-form the tumour by themselves. They have also been suggested to contribute to the initiation of tumour metastasis and are implicated in resistance to anti-cancer therapy due to their cancer initiation ability and reduced responsiveness towards anti-tumour drugs [6].



Extracellular Matrix

Cancer proliferation and metastasis can also be affected by the non-cellular components of the tumour microenvironment, known as the extracellular matrix (ECM). The ECM is composed of many growth factors, enzymes that digest other proteins, and elastic fibers and collagen, which normally provide structural support as well as help in cell-cell communication and adhesion in the body under normal circumstances. However, the composition of the ECM can be altered during carcinogenesis to favor tumour growth and metastasis.

Similar to normal stem cells that possess the ability to regenerate blood, skin, nails and hair in our body, these CSCs (Cancer Stem Cells) are able to re-form the tumour by themselves.

How does the tumour microenvironment affect treatment outcome?

Chemotherapy, surgery and radiation therapy are commonly used to treat cancer. Chemotherapy induces cell death of rapidly proliferating cells, which includes cancer cells as well as other normal cells that are constantly dividing such as intestinal cells, hair and bone marrow. Thus, it can also wipe out the immune cells that possess anti-tumour activity within the tumour microenvironment. In addition, during chemotherapy, the leaky blood vessels surrounding the tumour have been described to contribute to the buildup of fluid pressure around the tumour. This results in a higher fluid pressure in the tumour microenvironment compared to surrounding blood vessels, which impedes blood flow and reduces the diffusion of chemotherapeutic drugs into the tumour, thereby making the tumour more resistant to drugs [7].

With regards to delivery of chemotherapeutic drugs, many drugs are encapsulated within synthetic polymers to enhance their delivery. These synthetic polymers may release hyaluronan and lactic acid, which can bind to specific receptors on the cancer cells within the tumour microenvironment to support tumour growth and induce tumour hypoxia

(low oxygen state) [8]. As hypoxia can induce blood vessel formation in tumours and enhance the drug resistance of tumours, it would not be favorable to stimulate a hypoxic state near a tumour.

The tumour microenvironment can also be influenced towards an immunosuppressive state in post-surgery recurrent tumours [9]. Wound healing occurs as a normal process after surgery, during which an immunosuppressive state is induced to favour wound healing. However, this immunosuppression may also favour tumour growth and prevent the anti-tumour activity of immune cells.

Utilising a person's immune system to target and kill cancerous cells is one of the strategies which has successfully entered clinical trials for blood cancer in recent years [10]. But in the case of solid tumours, the existing immunosuppressed tumour microenvironment may inactivate the immune response against the tumour. Hence, it will be necessary to overturn the immunosuppressive environment around a solid tumour for a better treatment outcome.



In conclusion, continuous improvement based on studies and findings about the tumour microenvironment will be necessary to improve the efficacy of current therapies. In particular, careful analysis and selection of tumour conditions and treatment types should be done to minimize any pro-tumoural effects stemming from aspects of the tumour microenvironment. Due to the possible adverse interactions of materials and treatment conditions used with tumours and their microenvironments, it could be more difficult to treat or predict a patient's disease. Thus, targeting both cancerous cells and pro-tumoural components in the tumour microenvironment would most likely yield a better treatment outcome.

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Dating Cancer:

the historical timeline of a deadly disease

At a Glance

Cancer is a formidable foe that all of us hope never to catch a glimpse of, let alone have a personal encounter with. However, as the saying goes, we should 'keep our friends close, and our enemies closer.' Thus, this article seeks to provide an informative and conceptual understanding of the events that occurred throughout our known history with cancer. The timeline is not arranged in a strictly chronological way, as in reality, many discoveries and ideas are stumbled upon out of order, with their meaning or implications becoming clear only in retrospect. Many discoveries are also greatly abbreviated, and interested readers are encouraged to refer to the references for further information.

Earliest Stages

3000 B.C.-1600 B.C. Earliest Traces of Cancer



Ancient Egypt

The Edwin Smith Papyrus describes eight cases of tumours or ulcers of the breast treated by cauterisation [4]. Mummified remains reveal bone cancer in humans [1].

460 B.C.-370 B.C. Cancer Receives its Name



Classical Greece

Hippocrates labels cancerous tumours as 'karkinos', a Greek word that means crab, and that references the appearance of finger-like projections from cancerous tissue. The word becomes the predecessor to the modern term 'cancer' [1].

Theories abound on the origin of cancer

460 B.C-mid-20th century



460 BC

Humoral Theory

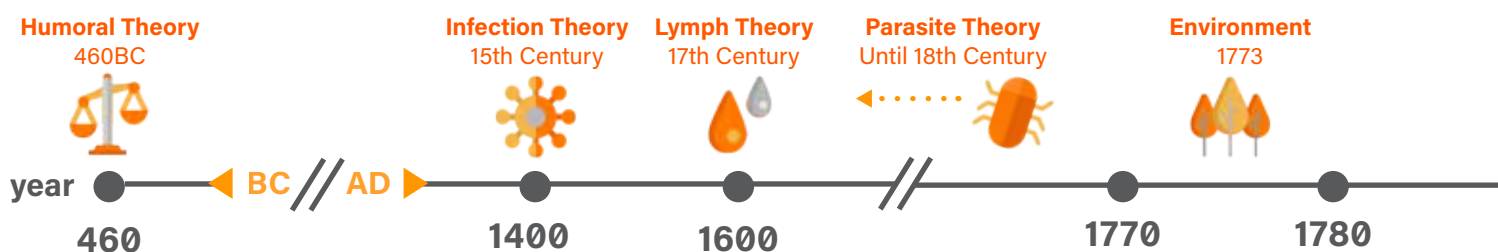
Hippocrates proposes that an imbalance in the 4 humours (body fluids)—blood, phlegm, yellow bile, and black bile—results in disease. Excess black bile in an organ is believed to be the cause of cancer throughout the Middle Ages for the next 1300 years, during which autopsies were banned due to religious reasons.



15th
Century

Infection Theory

Zacutus Lusitani and Nicholas Tulp suggested that cancer is contagious, based on multiple breast cancer cases observed within the same household [6].



Theories abound on the origin of cancer

460 B.C-mid-20th century (continued)



17th
Century

Lymph Theory

Continuous movement of lymph and blood is believed to be vital for life. Tumours are thought to grow from lymph constantly being thrown out by blood [6].



Until 18th
Century

Parasite Theory

Parasites are thought to cause cancer



1838

Blastema Theory

Johannes Muller demonstrates that cancer is made up of cells, not lymph. His student, Rudolph Virchow discovers that cancer cells come from normal cells.



1863

Chronic Irritation Theory

Virchow discovers white blood cells in cancerous tissue, proposes that chronic irritation causes cancer and is the first person to link inflammation with cancer.



1800-
1920s

Trauma Theory

Cancer is believed to be caused by trauma



Blastema Theory

1838



1790

1800

1810

1820

1830

1840

1850

Trauma Theory
1800-1920

Various sources of cancer are identified

1775 onwards



1886

Inheritance

Hilário de Gouvêa determines that two of seven children have inherited retinoblastoma (a malignant eye tumour) from their father and reports an inheritable susceptibility to cancer [2].



1775

Environment

Percivall Pott links chimney soot with squamous cell carcinoma occurrence in chimney sweeps, and is the first person to implicate environmental factors in cancer development [1,2].



1911

Viruses

Rous sarcoma virus is discovered to cause cancer in chickens by Peyton Rou, proving that some cancers are caused by infectious agents [1].



1915

Chemicals

Katsusaburo Yamagiwa and Koichi Ichakawa induced cancer in rabbits by applying coal tar to the skin, proving that certain chemicals cause cancer [2]. Tar, a potent carcinogen released from burning tobacco, would be freely ingested by humans via cigarettes for decades, right up to the present day [1].



1927

Radiation

Hermann Joseph Muller reports radiation causes gene mutation. He eventually receives a Nobel Prize for his discovery [3].

Chronic Irritation Theory

1863



Inheritance

1886



DNA Damage

1902



Viruses

1911



Chemicals

1915



1860

1870

1880

1890

1900

1910

1920

Trauma Theory
1800-1920

Damaged DNA is identified as cancer's 'elixir of life'

1902-1962



DNA Damage

Theodor Boveri suggests that chromosomal damage in single cells causes them to undergo uncontrollable division and to give rise to cancerous tumours [2].

1902



DNA Structure

Watson and Crick discover DNA's helical structure and receive a Nobel Prize.

1962

The good and the bad.

1970s

Suppressor Genes



1979

Tumour suppressor genes (genes that suppress cancer) such as TP53 are identified.

Oncogenes



1984

Oncogenes (genes that promote cancer), such as HER2, are discovered.

Associated Genes



1994-1995

Genes associated with inheritable cancers are discovered, such as BRCA1 and BRCA2, which are associated with breast cancer.

Radiation
1927



Pap Smear
1940s



DNA Structure
1962



Imaging
1970s



Suppressor
genes
1979



Oncogenes
1984



Associated
genes
1994-1995



1930

1940

1950

1960

1970

1980

1990

Detection frenzy.

1940s-1970s



Pap Smear

Pap smears, developed by George Papanicolaou in 1943, becomes the first cancer screening test for cervical cancer.

1940s



Imaging

Ultrasound, computed tomography (CT scans), magnetic resonance imaging (MRI) scans and positron emission tomography (PET scans) replace exploratory surgery. Doctors begin to use video cameras and scopes to find and remove tumours.

1970s

Seeking the Cure ^[1]

Late 20th century till now.



Lasers and liquid nitrogen sprays to freeze tumours are used as less invasive ways to remove tumours.



Hormone therapy: Hormones to avoid, curb, or treat breast and prostate cancers are used.



Radiation: Discovered to cause as well as cure cancer. Radiation is used to kill tumour cells directly and to irradiate adjacent tissue following the surgical removal of tumours.



Transplants: hematopoietic stem cell transplants are used for cancers affecting the blood or immune system.



Chemotherapy: Soldiers exposed to mustard gas in World War II developed bone marrow suppression [4]. Subsequently, nitrogen mustard (a similar drug) was discovered to be effective against rapidly dividing cells (like cancer cells) by destroying their DNA [2]. Drugs and radiation therapy were combined with surgery. Then, different drug combinations were studied, followed by new delivery methods employing the use of liposomes (fat droplets) and monoclonal antibodies to reduce side effects of drugs and to target cancer cells specifically [1]. Chemoprotective agents are also used to reduce side effects [1].



Immunotherapy: Naturally occurring biological agents (interleukins, interferons, cytokines) are developed in the lab to influence immune responses to help the body control cancer. Monoclonal antibodies are used to specifically target cancer cells. Vaccines are currently being developed to boost the body's immune responses.

2000

Therapies targeting tumours and not normal cells are developed ^[1]

Late 20th century till now.



Growth Signal Inhibitors

Abnormal growth factors are discovered to contribute to cancer. Therapies that block abnormal growth factors are developed.

Apoptosis Inducers

Apoptosis inducing drugs are used to force cancer cells to die without DNA repair.

Endogenous angioinhibitors

Drugs to inhibit blood vessel formation are used to starve tumours.

Vaccines against cancer ^[2]

21st century till now.

2006 Gardasil is approved by the FDA as a vaccine against human papilloma virus (HPV) infection, guarding against the two HPV strains that cause 70% of all cervical cancer.



2009 A second vaccine called Cervarix is developed that also guards against the two HPV strains that cause 70% of all cervical cancer is approved.

Getting personal about it ^[2]

21st century till now.

2010

FDA approved sipuleucel-T (Provenge) is developed as a cancer treatment vaccine that employs a patient's own immune cells (dendritic cells) to treat metastatic prostate cancer that is unresponsive to hormonal therapy. It is the first and only human cancer treatment vaccine currently approved for use.



DNA analysis and the birth of precision medicine ^[2]

21st century till now.



DNA Analysis

2014 - Researchers from The Cancer Genome Atlas (TCGA) project analyse DNA from cancers and report that stomach cancer can occur due to four different cancers, based on distinct tumour characteristics.



Precision Oncology

Research to identify and analyse the genetic profiles of cancers from patients begin, leading to cancer classification based on their molecular abnormalities as well as site of origin.

This approach which seeks to match and tailor treatments based on the genetic profile of the cancer and of the individual is termed precision medicine or precision oncology, and is considered to be the future of cancer therapy [5].

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Untangling the Genetic Mysteries of Cancer Using Computational Tools

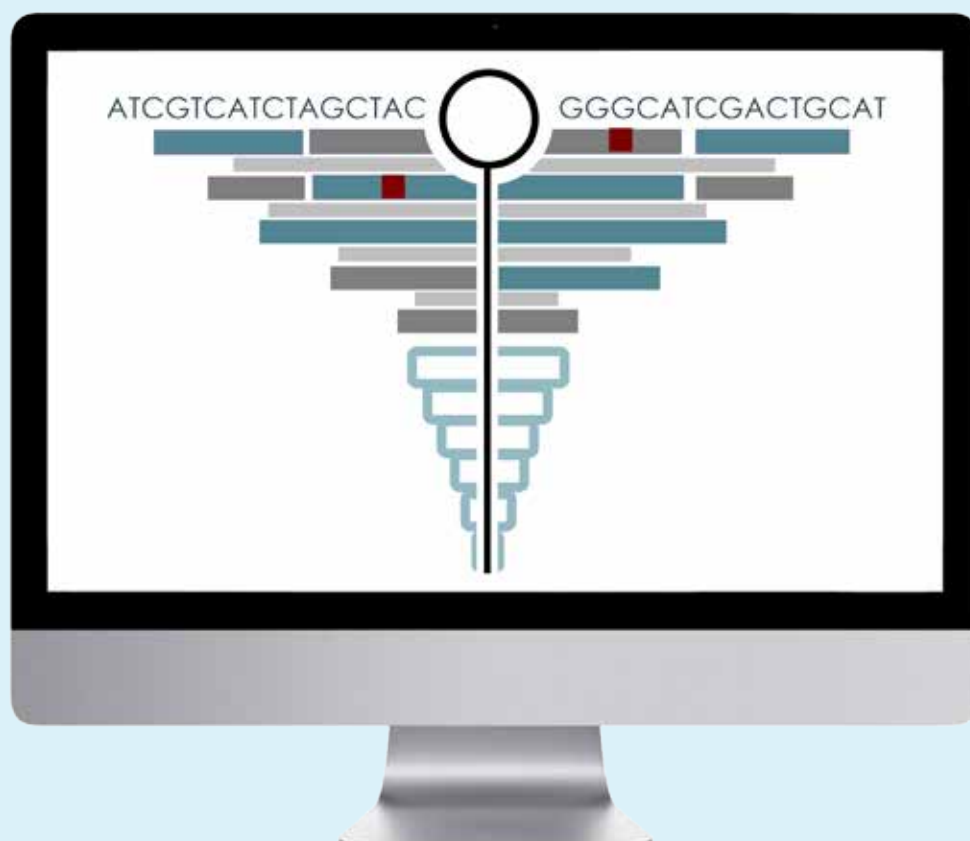
by Dr. Camelia Quek and Dr. Kelly Quek

At a Glance

Computational tools have revolutionised the study of cancer genomics. These tools are often implemented in the field of Bioinformatics, an interdisciplinary field that incorporates biomedical science, computer science, and mathematics to analyse and interpret complex biological data. The implementation of computational biology and bioinformatics in cancer genomic studies has made significant impact in the identification of clinically relevant mutational signatures and tumour heterogeneity.

For the past decades, we have witnessed how technologies affect our daily activities. The impact of technology in our lives can be observed in health and medicine, education, infrastructure development, discovery and communication. Cloud computing is one of the examples that impacts our lives on many levels, enabling us to conveniently organise and share data, and rapidly access files. In the context of cancer research, high-throughput sequencing technologies and computational methods have revolutionised the study of genes involved in cancer development, or cancer

genomes, with numerous discoveries relating to diagnosis and treatment. These emerging research technologies and computational methods are developed for researchers to provide further insight and better understanding of biological mechanisms underlying cancer disease progression. Cancer is the result of the accumulation of genetic and epigenetic (non-genetic) alterations in key genes, which ultimately lead to uncontrolled growth of mutated cells. The identification of genetic and epigenetic alterations in the complex cellular architecture of a cancer genome requires comprehensive



“...datasets have accumulated at an exponential rate, **outpacing one's ability to analyse its biological meaning**”



bioinformatics pipelines involving a great number of computational tools.


Bioinformatics is an interdisciplinary field, which incorporates biomedical science, computer science, and mathematics to analyse and interpret large collections of biological data with high diversity or variation (heterogeneity), such as genetic sequences, cell populations or protein samples. The goal is often to generate new predictions or discover new biology. This approach is important for integration of large heterogeneous data, with the purpose of acquiring a comprehensive understanding of multi-dimensional and complex biological systems. As the acquisition of sheer volume of biological data has become increasingly rapid and cost-efficient due to high-throughput sequencing technologies, datasets have accumulated at an exponential rate, outpacing one's ability to analyse its biological meaning. To process the large volume of data comprehensively and efficiently, many computational tools that use state-of-the-art algorithms have been developed. These tools are used to study genome and epigenome data, which contain vital information about tumour development, progression and metastasis (spread).

Ding et al. recently reviewed the computational tools for identifying genomic alterations, and for defining mutational signatures and patterns, and molecular networks that drive observable cancer characteristics (phenotypes) and genetic diversity in tumours [1]. The bioinformatics process is categorised into two main components: variant detection, and variant annotation and

interpretation. To discover variations of single DNA nucleotides (single nucleotide polymorphisms or SNPs) and indels (insertions and deletions) within the DNA sequence, the more common and highly cited bioinformatics tools include VarScan [2], GATK [3], Mutect [4], and Somatic Sniper [5]. Other tools such as BreakDancer [6], GenomeSTRIP [7] and ChimeraScan [8] have been successfully utilised to uncover large scale copy-number aberrations in structural variants and gene fusions. The next step is to further understand how genetic alterations deregulate signalling, regulatory or metabolic pathways, thereby influencing tumour growth. To do this, more sophisticated approaches can be used to assess the clustering of mutations in curated (known) pathways and interaction networks. These include the gene-set enrichment analysis [9], hypergeometric test, and significant mutated genes (SMG) test [10]. Databases such as KEGG [11,12], Reactome [13], STRING [14] and BioGrid [15] are publicly available for researchers. This data will help cancer researchers interpret biological pathways in cancer. In recent years, computing technologies have been routinely used in cancer genomics research for the development of computational algorithms, and in the identification of somatic events (sporadic alteration that arise in response to environmental and lifestyle exposure) and mutations for the discovery of cancer-causing genes. The implementation of computational tools has also provided further improvement in the analysis of the spectrum of mutations in cancer. Two interesting cancer genomic studies are worth mentioning regarding this.

The first example is the genomic study on skin cancer (melanoma), led by Australian researchers at the Melanoma Institute Australia and QIMR Berghofer Medical Research Institute. The researchers applied a variety of computational tools to identify a landscape of mutational signatures in melanoma that are not related to ultra-violet radiation [16]. These tools include OncodriveFML [17] (to detect promoters and untranslated regions (UTRs) of genes in tumorigenesis), Combined Annotation Dependent Depletion (impact of mutations in gene promoters) [18], qSNP and GATK (to detect genetic mutation) [3,19], and MSigDB (pathway identification) [20]. This led to the finding that mutations in acral melanoma (found on hands and feet) and mucosal melanoma (internal surfaces) are predominantly caused by structural changes and genetic mutations that were not previously implicated in melanoma. Examples of significantly mutated genes included *Braf*, *Nras* and *Nf1* in acral melanoma and *Sf3b1* in mucosal melanoma. Genes such as *Braf*, *Cdkn2a*, *Nras* and *Tp53* were also detected in cutaneous melanoma. In addition, most of the identified melanoma mutations are implicated in fundamental signaling pathways such as the mitogen-activated protein kinase (involved in cellular responses such as proliferation, differentiation, motility, stress response, cell death and survival) and phosphoinositol kinase pathways (involved in cell metabolism, growth, proliferation, survival, protein synthesis and transcription), providing a lead for further research on the role of these pathways in melanoma. Such findings demonstrate the efficacy of applying computational biology and bioinformatics for the discovery of novel cancer genes and characteristic mutational (genomic) subtypes of cancer.

The second example is the epigenomic study led by MD Anderson Cancer Center that investigated intra-tumour heterogeneity (i.e. cancer cells that harbour distinct molecular and phenotypic features within a given tumor) with respect to DNA methylation (the modification of DNA without changing the DNA sequence via attachment of methyl groups, usually resulting in repression of genes) in localised lung adenocarcinomas [21]. In this study, the authors employed a multi-region sequencing approach to quantify intra-tumour DNA methylation heterogeneity and investigated the relationship with clinical outcomes. Examples of computational and statistical analyses employed in this study are SWAN (to normalise raw methylation values) [22], IlluminaHumanMethylation450k.db annotation (to annotate the CpG probes locations), Euclidean distance matrix (to measure genetic diversity) and bootstrapping (to validate observations). Through these genomics and statistical analyses, the authors demonstrated that intra-tumour DNA methylation heterogeneity might occur later during cancer development and that it was associated with clinical characteristics such as tumour size, advanced age and increased risk of post-surgical recurrence. These findings are important for the understanding of the adverse clinical outcomes of cancer patients, and suggest that intra-tumour DNA methylation heterogeneity profiles can potentially contribute to the development of prognostic biomarkers.

From the examples described above, it is clear that computational biology and bioinformatics have been proven to be useful in identifying and defining mutational signatures, signalling pathways and molecular networks that drive cancer pathogenesis. In the fight against cancer, these technologies are a promising tool not only to assist researchers in better understanding cancer-causing mechanisms, but also to help clinicians with diagnosis and treatment decisions. 

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Cancer Conflicts:



**Ethical issues
in the tug-of-
war between
conventional
and alternative
treatments for
cancer.**



by Enakshi Sivasudhan

At a Glance

How does one decide whether to opt for conventional or alternative treatments when diagnosed with cancer? What are the challenges one has to face for each option? This article aims to bring forth some of the underlying ethical issues faced by cancer patients when considering treatment options that differ from mainstream medical intervention. The author hopes to provide an introduction to complementary and alternative treatments and discuss possible ways of dealing with the ethical implications, such as implementing governing and funding bodies, re-evaluating health insurance policies and also improving doctor-patient communication.

53-year-old Elaine, upon hearing the distressing diagnosis made by her oncologist, posed a typical question any patient diagnosed with Stage 2 breast cancer, or any debilitating disease for that matter, would do to their doctor: “Are there any alternative treatment options?”

Cancer patients who opt for ‘unconventional’ treatment methods mostly do so due to their fear of going under the knife and the adverse effects of chemotherapy and radiation concomitant with conventional treatment. More often than not, these alternative treatment options are met with lingering suspicion if not downright prejudice by the medical community due to the lack of firm scientific evidence of their safety and efficacy. This, in turn, gives rise to a plethora of ethical implications that needs to be addressed with the sole purpose of assisting patients make better-informed decisions concerning their health, especially those diagnosed with cancer.

Alternative medicine refers to treatments that are used instead of mainstream medicine whereas complementary medicine refers to treatments practiced alongside mainstream treatment. Collectively, they are often referred to as Complementary and Alternative Medicine (CAM) [1]. CAM comprises the use of dietary supplements and herbal medicine, the practising of traditional methods such as acupuncture, naturopathy



“The chemotherapy industry is a giant scam”

- A common response from cancer survivors and their loved ones on why they chose alternative treatments for cancer



and homeopathy, as well as modes of healing the body by touch (such as chiropractic medicine, massage and yoga). Some patients opt for electromagnetic therapy, as they believe external energies have a positive healing effect on the body. Meditation and prayer too are widely believed to improve one's emotional and mental health [2]. Quite often cancer patients find complementary and alternative interventions more appealing due to their being perceived as natural, safe and non-toxic, in contrast to mainstream

medicine which may be seen as being toxic due to the harsh side effects. Many patients prefer holistic approaches of CAM, which ensures overall care towards improving their quality of life. Financial constraints, poor prognostic outcomes, physician-patient conflicts due to differences in opinion, improvement of body immunity, and the desire to avert cancer recurrence are some of the common trigger points for choosing CAM over conventional medicine [3,4].

While doing my initial research for this article,

I spoke to a few cancer survivors and their loved ones who gave their personal insights into why they chose alternative treatments. Many of them, to quote their exact wording, said: “The chemotherapy industry is a giant scam”. According to their views, big pharmaceutical corporations seem to be pursuing the common goal of creating customers and increasing their profitability instead of working towards finding cures for cancer. According to the American Cancer Society, the direct medical cost of treating cancer was estimated to be \$87.7 billion

The use of CAM (Complementary and Alternative Medicine) has dramatically increased over the past 25 years and gained economic, medical and sociological importance

in the US alone in 2014 [5], and is expected to increase in the coming years with the increased frequency of this illness, as 1 in 2 men and 1 in 3 women are diagnosed each year [6]. It is a conspicuous fact that treating this disease is immensely profitable when compared to employing prevention measures to stop the disease from occurring. It is this very opinion that tends to make patients have misgivings about the conventional mode of cancer treatment, thus pushing them towards other alternatives.

As mentioned in the beginning of this article, the palliative care assured by CAM treatment is often looked down upon by the medical community, owing to their lack of cogent scientific evidence to verify their safety and efficacy [7]. For any treatment, irrespective of whether it is conventional or alternative or complementary, it is imperative that the following questions addressing ethical

implications are carefully reviewed: Is it safe? Is there any reliable evidence to prove its efficacy? What are the adverse or long-term side effects? Are the professionals who will be administering it properly qualified for the task? Is it a generic treatment or is the specificity dependent on age, gender, medical history of the patient, or allergies?

Many sceptics who frown upon conventional medicine, as well as those who have witnessed miraculous cures through the use of alternative methods, question the validity and purpose of rigorous scientific experimentation and the significance of statistical data. They think that if a treatment has successfully managed to produce results with an impressive 'survival rate', then the need for scientific proof becomes inconsequential, which often is a wrong conclusion to jump to. It is noteworthy to mention that over the years CAM has produced positive



results in certain aspects of medical treatment. For example, the following is a very good example of how an alternative treatment, initially met with suspicion, was later proved to be as good as, if not better than, conventional treatment options. Years of intensive research on CAM led to the discovery of a new group of antimalarial drug compounds found in an ancient Chinese herb *Artemisia annua*, a medicinal plant used to treat chronic fevers for thousands of years. Today Artemisinin and its semi-synthetic derivative artemisinin-based combination therapy (ACT) is used as an efficacious treatment option for Malaria [7, 11]. Whilst the complementary and alternative treatment practice is not to be

condemned entirely, we should not blindly follow spurious treatments that lack accurate systematic evidence. For example, Laetrile (Vitamin B17) has been promoted as an effective alternative cancer treatment for decades. However, a recent expert-reviewed study revealed that Laetrile showed an insignificant anticancer effect in addition to posing the threat of negative side effects, and it was eventually disapproved by the US Food and Drug Administration (FDA) [8, 9].

The use of CAM has dramatically increased over the past 25 years and gained economic, medical and sociological importance, with a prevalence rate of nearly 34% (range of 7% to 64%) among cancer patients

[2]. Thus, it is crucial for the relevant authorities to take necessary steps towards confronting a complex array of ethical dilemmas raised by CAM treatments. First and foremost, an authoritative organisation that investigates and carefully evaluates promising alternative and complementary medical practices should be formed. With this goal in mind, the National Centre for Complementary and Alternative Medicine (NCCAM) was formed in the US with multi-million dollar funding to conduct research into non-conventional medical options [10]. Similar initiatives should be encouraged and implemented by other governments around the globe. As part of this initiative, sufficient funds



should be allocated by governing bodies and capable young minds should be encouraged to pursue knowledge in this field by providing them with proper education and research opportunities.

Quite often certain CAM treatments tend to be expensive and time-consuming. The Gerson Therapy which is a nutritional approach of detoxifying the body and strengthening the immune system through a dietary regiment of coffee enemas, organic fruits and vegetables and various supplements is allegedly thought to have anti-cancer

effects [12]. Despite strong skepticism posed by the medical community due to the lack of scientific data to warrant claims of its effectiveness, this treatment method continues to be recommended to terminal patients around the world. However, this treatment requires time, dedication and exorbitant amounts of money. Rigorous research into CAM treatments (such as the Gerson Therapy) could eventually aid CAM practitioners to guide their patients to make better-informed decisions as well as provide health insurance coverage, should CAM treatments be given equal


prominence as conventional medicine in the future.

When discussing ethical implications concerning CAM usage, the nature of patient-doctor communication should be taken into immense consideration. It can be challenging for a medically-trained physician practicing conventional medicine to set aside his personal bias and advise patients on alternative treatment options. However, they should establish open lines of communication by discussing the possible outcomes of conventional, as well as CAM interventions before allowing the





patient decide. Even if a situation arises whereby the patient decides to pursue conventional treatment due to failure of CAM options and can no longer benefit from curative interventions, the physicians should deliver palliative care. Should the physician feel that a CAM care provider treated a patient inappropriately; he should report him to the authoritative licensing body for investigation [13].

There is constant hostility between those who practice conventional medicine and those who exercise alternative treatments. The former shuns the latter on the grounds of lacking proof or reliable evidence, while the latter discredits the former with claims of choosing profitability over altruism. We should keep in mind that in the end, we are all fighting the same war: the war against cancer. While we work on improving conventional medical care, we should also encourage conceptual as well as empirical research into CAM to increase the chances of developing better diagnostic and curative technologies. While doing so we should be on the look-out for possible ethical implications that would invariably arise and deal with them by implementing governing and funding bodies, re-evaluating health insurance policies and also enhancing doctor-patient communication. 

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About the Author

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EATING YOUR WAY TO COLON CANCER

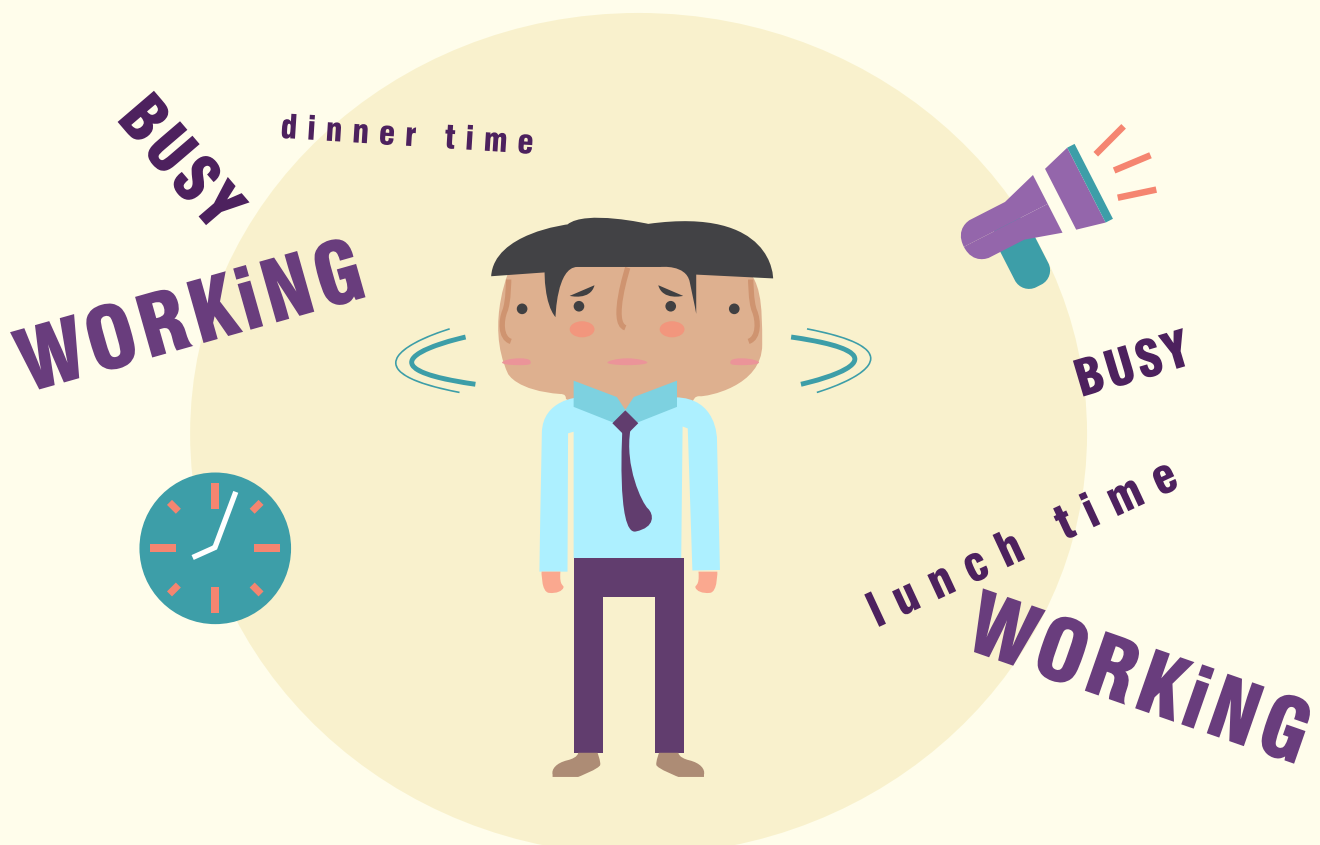
BY
JENNIFER
EYAHMALAY

Everyday due to work overload and a tight schedule, Mr. Ng was forced to consume unhealthy foods, such as fast food which is highly processed, abundant in fat and low in fibre content. Due to this unhealthy lifestyle, he frequently suffered from constipation, bloating, irregular bowel movement and sometimes even bloody stools.

Mr. Ng, a successful marketing manager in the hustle and bustle filled Kuala Lumpur, knew very well that he was leading a sedentary lifestyle, but he thought he

couldn't do anything about it since his busy days wouldn't allow him to spare even a few minutes to improve his diet and lifestyle. Even though he feared for his health, he still resisted visiting a physician for a check-up because he thought his symptoms were normal side-effects due to his unhealthy diet. Perhaps he was also naively overconfident that he wouldn't be so unlucky as to encounter a life-threatening disease.

Unfortunately for Mr. Ng, he was slowly falling prey to colon cancer. While the



JAPAN
SINGAPORE
SOUTH KOREA

MALAYSIA
&
OTHERS

COLON CANCER
in ASIAN COUNTRIES

incidence of colon cancer in Asian countries is higher in Japan, Singapore and South Korea compared to Malaysia and other developing countries, it is still the second most common cancer in Malaysia and the third most common cancer in the world [1]. In fact, studies inform that ethnic Chinese have the highest rate of colon cancer among Malaysians, followed by Malays and Indians. At the same time, Malaysian men are also known to have higher chances of getting colon cancer compared to Malaysian women, especially after the age of 40 [2].

COLON
CANCER

2

SECOND MOST
common cancer in
Malaysia

As he continued with his usual routines, Mr. Ng suddenly suffered from serious diarrhoea which lasted for 2 weeks. He had no choice but to seek medical advice. The doctor explained to him that his symptoms could be due to colon cancer. Mr. Ng was deeply saddened and unable to accept that his fears has finally become reality. He realised that his careless attitude towards health had led him to disaster. He then underwent a colonoscopy, where a thin and flexible tube with a small video camera called a colonoscope was inserted into his large intestine to look at the whole colon and the lower part of his small intestine. Ulcers, colon polyps, tumours and inflammations could be seen via the colonoscope and samples of abnormal growth were also removed using this method of screening [3].

COLON
CANCER

3

THIRD MOST
common cancer in
The World


Results from the colonoscopy revealed that his symptoms were due to the growth of polyps. Polyps are non-cancerous cells that occur in the outermost lining of the intestine before reaching the inner layers of the colon or rectum [4]. While they are not cancerous, there is a high chance for polyps to develop into cancer [4]. Fortunately for Mr. Ng, the early detection of this growth enabled the polyps to be removed, saving the lymph nodes and other intestinal tissues from potential abnormal growth of the polyps and any cancerous cells which

might have led to the life-threatening stages of colon cancer.

If the polyps had grown into cancerous cells and spread into his intestinal tissue and lymph nodes, he would have had to be treated either by undergoing a colonostomy, a surgical opening through the abdomen to expel the waste into a bag that will be worn by the patient, chemotherapy, to kill the cancer cells from spreading to other parts of the body, or radiotherapy, which uses high energy radiation to kill cancer cells. These therapies are often used to treat rectal cancer [4].

While an unhealthy diet and lifestyle could cause colon cancer, heredity is also a contributing factor. Those with family

members who have been diagnosed with colon cancer should be screened for the cancer, especially after the age of 40 since 90% of colon cancer patients are those who have passed this age [4].

In Mr. Ng's case, his close brush with cancer led him to begin allocating his time more wisely and to choose to eat only healthy food with a high fibre content. He began to do regular exercises and even include functional foods such as probiotics and prebiotics in his diet, which greatly enhances the digestive system and overall wellbeing by supplying beneficial bacteria to the intestines. Though unpleasant, this incident served as an important turning point and eye opener for Mr. Ng, that helped change his lifestyle for the better. 

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Me and Earl and the Dying Girl

By Joshua Teh

At a Glance

Me and Earl and the Dying Girl is a beautifully shot coming-of-age film intent on examining high school life, cancer, and mortality, but a lack of depth causes it to ultimately fall short of its lofty ambitions.



The best films are those that linger on our minds long after the credits have rolled and the screen fades to black. These films are an experience, possessing a curious power to impact our feelings, to change our perspectives, to influence our thoughts.

It is a shame, then, that *Me and Earl and the Dying Girl*, a movie deeply concerned with the power of film, is not one of those films.

The titular 'Me' is Greg Gaines (Thomas Mann), a high school senior deftly adept at navigating the social cliques that exist at school without forming any real bonds. The only person he has resembling a friend is Earl (RJ Cyler), who Greg calls his "co-worker", largely because the two of them share a love of creating spoofs of movie classics such as *A Clockwork Orange* (A Sockwork Orange),

Midnight Cowboy (2:48pm Cowboy), and *Apocalypse Now* (A Box O' Lips, Wow).

But Greg's carefully constructed existence of non-engagement is thrown into turmoil when his mother (a woefully underused Connie Britton) discovers that Rachel (Olivia Cooke), one of Greg's classmates, has been diagnosed with leukemia. Forced to spend time with her, Greg slowly begins to open up to Rachel, and they begin to bond over viewings of Greg and Earl's home-brewed short films. And as her condition begins to deteriorate, one of her friends prompts Greg to create a film to help lift Rachel's spirits.

There is much to like about director Alfonso Gomez-Rejon's 2015 Sundance hit. While Greg might have been initially forced into a friendship with Rachel, there is an easy,

Rating	Director	Cast		Runtime
3 / 5	Alfonso Gomez-Rejon 	Greg Gaines Earl Rachel Kushner Denise Kushner Greg's Mom Greg's Dad	THOMAS MANN RJ CYLER OLIVIA COOKE MOLLY SHANNON CONNIE BRITTON NICK OFFERMAN	105 Minutes

believable chemistry between Mann and Cooke. Gomez-Rejon demonstrates a skilled hand in the film's direction – the camera angles which he employs and the snippets of Greg and Earl's short films peppered throughout are both funny and inventive. They provide the film with a light-hearted playfulness that contrasts nicely with its heavy themes of death and the cost of friendships.

Still, it is hard to shake the feeling that these flourishes are being used to disguise a lack of depth. All the characters are poorly fleshed out, with broad strokes taking the place of actual personalities. Most disappointing of all is how Rachel is reduced to the caricature of a cancer patient. In a film dealing with the question of youth facing their mortality,

she is merely Cancer Girl. Her struggles with leukemia chiefly surround her insecurities over her baldness caused by chemotherapy. She is a plot device, a means by which Greg can learn to grow beyond his hang-ups and weaknesses. We are never privy to her fears and doubts, or her having to let go of her dreams of attending the school dance and college and being able to live a normal life.

It is not much better for the other characters either. Earl is the sum total of a collection of black stereotypes – a poor kid living in

a dodgy neighbourhood with a deadbeat older brother. We do not even get to know Greg, the central character and narrator of the film, beyond his love for the classics and his lackadaisical approach to his own future. Mann is likable enough as Greg, but we are never fully invited behind the veil to understand how his relationship with Rachel has impacted him. This causes any epiphanies which he gathers toward the end to feel hollow and somewhat unearned. It is

hard to invest in characters which the film itself seems almost uninterested in them; at times, *Me and Earl and the Dying Girl* seems to deliberately mirror Greg's aloofness, holding us at arm's length while it whirls and twirls its way through Greg, Earl, and Rachel's high school life in Pittsburgh.

There is a scene close to the end where a clearly distressed Greg is overlaid with the

beautifully abstract short film that he has created for Rachel. It is both gorgeous and sad, driven home by the juxtaposition of life's impermanence captured forever in Greg's film. It is a reminder of what cinema is capable of, the feelings it can stir within us, and its ability to express what for many of us remains inexpressible. It is then that the emotions stirring beneath the film's surface become tangible enough for us to grasp, but the moment is fleeting and soon lost.





About the Author

Joshua Teh is a human rights advocate, and recently completed his L.LM in Human Rights at SOAS, University of London. In his spare time, he loves watching and reviewing films, and his favourite films are Aliens, There Will be Blood, and Moonlight.

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SIMULACRUM

BY LEE EE LEEN

AT A GLANCE

In a not too distant future Kuala Lumpur, the limited availability of cancer treatment gives rise to a thriving medical black market but one enforcer is not sure which side of the law she is on.

When the backlane surgeon off Chow Kit Road saw my medical tattoo, she gave me three vials of insulin. I smiled, pocketed the vials and left the clinic. Being an undercover enforcer with the Gene Therapy Unit (GTU), my tattoo was just for show. If I had to hand over my ID, it would say: “officer on suspension.”

The GTU targeted the black market for telomerase gene therapies, a lifeline to desperate patients. Conosulan, was an approved retroviral treatment for various cancers but intellectual property laws did not grant a patent for any derivatives. Conosulan could prolong patients’ lives by six years and counterfeits flooded the market as illegal labs mushroomed in derelict buildings.

As part of my suspension, I had to report to the Kuala Lumpur General Hospital for weekly medical checkups. I arrived during lunch hour and queued up in the canteen with other hospital staff who were lucky that they didn’t have to frequent back alley clinics for pirated drugs.

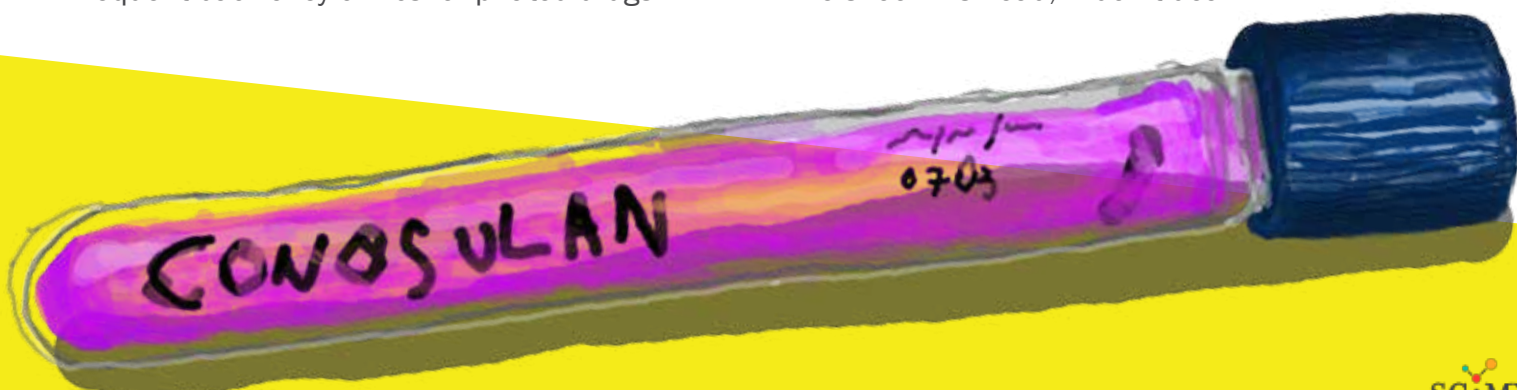
Sierra, my partner, joined me after I’d paid for my coffee. I hadn’t noticed her enter the canteen. “Unpaid medical leave looks better on your record than suspension,” she said.

Sierra and I had raided a shopping centre in Subang, long since succumbed to vandals and monsoon downpours. Once inside we located a graffitied storefront. According to the departmental database the electricity bill at this address was suspiciously high—overworked generators are a telltale sign of an illegal lab. I lifted the rusted shutter and prayed I wouldn’t contract tetanus.

“What you want, lady?” A raspy voice carried over TV static. I recognised the figure shambling out of the storeroom. Chan had been hauled in twice for questioning.

“Conosulan.”

He shook his head, “I don’t deal...”



But I could tell Chan was lying to me in the midst of all his pilfered high-end lab equipment. I took out a crumpled red packet of Koh Samurai cigarettes and offered him one. He accepted before kicking me in the groin. As I unsheathed my baton the packet tumbled to the grimy cement floor.

Sierra kept going over what happened that night, much to my chagrin. “We went in as plainclothes officers, ruling out the use of wearable cameras.” The canteen was getting more crowded by the second.

“So there was no way to know what really happened. We had to wait for our suspect to come out of his coma.”

Chan emerged from his coma in a week and gave his statement. Still on suspension, I followed him to a noodle stall under a flyover in Kota Damansara.

“You shouldn’t be here.” Chan’s voice was barely audible above the late-night traffic.

By way of explanation and apology I started to sound technical. “Our police batons are military surplus; composite ceramics derived from nacre. Supposed to be unbreakable but the one issued to me was defective. Coconut shell filler expands when wet and becomes heavy. It was raining that night.”

“So you hit me too hard with it. Apology acknowledged but not accepted,” Chan shrugged and lit up a cigarette. “Corrupt cops like you always get greedy and screw up.”

“Thanks for the info,” I said and reached out to pat him on the shoulder.

I jabbed the syringe full of insulin into Chan’s shoulder. The hypoglycaemic shock set in within seconds and Chan passed out. He would enter a coma in four or five minutes, depending on his

weight and blood glucose level. But criminals never eat well. I left him slumped over the table. No matter, I still had time. I made my way to his car and pried open the boot where I found seven test tubes of illicit Conosulan.

“We are still in this together,” Sierra said in the hospital canteen.

She never smoked and now her hands trembled when she placed a red packet of cigarettes, Koh Samurai, on the table. Was it her way of sending a not-too-subtle message to me?

“Where is Chan’s stash? It’s vital evidence but you were acting off-duty.” Sierra asked.

“I handed it over to someone who knows someone who knows some other people. Smugglers’ networks are great hiding places.” I described a dendritic pattern on the chrome table top.

Sierra sighed and crushed the packet. “Internal Affairs made me talk to you. They thought since we were partners we were also friends. Now it looks like we’ll be repeating this conversation - again.”

As I stood up to leave, Sierra handed me a stick-on tattoo.

“You’ll need this for your next appointment in the back lane.” Sierra said with a patience that bordered on kindness. “Chan really hit you hard on the head that night.”

“I would remember—”

“Do you think only drugs and antiviral treatments can be synthesized?” interrupted Sierra. She tapped her left earlobe, signalling to an unseen audience monitoring our meeting. “Restart entire Interrogation Simulation Sub-Routine.”

The canteen noises subsided as my field of vision darkened. My suspension was far from over.



ABOUT THE AUTHOR

Lee Ee Leen was born in London, UK. She graduated from Royal Holloway College, University of London, with an MA in English Literature. Her short fiction and reviews have been published by Mammoth Books UK, Monsoon Books Singapore, Solarwrym Press Australia, Esquire Magazine, Intellect UK and Fox Spirit Books UK. Her debut collection of short horror fiction, *13 Moons*, was published by Fixi Novo in 2014. She tweets at @EeleenLee.

ASK ME ANYTHING: CANCER RESEARCH MALAYSIA

On the 18th of August to the 5th of September 2017, an Ask Me Anything (AMA) session with Prof Dr Cheong Sok Ching and Dr Muhammad Mamduh Zabidi of Cancer Research Malaysia (CRM) was organised on the Scientific Malaysian online platform.



Prof Cheong (SC) is the Senior Group Leader of the Head and Neck Cancer research team in CRM with 20 years of experience in molecular and cell biology. Her research team is working on the development of novel therapeutics for the treatment of head and neck cancers. She is also an adjunct professor at the Faculty of Dentistry at the University of Malaya.

Dr Muhammad Manduh (MZ) is a postdoctoral scientist in the Breast Cancer research team at CRM. Hailed from Bagan Datok, Perak, he obtained his degree in Biochemistry from Purdue University, USA, before returning to work with CRM while completing his Master's degree in Molecular Medicine at the University of Malaya. He later moved to Austria to pursue a doctoral degree in computational biology before returning to CRM.

During this 2-week session, members of Scientific Malaysian had the opportunity to post any question to these CRM scientists. Below are some of the questions posted:

Q1

What are the advantages of using zebrafish phenotype screening approach for drug discovery?

SC:

Phenotypic screens could afford us an opportunity to identify and prioritise compounds that can target a defined cancer pathway. This is because there is significant overlap between embryogenesis signaling pathways and those involved in cancer development, which would be manifested in a phenotypic observation when an active compound inhibits these pathways during the screening process.

One unique attribute of

using zebrafish is the large number of embryos that can be obtained from a single mating, and this can then be arrayed readily into multi-well plates for the testing of natural products, allowing a moderate to high-throughput screenings. Furthermore, because the zebrafish embryo is transparent, changes during development can be imaged directly and in real-time. In addition, this approach enriches the screen for potential drug leads

that are cell-permeable, bioavailable, and feasible for oral administration and gastrointestinal absorption, and those that exhibit favourable pharmacodynamics profile which increases the possibility of these compounds to advance in the drug development pipeline.

For more information about the use of zebrafish in drug discovery, we have recently published a review on this topic [1].



Q2

Do you think current models of cancer (e.g: immortal cell lines, mouse models that are bred to develop similar cancers that manifest in humans) are effective and suitable for research? What models do you ideally hope to see develop in the future, or what models are currently being developed that you hope to improve on?

SC:

Cancer models will remain as what they are – models, which means that they are not and will not be identical to actual patients that we, for ethical reasons cannot experiment on. The advantages of cancer cell lines are that they are simple and relatively cheap to set up. Genomics studies have demonstrated that generally, cancer cell lines carry key genetic alterations that recapitulate the cancer from which they are derived. Whilst there is in excess of 1,000 cancer cell lines, we need to choose cell line models carefully based on the biological questions. The ability to catalogue the genomics of cancer cell lines has enabled us to model drug response (see references [2] and [3]).

Other more complex models such as patient-derived tumour xenografts contains other cell types within the tumour and therefore enables the testing of drugs in the presence of the tumour microenvironment. These involve more complex ethical considerations, resources and expertise. There are efforts from the ethical point of view to replace animal models with animal-free ones, and the balance we are trying to achieve is to achieve the best representation of the disease and ensuring humane use of any animal-based models.

Q3

Are there any new ways to treat cancer that do not involve non-specific methods like chemotherapy that harm non-cancerous cells and that might also give rise to other cancers? How soon do you think these might be available for patients, and what might they involve?

SC:

Drugs that are more specific are currently already used in the clinic and there are intense efforts to continue developing such drugs by understanding the critical genes that drive certain cancers. For example, Cetuximab that targets the epidermal factor receptor (EGFR) is already

approved for several types of cancers including lung cancer and head and neck cancers. While targeted therapy does not give rise to cancers directly, they may selectively eliminate cancer cells containing the genetic alterations it targets, and therefore could provide opportunities for cancer

cells not containing the genetic mutation to flourish which would manifest as a relapse of the cancer. We have recently developed a bioinformatics tool that enables our research to identify targeted therapies that will work best for a defined genetic signature of cancer.

Q4

Referring to the current trend of cancer treatment based on precision medicine and cancer immunotherapy - does the infrastructure and regulations in Malaysia and/or Asia prepare us for such treatment?

SC:

Yes, infrastructure for cell-based cancer immunotherapy exists in Asia. However, these are not routinely available in treatment centres but instead are tightly regulated and well-

controlled facilities. In Malaysia, the framework to regulate these is still in development and these facilities are required to be inspected by the National Pharmaceutical Regulatory Agency (NPRA), Ministry

of Health. To know more about the different types of therapies and the guidelines on regulation please visit their website [4].



Q5

What are the difficulties/setbacks in conducting cancer research in Malaysia, and what are your proposed solutions?

SC:

Having conducted research in Malaysia, the UK and USA, what I miss when working in Malaysia is the openness in discussing science with no inhibition or the fear of sounding silly. The challenges that I have faced include having to face steep learning curves on almost every project we embark on as there isn't an expert you can run to, to simply ask "show me how

you do it" or core facilities that has perfected specific techniques. Science is moving at an incredible speed therefore, it counts to be able to leverage on each other's expertise and experience. One of the ways I try to overcome this is to network extensively and learn about what others do, even though these may not be directly applicable to you today,

but you'll never know when information or contacts come in handy. Addressing the challenge in creating an environment to discuss science openly, I think this will take time, but starting to talk about our work openly (and not worrying that I sound silly!) would hopefully motivate others to do so.

MZ:

Previously when I first started, the "can-do" attitude not just in cancer research but in science in general in Malaysia was severely lacking. However, the Malaysia Boleh spirit in research is slowly catching up to our attitude in other areas such as business or sports. We are indeed seeing more and more of

the younger generation interested in science in general. In line with this, I totally support the efforts of Scientific Malaysian to showcase our efforts and successes, both here and abroad.

The country is also running on a tight budget currently and this has affected

the funding climate for research in general. We indeed continuously explore other funding sources, especially from non-governmental as well as overseas.

Q6

There has been a lot of focus on genetic screens in the fight against cancer, but what about epigenetics? Is there any research on epigenetics in Cancer Research Malaysia, and what approach would you take to study the role of epigenetics in cancer?

MZ / SC:

Whilst we have studied the effects of HDAC inhibitors on oral cancer previously, epigenetics research is not currently the major focus of our lab.

Q7

Do you think that immunotherapy is the most promising treatment for cancer thus far? What are the limitations and where are we in terms of clinical trials and/or research on immunotherapy as cancer treatment in Malaysia?

SC:

I think it is important to put things into context. Whilst immunotherapy (at least in the form of checkpoint inhibitors) have seen a lot of successes in melanoma, renal cell carcinoma and head and neck cancers, some other cancers such as prostate cancer are less responsive to these therapies. The reason why immunotherapy is considered to be a promising treatment for cancer is because

patients who respond to these therapies have a durable response which has been rare with other forms of therapies where patients experience extended lifespan but ultimately succumb to the disease. The limitations of immunotherapy are very much dependent on the type of immunotherapy. One major challenge is there is currently no robust biomarker to identify the subset of patients who would most likely respond

to immunotherapy, furthermore, the currently available immunotherapies are very expensive. The head and neck cancer research team at Cancer Research Malaysia has developed a peptide vaccine that is currently in preclinical testing. We are raising funds to conduct toxicity studies before these can move into first-in-man clinical trials.

MZ:

Immunotherapy is indeed a viable treatment option for cancer treatment as SC have noted above. Additionally, the response of tumors towards immunotherapy has been thought to be related to how many mutations they carry. Indeed, cancers with high mutation rates such as melanoma have shown higher success with immunotherapy. Whether cancers that carry 'less' mutation, such as breast cancers,

would also be successful in immunotherapy is currently being investigated by researchers worldwide. Interestingly, we have discovered that a group of Malaysian breast cancers carry a genomic deletion that is associated with an increased immune response. These lines of evidence hint that this group of patients would be more amenable to immunotherapy. We are currently determining the

genomic characteristics of these tumors (especially how much mutation it carries). Additionally, we are also determining the immune and transcriptomic characteristics of these tumors. We hope that this effort would be able to tell us whether immunotherapy would be a viable treatment option for breast cancers that carry these genomic deletions.

For the full AMA session (including some great answers by other members of our network), please visit www.scientificmalaysian.com/AMA-CRM. We thank Cancer Research Malaysia for having this interactive Q&A session with members of the Scientific Malaysian network.

References:

- [1] <https://www.ncbi.nlm.nih.gov/pubmed/25348017>
- [2] <https://www.ncbi.nlm.nih.gov/pubmed/22460902>
- [3] <https://www.ncbi.nlm.nih.gov/pubmed/22460905>
- [4] <https://npra.moh.gov.my>
- [5] Cancer Research Malaysia – official website:
www.cancerresearch.my/

PROJECT COLLAB



A SCIENTIFIC MALAYSIAN PROJECT

Project Collab (www.scientificmalaysian.com/project-collab/) is a project by Scientific Malaysian, with the aim of fostering research collaborations between scientific researchers in Malaysia and abroad. As part of this project, we compile the research profiles of participating scientists and their contact details into a directory style listing – to make it easier for other researchers to browse or search for someone to collaborate with.

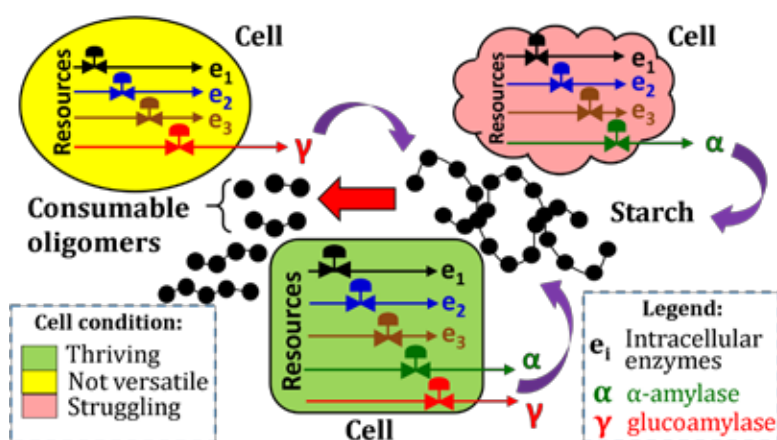
In this Issue, we highlight two of our most recent Project Collab profiles: Dr Ho Yong Kuen from Monash University Malaysia and Dr Marfiah Abdul Wahid from Universiti Teknologi MARA.

Distributed Multi-Scale Population Dynamics of Biological Dispersed Phase Systems – Numerical Techniques, Modelling, Optimisation and Control

The need to understand and manipulate chemical and biological systems at the micro-molecular level to obtain the desired macroscopic specifications calls for the development of multi-scale distributed approaches to model physical systems. Our research is motivated by the use of interesting mathematical ideas to solve important problems in chemical and biochemical engineering, and the main thrust of our work is fundamental. Our primary focus is in the advancement and application of population balances – a broad collective wealth of knowledge which deals with the evolutionary aspects of dispersed phase systems, both in time and perhaps also in

space. On-going projects revolve around the application areas of biomass depolymerization, complex biomass polymer fermentation, distributed emulsification dynamics and advanced control of biomedical devices.

We especially welcome potential collaborators in the field of lignocellulosic hydrolysis and fermentation who can provide us with distributed experimental data. We are also keen to work with experimentalists in protein engineering who might be interested in exploring new computational methods. For more information on our research findings, please refer to the google scholar website below.



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Micropollutant and Pathogen in water (WaterµPath)



Micropollutant and Pathogen in Water Research Group (WaterµPath) consists of seven (7) group members from various academic background and is led by Dr Marfiah Ab. Wahid from the Faculty of Civil Engineering, UiTM Shah Alam. Research interest of this group is on the monitoring and modeling of micropollutants including residual Personal Pharmaceutical Care Products (PPCPs), antibiotics, residual drugs and pathogenic bacteria in water environment. Our research on pathogenic bacteria is mainly focusing on their antibiotic resistance and survival in water and wastewater. Modeling of the dynamics and transport of residual micro pollutant and pathogens in river water is done using DEFLT software, whereas modeling of wastewater treatment plant is done using

Computational Fluid Dynamics (CFD) software. In order to reduce the risk to human and ecological health in water environment, several different treatment technologies are being tested in a laboratory scale including UV, membrane and Advanced Oxidation Processes (AOPs).

We are seeking collaborators with experience and interest in developing technology for the detection, measurement, treatment and modeling of micropollutant and pathogen in water environment.

We have designed and built prototype for on-site detector of pathogenic bacteria in water environment. Therefore, we are also seeking industries interested in helping us commercialise our prototype equipment.

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If you are a researcher and you would like us to highlight your research in Project Collab, visit:
<http://www.scientificmalaysian.com/project-collab>



GEMS FROM THE WEB

Running Hot & Cold Towards Ending AIDS

Two Malaysian AIDS Foundation Red Ribbon Youth Club advocates battled heat strokes and frostbites to inspire young Malaysians in the fight to end AIDS.



CLICK

<https://www.scientificmalaysian.com/2017/07/22/running-hot-cold-aids/>



UTM researcher wins Falling Walls Lab Malaysia 2017

Falling Walls Lab is a forum to promote interdisciplinary exchange of ideas between young academics, entrepreneurs and professionals of all fields. The Falling Walls Lab Malaysia event was held recently at the Asia Institute of Medical, Science and Technology (AIMST) University.



CLICK

<https://www.scientificmalaysian.com/2017/07/23/utm-researcher-wins-falling-walls-lab-malaysia-2017/>



GEMS FROM THE WEB

Science, technology and innovation community comes together at Newton-Ungku Omar Fund Open Day

Malaysia and the United Kingdom are extending the Newton-Ungku Omar Fund – a collaborative effort between the governments of both countries to promote science, technology and innovation collaboration, paving way for greater UK-Malaysia collaborations.



CLICK

<https://www.scientificmalaysian.com/2017/08/09/science-technology-innovation-community-newton-ungku-omar-fund-open-day/>



L'Oréal-UNESCO honours three outstanding Malaysian women scientists in annual award

Three women scientists in Malaysia has won the coveted L'Oréal-UNESCO For Women in Science Fellowship recently for research that tackles global challenges while potentially saving the lives of millions.



CLICK

<https://www.scientificmalaysian.com/2017/11/08/loreal-unesco-honours-outstanding-malaysian-women-scientists-annual-award/>



Citizen scientists discover 6 new species of beetles in Borneo

Taxon Expeditions has become the first organisation in the world to initiate field courses for citizen scientists in biodiversity hotspots, with the aim of discovering, describing, naming, and publishing new species under the slogan "You can be Darwin too".



CLICK

<https://www.scientificmalaysian.com/2017/12/15/citizen-scientists-discover-6-species-beetles-borneo/>

UK-Malaysia project generating bioelectricity from wastewater wins RM635,000 Newton Prize

A ground-breaking project that generates sustainable electricity from effluent waste processing has been awarded the Newton Prize worth £112,000 (approximately RM 635,000). This project, spearheaded by lead researchers from Malaysia and the United Kingdom, paves the way for greater access to energy supply particularly for the rural population in Malaysia.



CLICK

<https://www.scientificmalaysian.com/2017/12/15/uk-malaysia-project-generating-bioelectricity-wastewater-wins-rm635000-newton-prize/>



MOUNT MIRIAM
CANCER HOSPITAL

MOUNT MIRIAM CANCER HOSPITAL



A Little Hospital with a Big Mission

On a little hill, surrounded by a peaceful neighborhood, there located a mission-driven Cancer Hospital. This ***not-for-profit Catholic Hospital*** goes by the name ***Mount Miriam Cancer Hospital***, and it has been serving the community with love in cancer care for ***42 years, since 1976***. Mount Miriam Cancer Hospital is a single discipline cancer hospital, aspires to bring God's healing presence through the best and affordable cancer treatment, diagnosis and care. The Hospital shares the ***global mission of the Franciscan Missionaries of Divine Motherhood***, to provide high quality and compassionate care to all persons regardless of nationality, belief or financial status with love, respect and compassion.

Mount Miriam Cancer Hospital has always ensured that each patient is given the best possible treatment regardless of their financial status. It is their Vision that no cancer patient should be denied of treatment and care. The access to cancer treatment and management is based on need, not ability to afford the rising medical cost. Every year, the Hospital disburses an average of RM 1 million to help destitute cancer patients. More than 4,058 needy cancer patients have received financial assistance since 2010, with more than ***RM 7,065,733.00*** disbursed from the ***Hospital's Needy Cancer Patient Fund***.

SERVING WITH LOVE IN CANCER CARE

Despite its not-for-profit status, the Hospital strives to always improve its quality of treatment and care with the latest technology. The Hospital is currently the only hospital in the northern region of Malaysia to have the Tomotherapy radiation treatment machine. Tomotherapy is an advanced, image-guided, intensity-modulated radiation technology, which is effective to treat tiny and large tumours in multiple areas of the body. It is one of the most integrated, state-of-the-art system which can target cancer cells with high precision and minimizes damage to surrounding healthy tissues.

Next year, 2018, Mount Miriam Cancer Hospital will be acquiring the **Cyberknife** robotic-arm radio-surgery that allows painless non-invasive surgery on tumors that are too deep & too risky to be operated. The Cyberknife technology is extremely effective in treating moving tumours so it is highly recommended for cancer treatment in lung, spine and other organs such as brain, liver, pancreas and prostate, with highest level of safety, minimizing the adverse side effects and significantly shorten the treatment time.

The Hospital's mission can only be realized mainly with the help and financial support from the community. The community's donations, through personal and corporate companies, have allowed the Hospital to continue to fulfil their vision and mission. Today, Mount Miriam Cancer Hospital has six exceedingly committed and dedicated Clinical Oncologists and a team of **mission-driven individuals to serve in cancer care**. Along with the latest cancer treatment technologies and support from the community, the Hospital is defined as Penang's source of hope against the rising cancer epidemic.

For more information, please contact Mount Miriam Cancer Hospital, visit their official website at www.mountmiriam.com, or like them on Facebook at www.facebook.com/MountMiriam.



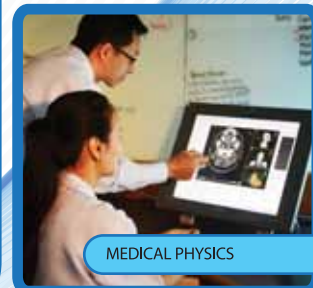
CYBERKNIFE



TOMOTHERAPY



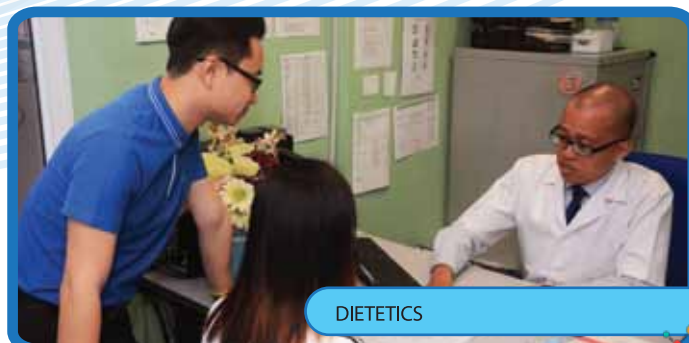
TOMOTHERAPY



MEDICAL PHYSICS



PASTORAL CARE



DIETETICS

Interested in joining the Scientific Malaysian team?

By being part of us, you will have the opportunity to enhance your skills and improve your CV by working flexibly and contributing remotely from wherever you are.

We are now seeking for enthusiastic and passionate volunteers to join our team for the following positions:

Web Developers

Role: Maintaining and adding new functionalities to our websites

Knowledge in Wordpress is essential

Magazine Illustrators or Designers

Role: Producing original illustrations/photos or layout for the magazine

Knowledge in Adobe InDesign or Photoshop is desirable

News Editors

Role: Writing short news reports on scientific research and development news in Malaysia, to attend/report on scientific events/conferences

Good writing and reporting skills are essential

Publicity Officers

Role: Promote awareness of Scientific Malaysian especially via social media, distributing SciMy digital magazine, liaising with relevant organisations

University Ambassadors

Role: Promote awareness of Scientific Malaysian at university campuses and research institutes locally (Malaysia) or abroad. May involve organising events (such as talks or discussion forums)

If you would like to contribute to Scientific Malaysian in other ways not mentioned above, please do contact us -

we are always looking forward to new ideas!

Contact us: team@scientificmalaysian.com

