WhitePaper

Oncology:
The Disease, the Dynamics and the Difficulties of Global Marketing Research

Ipsos Healthcare
The Healthcare Research Specialists

Introduction

The World Health Organisation estimates that the incidence of cancer could increase by 50% to 15 million cases by the year 2020. To put that into context, that is nearly the entire population of the Netherlands. Each and every one of us will likely be impacted by cancer in some way during our lifetime. Whether we experience the disease personally or know someone who has, cancer is a disease that we are all vaguely familiar with. Cancer was first discovered over 3,000 years ago and yet in some ways, it is as much a mystery today as it was then. While we have learned more about the causes and effective treatments, those who work in the oncology market are constantly faced with the challenges of this everchanging disease.

Cancer is complex as it is one term that encompasses many different diseases. There is no one cause for cancer, nor is there a single treatment protocol. The biology of cancer is also very complex, leading to an abundance of treatment approaches. Incidence and prevalence rates differ globally and treatment of the disease is managed by numerous physician specialties. In addition to these challenges, the ever changing nature of the disease and treatment approaches makes it challenging for marketers to remain current. In the last several years alone, we have seen the introduction of at least 10 new agents. We have also begun to understand the impact of biomarkers and have watched as physicians have changed their already complex and variable treatment approaches for diseases like NSCLC to an approach including maintenance therapy (although this term in itself is not consistently understood).

Oncology market researchers and marketers are faced with these challenges every day. This paper provides an introduction to this complex market and highlights some of the distinct challenges to help marketing professionals avoid pitfalls when marketing or conducting marketing research for oncology products globally.

Complexity of the disease area

Cancer and its causes

Simply put, cancer is ***the uncontrolled growth of poorly differentiated (nonfunctioning) cells.*** These cells' unchecked proliferation causes them to crowd out normal functioning cells, which eventually leads to death.

In philosophy, a **proximate cause** is an event which is *closest* to, or immediately responsible for causing, some observed result.

The proximate cause of cancer is **mutations** of genes that keep normal cellular growth regulated. Mutations in key regulatory genes alter the behaviour of cells and can potentially lead to the unregulated growth seen in cancer. **Tumor suppressor genes**, such as p53, regulate the cell cycle and thus function as a tumor suppressor involved in preventing cancer. When mutated, they lose this protective function.

Proto-oncogenes are genes that, when mutated, lead to unlimited cellular proliferation. It appears that a number of mutations are likely involved in cancer, and no single tumor relies on one mutation alone; it is the *accumulation* of such mutations that lead to cancer arising.

The fact that cancer is caused by mutations has many implications for its treatment. In a subsequent section of this paper, the link between mutations and treatment will be further examined.

However, mutations do not occur in a vacuum. Many factors can be involved in the mutation of genes, including:

- Lifestyle choices: Smoking and alcohol are chemical teratogens (chemicals that cause mutations), while a diet high in fat and a sedentary lifestyle are thought to increase body fat, which stores teratogenic chemicals
- Environmental factors: Radiation causes mutation directly by altering DNA; chemicals work to disrupt transcription and translation processes or act as endocrine disrupters that can stimulate cell growth
- Infectious agents: Viruses are thought to insert their own DNA into the nucleus, thus causing mutations. It is also thought that some bacterial infections may contribute to the proliferation of cancer cells.

Our bodies constantly fight off cancer. If damaged DNA is detected, our DNA repair mechanism typically restores the cell's genetic material. If this mechanism fails, and mutated cells are detected, our immune system destroys the damaged cells before they are able to multiply and spread. However, increasing age has an impact on these processes.

- Mutated DNA gets repaired before the cells have a chance to proliferate. These DNA repair mechanisms are less effective when older.
- In addition, it is thought that there is a delay in cancer development, meaning that someone who was exposed to cancer-causing agents as a young adult probably won't develop the disease until they are in their 60s or older. There's also a likely accumulative effect of lifetime exposure to certain chemicals.
- Lastly, the development of cancer usually requires multiple mutations to accumulate in the same cell; the likelihood of this occurring increases with age.

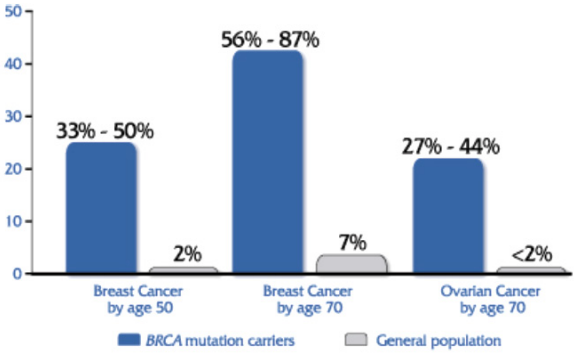
Some people inherit genes that predispose them to developing cancer; cancer is not inevitable, but it is much **more likely to develop** in people with so-called cancer genes than amongst the general population.

About **10-15% of cancers are hereditary,** depending on the type of cancer.

- Hereditary cancer tends to occur at an **earlier age** than the sporadic form of the same cancer, so screening is recommended

For example, mutations to tumor suppressor genes **BRCA1** and **BRCA2** predispose individuals who carry them to both **breast and ovarian cancers,** in addition to a higher risk of other tumors as well.

Rates of cancer in women w/BRCA mutation vs. general population



Source – http://www.bracnow.com/

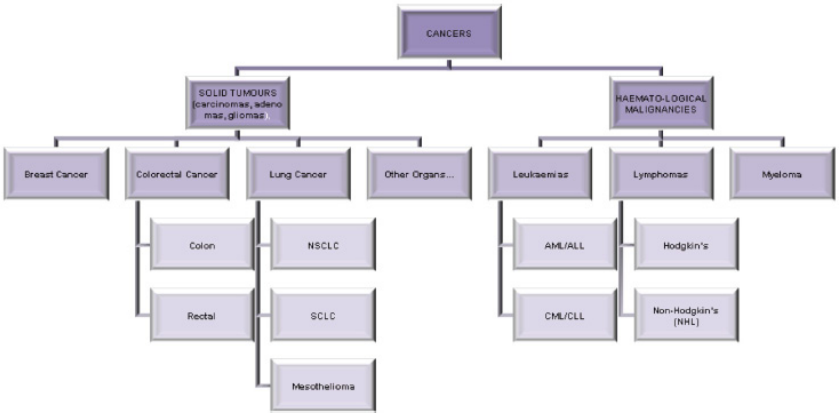
Cancer – One disease or many?

Cancer is a broad term used to encompass several malignant diseases. There are over 100 types of cancer, affecting various parts of the body.

Each type of cancer is unique with its own causes, symptoms, and methods of treatment. As with all groups of disease, some types of cancer are more common than others. Leukemias, Lymphomas and Myelomas are considered hematologic malignancies, while tumors affecting a specific organ are considered solid tumors.

Each cancer type is a disease within itself with unique biology, variable causes and treatment approaches.

Tumor Types and Sub-Types



Classifying cancer becomes more comprehensive when linking it to the specific organ or system in the human body affected. The following table shows this classification.

Brain Brain Stem Glioma Cerebellar Astrocytoma Cerebral Astrocytoma/Malignant Glioma Ependymoma Medulloblastoma Neuroblastoma Primary CNS Lymphoma Supratentorial Primitive Neuroectodermal Tumors and Pineoblastoma Visual Pathway, Hypothalamic Glioma	**Genitourinary/Germ Cell** Bladder Cancer Germ Cell Tumor Kidney (Renal Cell) Cancer Ovarian Germ Cell Tumor Penile Cancer Prostate Cancer Renal Pelvis and Ureter Cancer Testicular Cancer Urethral Cancer Wilms' Tumor	**Leukemia** Acute Lymphoblastic Leukemia Acute Myeloid Leukemia Chronic Lymphocytic Leukemia Chronic Myelogenous Leukemia Hairy Cell Leukemia Myelodysplastic Syndromes	**Skin Cancer** Kaposi's Sarcoma Melanoma Merkel Cell Carcinoma
Endocrine Adrenocortical Carcinoma Neuroendocrine tumors Parathyroid Cancer Pheochromocytoma Pituitary Tumor Thyroid Cancer	**Gynecologic** Cervical Cancer Endometrial Cancer Gestational Trophoblastic Tumor Ovarian Cancer Vaginal Cancer Vulvar Cancer	**Lung/Respiratory** Lung Cancer, Non-Small Cell Lung Cancer, Small Cell Malignant Mesothelioma Thymoma/Thymic Carcinoma	**Musculoskeletal (Sarcomas)** Ewing's Family of Tumors Osteosarcoma/Bone Histiocytoma Rhabdomyosarcoma Soft Tissue Sarcoma Uterine Sarcoma
Gastrointestinal Anal Cancer Bile Duct Cancer, Extrahepatic Carcinoid Tumor, Gastrointestinal Colon and Rectal Cancers Esophageal Cancer Gastric Cancer Gallbladder Cancer Liver Cancer Pancreatic Cancer Small Intestine Cancer	**Head and Neck** Hypopharyngeal Cancer Laryngeal Cancer Lip and Oral Cavity Cancer Melanoma, Intraocular Nasopharyngeal Cancer Oropharyngeal Cancer Paranasal Sinus & Nasal Cavity Cancer Retinoblastoma Salivary Gland Cancer	**Lymphoma/Plasma Cell** AIDS-Related Lymphoma Cutaneous T-Cell Lymphoma Hodgkin's Lymphoma Multiple Myeloma Mycosis Fungoides Non-Hodgkin's Lymphoma Sezary Syndrome Waldenström's Macroglobulinemia	**Breast Cancer** **Unknown Primary**

Once the cancer type is identified; however, there is still a need to understand the histology or cell type of the disease. This process allows pathologists and physicians to understand which cells are primarily being impacted within the cancer.

For example, in NSCLC alone, there are six different histological categories, some further divided into subcategories.

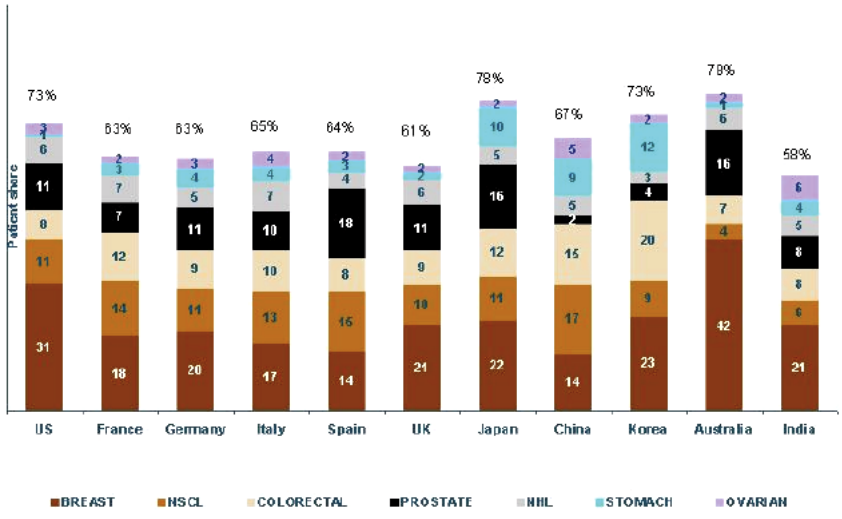
Selected NSCLC Histologic Subtypes from the WHO Classification of tumors	
I. Squamous cell carcinoma	A. Variants: papillary, clear cell, small cell, basaloid
II. Adenocarcinoma	A. Acinar B. Papillary C. Bronchioloalveolar carcinoma 1. Nonmucinous 2. Mucinous 3. Mixed mucinous and nonmucinous or indeterminate D. Solid adenocarcinoma with mucin formation E. Mixed F. Variants: well-differentiated fetal adenocarcinoma, mucinous ("colloid"), mucinous cystadenocarcinoma, signet ring, clear cell
III. Large cell carcinoma	A. Variants: large cell neuroendocrine carcinoma, combined large cell neuroendocrine carcinoma, basaloid carcinoma, lymphoepithelioma-like carcinoma, clear cell carcinoma, large cell carcinoma with rhabdoid phenotype
IV. Adenosquamous carcinoma	
V. Carcinomas with pleomorphic, sarcomatoid, or sarcomatous elements	
VI. Unclassified carcinoma (i.e., poorly differentiated)	

Historically, it was thought that the exact histology of NSCLC cancers was not an important factor in making treatment decisions. In recent years however, multiple clinical trials have demonstrated that the cell type can have a significant impact on the response rate to certain therapies. Pemetrexed, for example, has been shown to be more efficacious in tumors with a non-squamous histology. Bevacizumab is contra-indicated in squamous tumors, due to the high risk of fatal haemoptysis. Certain cell types, such as bronchiolaveolar carcinoma, are characterised by a higher incidence of EGFR mutations (see the section on biomarkers for further details on this mutation), which in turn has an impact on the response rate to EGFR inhibitors.

As cancer and treatment approaches vary significantly from country to country, oncology marketers need to understand not only the biology of each of the diseases but also the global differences.

Firstly, the distribution of drug-treated prevalence of cancer types shows significant differences across the globe. Due to higher incidence of smoking and pollution, as well as differences in diet and genetic predisposition, there are proportionally more gastric, liver and lung cancers in Asia. Breast cancer is the most common drug-treated tumor type in most countries; however, prostate and NSCLC show the highest drug-treated prevalence in Spain and China, respectively. There are proportionally fewer drug-treated patients with the top seven tumors in India, as oral cavity cancer has a very high prevalence linked to the chewing of tobacco and the smoking of bidis. When viewing cancer globally, it is important to understand these local and regional considerations that impact incidence, prevalence and ultimately treatment of cancer.

Global distribution of drug patients by tumor type (top 7) – 2009



Cancer – One treater or many?

When conducting global research with cancer treaters, many researchers may believe it would be safe to focus on Medical Oncologists. The reality is that many physician specialties treat cancer with drug therapy including surgeons and radiation oncologists. In the US, according to Ipsos Healthcare's annual market sizing study, there are seven specialties responsible for the anti-cancer drug treatment of 94% of existing cancer patients.

The specialties most involved in cancer drug treatment in the US are Medical Oncologists, Haematologists and Medical Oncologists/Haematologists. Med Onc/Hem specialty only became recognised by the AMA in the mid 90s but is now the fastest growing specialty in cancer treatment in the US. When comparing the US to Japan however, there is a stark difference in the physician specialties that treat cancer. Up until 2007, there was no "oncology" specialty. To this day, there are only 200 Oncologists in all of Japan. As such, the main cancer treaters in Japan are either surgeons or physician specialties that treat a specific area of the body. For example, gastric cancer is drug treated by surgeons as well as gastroenterologists.

Lack of Oncologists in Japan

	Japan	US	Ratio (%) Japan/US
Population	127 million	391 million	32
Total Doctors (2008)	286,699*3	632,818	45
General Internists	62,845*3	182,253	34
Medical Oncologist	**451***2	**10,598***1	**4.0**
Hematologist	1,867*3	6,303*1	30

Source:
*1 2009 ABMS Certificate Statistics, American Board of Medical Specialties
*2 The List of Certified Medical Oncologists (2010), Japanese Society of Medical Oncology
*3 Survey of Physicians, Dentists and Pharmacists (2008), Ministry of Health, Labour and Welfare

Treaters of cancer in Japan

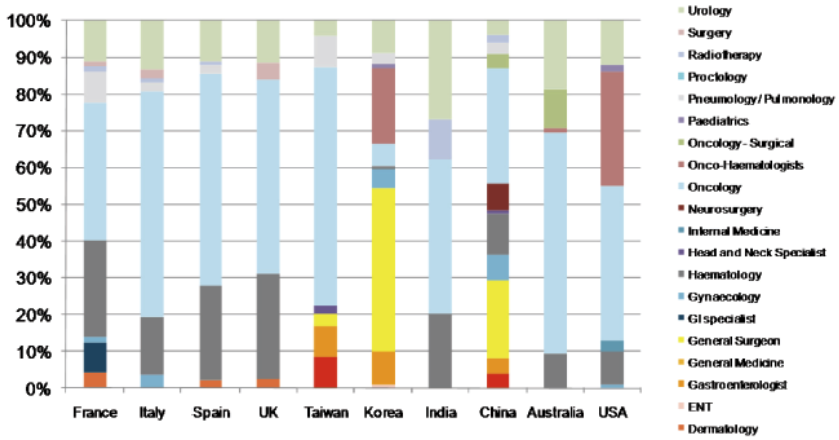
Gastric	GI surgeon 66%	GI internist 17%	General surgeon 13%
Colorectal	GI surgeon 73%	GI internist 11%	General surgeon 11%
Breast	Breast surgeon 80%	General surgeon 13%	GI surgeon 6%

Source – Ipsos Healthcare Japan "Japan Oncology Market Observer" 2009 Edition

Europe is somewhat of a hybrid of the US and Japan. In certain EU countries, medical oncologists are the main treaters, however, in Germany the main treating specialty is medical hem/onc. In France, pulmonologists are major drug treaters of lung cancer while in Germany, dermatologists treat melanoma.

Understanding the correct specialties to target and talk to globally can be as much of a challenge as understanding the nuances of cancer treatment.

Specialties by country – Overall



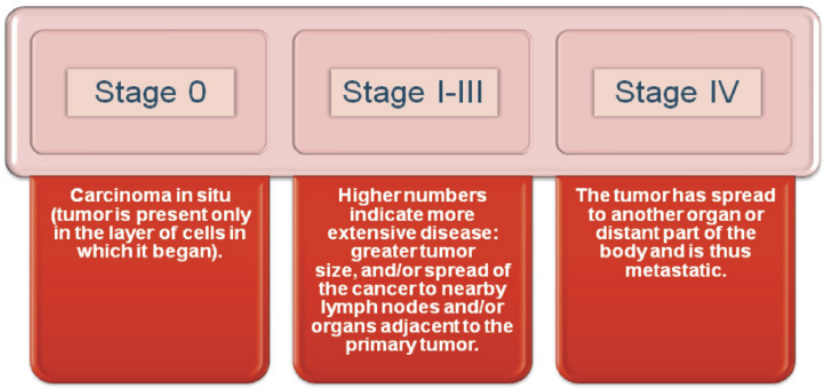
*Source – Ipsos Healthcare Global Oncology Monitor

Treatment challenges

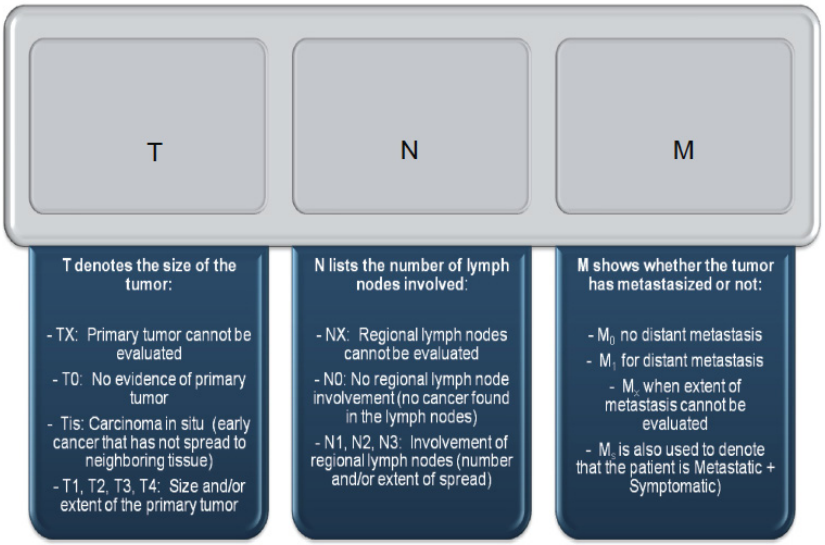
Cancer staging - One system or many?

In order to determine the proper treatment for cancer, it is critical to assess the extent of the disease. Several staging systems have been developed to do this.

Solid tumors are commonly staged by a Roman numeric system that is directly proportional to the extent of disease, with 0 being least extensive and IV being most extensive (metastatic) disease. However, Gliomas and other CNS diseases are solid tumors that are exceptions to this system.



The TNM system is also commonly used for solid tumors and provides more detailed information than the Roman numeric system.



As each tumor type has unique features, many staging systems have been developed to describe particular tumors.

The table below is a small sampling of some of the more common different solid tumor staging systems:

System	Tumors Used	Description
FIGO	Ovarian	**I** - Malignancy of one or both ovaries, without ascites; **II** - Malignancy of one or both ovaries, with pelvic extension and ascites **III** - Malignancy involves one/both ovaries, intraperitoneal metastases outside pelvis and/or positive retroperitoneal lymph nodes **IV** Involvement of one/both ovaries with metastases and histologically confirmed extension to pleural cavity or liver
CIN	Cervical	Cervical Intraepithelial Neoplasia – grading system for pre-Cancerous cervical lesions
Dukes	CRC	Stages A, B, C, and D roughly correspond with Stages I-IV.
Jewett-Whitmore	Prostate	Stages A and B Cancers are considered curable. Stages C and D are treatable, but their prognoses are discouraging.
Clark Level/ Breslow Depth	Melanoma	**Breslow thickness** is defined as the total vertical height of the melanoma, from the very top (called the "granular layer") to the area of deepest penetration in to the skin. **The Clark level** refers to how deep the tumor has penetrated into the layers of the skin.

Due to the more diffuse and 'dimensionless' nature of these malignancies, staging of haematological tumors is generally very different to staging of solid tumors.

The table below illustrates the staging system of a number of haematological cancer types.

System	Tumors Used	Description
Ann Arbor	NHL	I - NHL is limited to one lymph node group (e.g., neck, underarm, groin, etc.) above or below the diaphragm, or NHL is in an organ or site other than the lymph nodes (extranodal) but has not spread to other organs or lymph nodes. II - NHL is limited to two lymph node groups on the same side of the diaphragm, or NHL is limited to one extranodal organ and has spread to one or more lymph node groups on the same side of the diaphragm. III - NHL is in two lymph node groups, with/without partial involvement of an extranodal organ or site above and below the diaphragm. IV - NHL is extensive (diffuse) in one organ or site, with/without NHL in distant lymph nodes.
Rai	CLL	0 - The blood lymphocyte count is too high, usually defined as over 10,000 lymphocytes/mm^3 of blood. The lymph nodes, spleen, and liver are not enlarged and the red blood cell and platelet counts are near normal. I - Lymphocytosis plus enlarged lymph nodes. The spleen and liver are not enlarged and the red blood cell and platelet counts are near normal. II - Lymphocytosis plus an enlarged spleen (and possibly an enlarged liver), with or without enlarged lymph nodes. The red blood cell and platelet counts are near normal. III - Lymphocytosis plus anaemia, with or without enlarged lymph nodes, spleen, or liver. Platelet counts are near normal. IV - Lymphocytosis plus thrombocytopenia, with or without anaemia, enlarged lymph nodes, spleen, or liver.
Binet	CLL	A - <3 areas of lymphoid tissue are enlarged, with no anaemia or thrombocytopenia. B - 3 or more areas of lymphoid tissue are enlarged, with no anaemia or thrombocytopenia. C - Anaemia and/or thrombocytopenia are present.

Some haematological malignancies, such as AML and ALL, are not commonly staged.

Cancer treatment – Art or science?

Another significant challenge associated with cancer is that treatment is as much an art as it is a science. Even after the cell type, stage and grade have been determined, other variables such as the patient's general health, treatment history, preferences and support system, as well as the doctor's training, come into play to decide the appropriate treatment approach.

The table below illustrates the degree of fragmentation of late line treatment for ovarian cancer in the US.

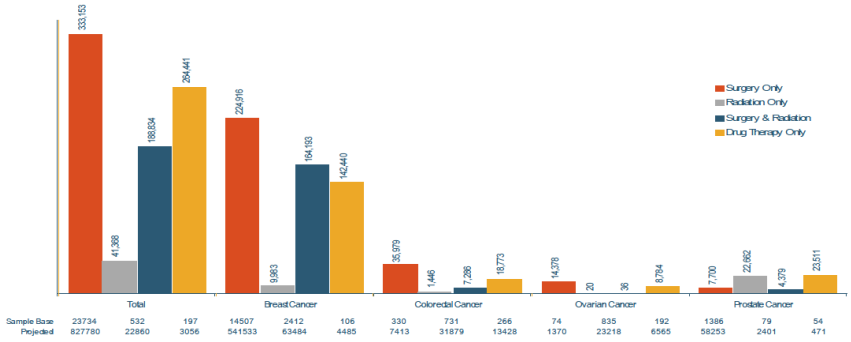
Treatment	% of Patients Receiving
PEG-Lipo Doxorubicin	23.8
Topotecan	12.9
Gemcitabine	7.5
Carboplatin/Paclitaxel	6.2
Carboplatin/Gemcitabine	5.9
Paclitaxel	4.6
Tamoxifen	4.2
Cisplatin/Gemcitabine	2.7
Bevacizumab	2.6
Carboplatin	2.2

*Source – Ipsos Healthcare US Oncology Monitor

Surgery and radiation are first options for many tumor types, including early stages of colorectal cancer, ovarian cancer and prostate cancer, while other cancers are treated with chemotherapy as a first line approach.

The table below shows the number of patients with specific cancer types receiving surgery, radiation and drug therapy in the US.

Adjuvant treatment modalities by tumor type



*Source – Ipsos Healthcare US Oncology Monitor

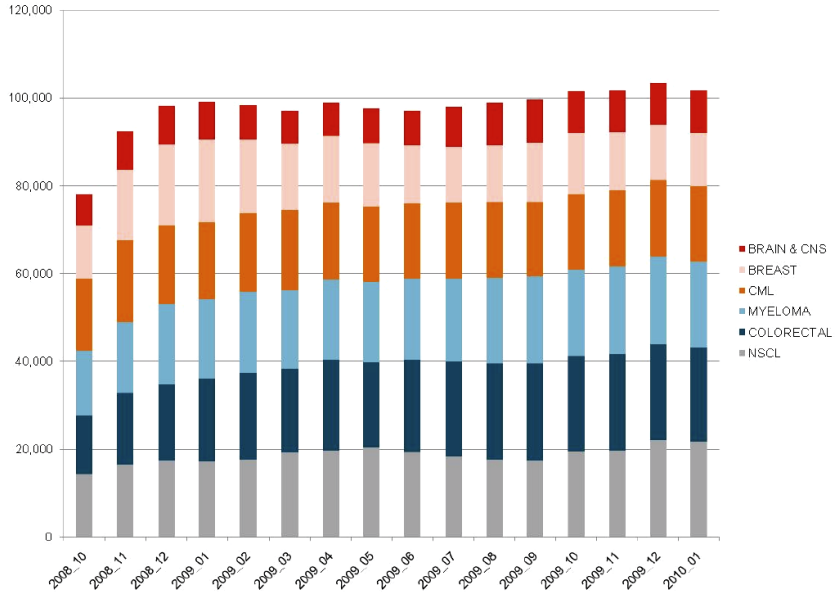
Cancer treatment - Oral versus IV

Even when specifically focusing on drug treatment, options include several different classes of drugs available in both oral as well as IV form.

The increasing trend towards introduction and use of oral agents has caused difficulty with respect to understanding compliance as well as with quantifying the proportion of anti-cancer drug treatment which is oral.

In just the past year, use of oral anti-cancer agents has increased from about 80,000 to 100,000 patients in the US in a typical month.

Patients treated with oral drugs by tumor type



*Source – Ipsos Healthcare US Oncology Monitor

The challenge becomes even more intense when you look at use of oral therapies globally.

Cancer treatment – Too many drugs or just enough?

Adding to the complexity of cancer treatment is the fact that more drugs are approved for cancer than any other single disease. In recent years, several drugs have been introduced that have changed certain cancer types to more of a chronic condition with longer survival rather than an acute disease with near immediate morbidity.

The table below highlights just some of the new agents which have become available in recent years. As a result, patients now have more options than ever before, though many unmet needs remain.

Drug	Drug class	Mode of action
Avastin (bevacizumad)	Monoclonal antibody	VEGF-inhibitor
Afinitor (everolimus)	Small molecule inhibitor	mTOR-inibitor
Arzerra (ofatumumab)	Monoclonal antibody	Anti-CD20 antibody
Erbitux (cetuximab)	Monoclonal antibody	EGFR-TKI
Folotyn (pralatrexate injection)	Antifolate	DHFR-inhibitor
Ixempra (ixabepilone)	Epothilone B analog	Microtubule stabilisation
Nexavar (sorafenib)	Small molecule inhibitor	Multi-RTK-inhibitor
Provenge (sipuleucel-T)	Therapeutic vaccine	Immunotherapy
Sutent (sinitinib malate)	Small molecule inhibitor	Multi-RTK-inhibitor
Tasigna (nilotinib)	Small molecule inhibitor	BCR-ABL-TKI
Torisel (temsirolimus)	Small molecule inhibitor	mTOR-inhibitor
Tykerb (lapatinib)	Small molecule inhibitor	EGFR/HER2-TKI
Vectibix (panitumumab)	Monoclonal antibody	EGFR-TKI
Votrient (pazopanib tablets)	Small molecule inhibitor	Multi-RTK-inhibitor

As illustrated by the modes of action listed above, many pharmaceutical companies have begun developing therapies that specifically counteract the results of the cell mutations which cause cancer.

Biomarkers - A further complication or a valuable predictor?

Owing to the more targeted approach of anti-cancer drug therapy described above, the introduction and expansion of biologic markers to assess a patient's ability to respond to treatment are increasingly driving the development of these drugs.

The availability of molecules that are only effective or approved for patients with a positive biologic test result has further increased the difficulty of understanding cancer treatment. The table below shows just some of the biologic markers that patients are being tested for.

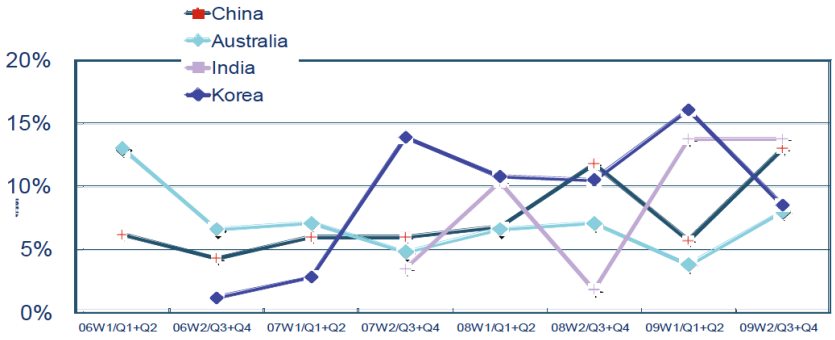
Breast	• **ER/PR Status** • **Her2 neu** • **BRCA**
Colorectal, NSCLC and Head and Neck Cancer	• **EGFR** • **KRAS**
CML	• **T315i Mutation** • **Philadelphia Chromosome**
Melanoma	• **BRAF**

One of the challenges with tumor marker testing is determining just how strongly linked a positive test result is to the efficacy of certain targeted treatments.

Further complicating the picture is the fact that certain tests aim to measure the degree of over-expression of an oncoprotein, whereas other tests aim to determine whether the actual oncogene is mutated or not. Different oncoproteins are often closely linked parts of the same cellular pathway, which means that knowing the mutation status of one oncogene is not necessarily enough to predict the response of therapies targeting that gene's protein.

A good example is the *EGFR/KRAS* duo of oncogenes: mutated *EGFR* genes may (but don't always) lead to EGFR over-expression, which generally increases the response rate of EGFR inhibitors. However, if the KRAS gene is also mutated, the response rate is greatly reduced.

The chart below shows that the uptake of testing – in this case for EGFR overexpression in NSCLC tumors - varies somewhat between countries. Furthermore, it is clear that the initial uptake of such tests has slowed down, and in some cases, been reversed. This is likely a reflection of the fact that such tests are currently only deemed useful for patients for which available first-line options no longer work, as well as the usefulness of over-expression testing in predicting a response is still being a widely debated issue.

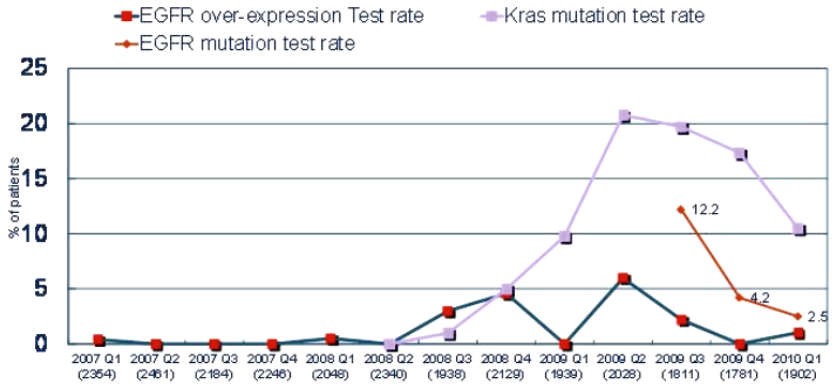


Source – Ipsos Healthcare Asia Pacific Oncology Monitor

Another tumor type in which EGFR/KRAS testing is considered is Colorectal cancers. The chart below compares EGFR over-expression, EGFR mutation and KRAS mutation testing rates in Australia. Again, the initial uptake of EGFR over-expression was reversed and largely replaced by KRAS mutation testing.

Furthermore, both KRAS and EGFR mutation testing appear to be on the decline in metastatic CRC - another example of the fact that tumor marker testing is far from being an integral part of all physicians' treatment approaches.

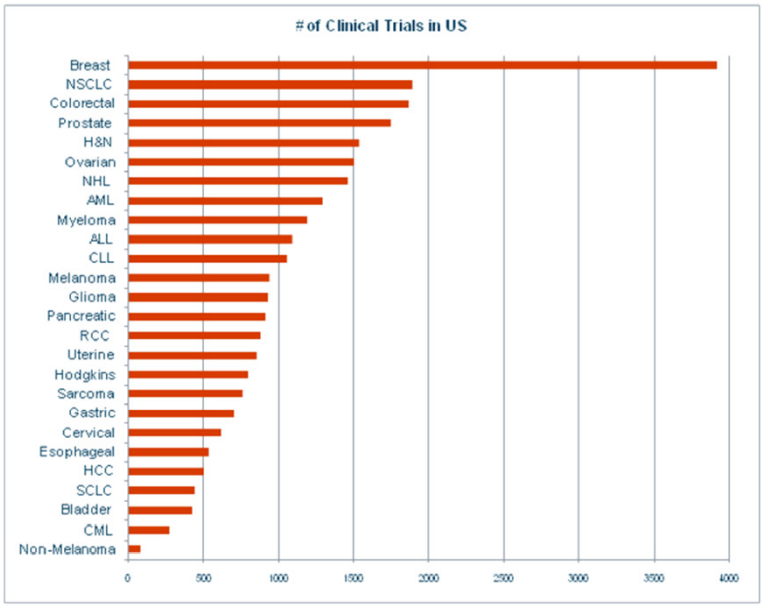
mCRC KRAS mutation testing vs. EGFR Over-expression Testing vs. EGFR mutation testing in Australia



Source – Ipsos Healthcare Asia Pacific Oncology Monitor

More drugs in development

There are well over 300 approved medications for the treatment of cancer. Yet in the US alone right now, there are currently over 4,000 clinical trials.



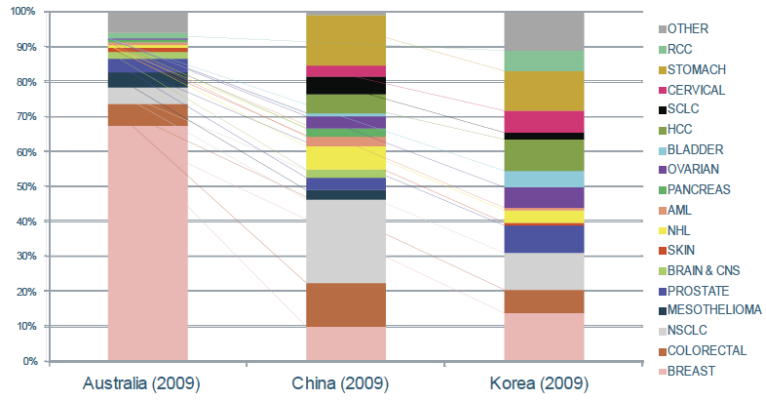
Source: http://clinicaltrials.gov/ct2/search

When reviewing the Ipsos Healthcare Global Oncology Monitor to compare clinical trials, the chart below illustrates that the distribution of ongoing clinical trials by tumor type is highly variable between countries.

Even more so than in the US, breast cancer accounts for the majority of trials in Australia.

In China and Korea on the other hand, the high rate of trials involving HCC and gastric cancer should be noted. This is reflective of the relatively high prevalence of those diseases in both Asian countries. The actual drugs being studied vary significantly between these countries too. Just 13% of all patients on a trial in China are taking targeted therapies, compared to 18% in Korea and 26% in Australia (note that this excludes patients in the control arm of trials).

Trial patients: distribution by tumor types in APAC



*Source – Ipsos Healthcare Asia Pacific Oncology Monitor

The challenge of understanding line extensions

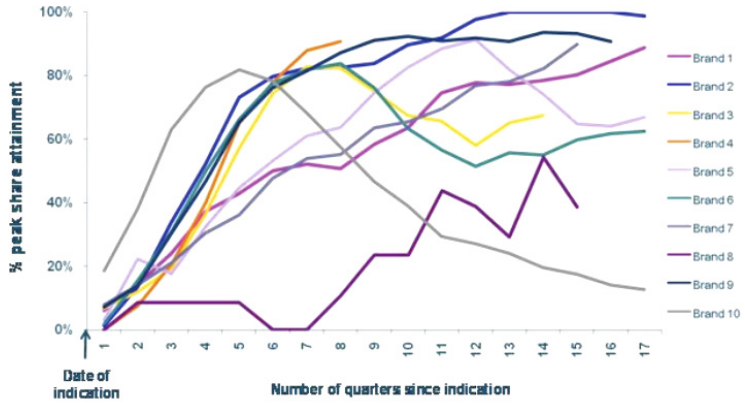
An additional complexity in the world of oncology is off-label use and the impact it has on a forecaster's ability to understand uptake of existing drugs for new indications or new lines of therapy. Historically, oncology compounds have experienced significantly faster prescribing uptake post approval than other therapeutic launches. Factors contributing to this trend may include:

- Extremely high pre-launch awareness levels
- A larger portion of physicians who are willing to be early adopters
- Compounds that fulfil a significant unmet need with regards to efficacy, tolerability, safety or all these parameters

Due to limited physician-level and patient-level secondary data, it can be difficult to analyse and segment prescribing patterns for individual indications of newly launched oncology products in granular detail.

The table below was compiled as a result of substantial analyses from the US Oncology Monitor and illustrates how different cancer products reach their peak share at different times after the indication.

First indication uptake – percent of peak share attainment



Note: end of line indicates 2009 Q4 data

*Source – Ipsos Healthcare Line Extension Uptake Forecasting Model

Changes in treatment approaches (sequential and maintenance therapy)

Complicating this matter further are paradigm shifts in the treatment approach. We first saw this several years ago in breast cancer when sequential therapy was introduced. Over the last two years, we have seen a shift to Maintenance therapy in NSCLC. Alimta® was the first chemotherapeutic agent to receive approval for a Maintenance indication in non small cell lung cancer. Creation of new treatment approaches and new maintenance indications will cause confusion at least initially as many physicians do not use the term "Maintenance". Rather, they describe it as treating with the same agent(s) until disease progression.

The following excerpts regarding Maintenance treatment come from a 2009 interview with a Key Opinion Leader in Europe.

Interviewer: "You touched on progression-free survival, which I know is a 'hot' subject of yours. Would you like to reflect on that a little?

KOL: "I mentioned progression-free survival (PFS) in terms of the maintenance use of pemetrexed, which is a new entity in non-small cell lung cancer.

Previously, we didn't actually think maintenance was of any value. Many people would stop - like ASCO regulations stop - treatment after six courses or, particularly in Europe, four courses of chemotherapy. So maintenance is new. And we know that the primary endpoints of both Alimta® and the Tarceva® SATURN maintenance trial both achieved their primary endpoint, which was PFS. So I would guess they would both be licensed on the fact they achieve their PFS. The question I always pose is: How useful is that to the patient? Not: How useful that is to the doctor? Who's going to prescribe the drug and perhaps give some reimbursement money back? And that is an open question in my mind, because what I'd like to see, of course, is overall survival benefit. And a lot of studies which show PFS differences do not go on to show any overall survival benefit".

Interviewer: "You've touched on maintenance therapy a couple of times, is there such a thing as maintenance therapy in lung cancer?"

KOL: "There was no evidence until recently that starting a treatment, either the same treatment or a new treatment, immediately after response or stabilisation of disease with the initial chemotherapy could give a benefit. The general approach was patients that had their first-line treatment and if they responded or had stable disease, they had a break from this treatment and then received second-line therapy when, as usual, the patient's disease progressed. So everyone gets wound up about maintenance consolidation, early secondline. In my mind I don't really care, and I'm not sure the patients would care either. All they want is a treatment programme which results in longer survival. And we may hear that maintenance treatment either with pemetrexed or, in the SATURN trial, with Tarceva® achieves that aim. We know it's achieved the progression-free survival goal, and some patients would want to take the drug on the basis of PFS. I would, as I mentioned earlier, like to see an overall survival as well."

Closing

This paper is a top line view of some of the clinical challenges that oncology researchers need to be aware of in order to better understand the specifics of oncology which can enhance market research.

Other main challenges of the cancer market include; the influence of patients and their caregivers, challenges with pricing and reimbursement, different approval processes and global healthcare systems, future trends with biosimilars, as well as the challenge with access to sales data for oncology products.

This paper will be the first in a series with future papers focusing on some of these challenges as well as Ipsos Healthcare's approach to ensuring that these do not impact the quality of Oncology marketing research.

Contact

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About Ipsos Healthcare

Ipsos Healthcare is a global business division focusing on research in the pharmaceutical, bio-tech, and medical device markets. It is also the leading provider of global syndicated therapy monitor data. Operating in over 40 countries, the team of 600 pharmaceutical market research experts, marketers and client-side brand-builders focus on delivering outcome-oriented research for its clients. Drawing from a broad range of qualitative and quantitative techniques, Ipsos Healthcare offers custom and syndicated research programmes to evaluate motivations, experiences, interactions and influence of stakeholders forming the multi-customer markets which increasingly drive business success in the healthcare industry.

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