Marisa Cordella · Aldo Poiani

Behavioural Oncology

Psychological, Communicative, and Social Dimensions

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For Eddita

Preface

Today, when many diseases seem to be on the retreat, thanks to better education, hygiene, improved living standards and medical treatments, cancer still remains a serious problem around the world. It is often the case that a cancer diagnosis is received with great apprehension by patients and family, almost as if it was the closest medical equivalent to a death sentence. But such apprehension is not always medically justified. There is much we can do to prevent cancer in the first place and also to help with the effectiveness of treatment once cancer has been diagnosed. We can indeed be proactive, as far as preventing the occurrence, progression or recurrence of cancer is concerned. Through our behavior, mental activity can directly or indirectly affect some of the physiological and molecular processes that may interfere with cancer development and/or progression. Behaviour can achieve this in very many ways, from the regulation of our alimentary and exercise habits that may boost our immune system, to preventing contact with potentially carcinogenic compounds, radiation and infectious agents, but also through the modulation of the nervous-endocrine-immune system that can affect the probability of our cells undergoing cancerous mutations or epigenetic alterations. Important to this mind–cancer interface is the biology and psychology of distress and relief from distress.

The human mind is a complex product of our organic self in interaction with the outside world, including other people and their own minds. It is this ultimate anchoring of the mind in our biology that allows our psychology to aid in the fight against cancer. If chronic psychological stress, perhaps leading to major depression, can affect the probability of cells to experience cancerous mutations, then being able to effectively cope with stress can help the individual, at least to a degree, in the prevention of some types of cancer. Social interactions are also a great aid in our struggle against disease. We benefit from the material and psychological aid received from others, starting with family and friends but also from technically trained health professionals: doctors and nurses. Such benefits are mediated by an effective communication between all people concerned, achieved through the use of both linguistic and paralinguistic means. Unless the discourse of patients is heard and understood there is little hope to improve our ability to provide effective care.

Health professionals not only do provide support through pharmaceutical and surgical interventions but also their psychological counseling is often equally important. Such counseling should also embrace the use of complementary psychological therapies of proven effectiveness that can help the patient recover physically, emotionally and also cognitively from a close encounter with cancer.

Psychological support for cancer patients remains important throughout the full course of the disease. This is also true for terminal patients, who may rightly wish for a smooth and gentle transition to an end of life. But cancer survivors also deserve special attention. A considerable proportion of them may still have many years of life ahead and the quality of that life is of central concern to them and their family. Re-incorporation into a useful role in society, job opportunities, overcoming stigma, maintaining a healthy physical condition, a supportive social network and being able to meaningfully integrate the cancer experience into a new form of self are important challenges that survivors are likely to confront and can successfully overcome. But survivors should not be expected to meet those challenges on their own, help is often required.

As we improve our understanding of the biology of cancer we must also improve our comprehension of the psychology of cancer. We will be aided in this process if we maintain an open mind about the possibilities of our capacity to understand cancer and its psychological underpinnings, whilst subjecting our ideas to the strict requirements of the scientific method. But research into behavioural oncology is more likely to make a valuable contribution when the task of answering technical questions is also undertaken with a degree of much needed empathy. No matter how narrowly we may wish to focus on the body in need of treatment, a cancer patient is and will always remain first and above all a person.

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Many thanks to David Hahn for permission to reproduce the text from his blog *Chronicles of a cancer patient* (Chap. 6). In the same chapter we also quote an extensive text from another blog authored by Kimberly Begay: *A different kind of journey*…. We tried to contact Kimberly in late 2012, only to learn that she had passed away on 25 November 2012. We can only hope that her words may remain a testimonial of her own journey. We also thank Carol Pepe from the *Trinitas Regional Medical Center* in New Jersey, USA, for providing additional information about *Inker* (Chap. 8).

But in our acknowledgments, we cannot overlook all those cancer patients from around the world who took part in the very many studies cited in this book. Terminal cancer patients are particularly deserving of praise. By learning from them we may perhaps improve the ways we relate to future cancer patients and help them make their journey a more bearable one.

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Contents

Chapter 1
Introduction

Cancer is a collective term for a group of diseases that are not easy to define due to their multicausal nature and the diversity of processes that can lead to their development in different parts of the body. Yet, there is a basic characteristic common to all varieties of cancer that is clearly rendered in the National Cancer Institute (2010) definition: "Cancer is a term used for diseases in which abnormal cells divide without control and are able to invade other tissues". Such process of invasion of other areas of the body by cancer cells is called *metastasis*. That is, cancerous cells have typically lost their ability to operate as functional components of a tissue, becoming rogue entities that proliferate out of control. More than 100 types of cancer have been already identified, but the list is likely to continue to grow in the future.

Why would cells which are part of our body start a process that could potentially damage, sometimes quite seriously, that same body? Is this seemingly paradoxical process a result of recent changes in our environment, or perhaps changes in our habits, or simply the result of molecular and cellular mechanisms that are prone to errors? Or is it something much more ancient that needs to be explained by referring to our evolution as a species?

From an evolutionary perspective, cancer is not a new phenomenon. The oldest reported fossil evidence of tumours in vertebrates has been found in Lower Carboniferous fish about 300 million years old and dinosaurs 70–195 million years old (Rothschild et al. 1999; Weiss 2000; Rothschild et al. 2003, see Capasso 2005 for a recent review of comparative and archaeological evidence of cancer). Tumours are formed by localised abnormal cell growth, and they may be either benign, or pre-cancerous, or malignant (cancerous). It is possible to detect evidence of metastatic tumours in fossils when the cancer affects bone structures, as these can leave an identifiable fossil imprint (Weiss 2000; Halperin 2004; Roberts 2005).

Although the identification of a bone abnormality with a metastatic tumour is not straightforward, being open to errors (David and Zimmerman 2010) archaeological data do suggest that we humans have been affected by various forms of cancer for a considerable part of our evolution. The apparent presence of lymphoma has been described in fossils of *Homo erectus*, one of our extinct close relatives—although

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the evidence is disputed (David and Zimmerman 2010)—and some ambiguous evidence of meningioma is available from *Homo neanderthalensis* (Capasso 2005). But more recent remains of modern humans (*Homo sapiens*) provide more compelling historical evidence of cancer. Such increased evidence of cancer in modern humans is not just the result of larger sample sizes available, because relative to the total number of specimens examined, the frequency of bone neoplasms (localised abnormal proliferation of cells) in modern humans is much higher than in any other vertebrate (Capasso 2005).

We also possess historical written records, especially from ancient Greece, that describe cases of cancer, breast cancer in particular (David and Zimmerman 2010), and up to 5,000-year-old medical records from Egyptian papyri are also consistent with what we would describe as breast tumours (Manton et al. 2009). More direct signs of cancer in antiquity include evidence of melanoma found in a Peruvian mummy about 2,400 years old and bone evidence of cancer coming from Bronze Age samples about 4,000 years old (Manton et al. 2009). Schultz et al. (2007) have recently described a 2,700-year-old case of metastasising prostate carcinoma in the skeleton of a Scythian King from Siberia, whereas evidence of nasopharyngeal carcinoma comes from 3,000- to 4,500-year-old skulls from both Mesopotamia and Egypt (Weiss 2000). Additional reports of metastatic carcinoma come from a 1,360 (or less)-year-old Anglo-Saxon burial from England (Manchester 1983) and a 900–1,100-year-old preincaic burial from Peru (Baraybar and Shimada 2005). Various Chilean mummies have provided 800–1,700-year-old evidence of ancient cases of lipoma and rhabdomyosarcoma (David and Zimmerman 2010 and references therein). In addition, Fornaciari (1993) recorded a case of colorectal adenocarcinoma in a 518-year-old mummy from Italy. Molecular genetic analyses based on microsatellite markers associated with the *MLH1* gene, that controls mechanisms of repair of mismatched DNA, suggest that two mutations that frequently occur among Finnish individuals suffering from hereditary nonpolyposis colorectal cancer first appeared in Finland between 1,075 and 400 years ago (mutation 1) and 125 and 525 years ago (mutation 2) (Moisio et al. 1996).

Consistent with the general trend towards increased cases of cancer in humans, archaeological evidence suggests that in Europe cancer prevalence has been steadily increasing from pre-Christian times (13.6 % of all reported cases of cancer from bone samples) to the first millennium of the current era (38.6 %) to the second millennium (47.7 %), a pattern that could be associated with increased lifespan, urbanisation and population densities (Capasso 2005). In addition, there may be also an effect of increased time spent indoors that exposes the individual to various carcinogenic pollutants and increased chances of transmission of viruses and other cancer-causing pathogens in large urban settings. Increased lifespan will compound the effect of both factors on cancer, apart from having its own specific effect as explained below.

Those recent historical trends notwithstanding, it is clear from this review that cancer has had a presence in humans for a very long time, and environmental/cultural factors, such as smoking, that promote development of cancer in modern societies, simply trigger biological processes that are deeply ingrained in our reality as evolved organisms. From an evolutionary perspective, most of the current structures and functions of our body are a result of selective and other processes that occurred

Table 1.1 Reproductive contrasts between women of two ethnic groups

Reproductive variable	Hunter-gatherers	Americans
Age at menarche	16.1[a]	12.5
Age at first birth	19.5	26
Menarche–first birth interval (years)	3.4	13.5
Duration of lactation per birth (years)	2.9	0.25
Lifetime duration of lactation (years)	17.1	0.4
Lifetime number of live births[b]	5	1.8
Age at menopause	47	50.5
Total lifetime ovulations	160	450

Adapted from Eaton and Eaton (1999)
[a]Age in years
[b]Up to a maternal age of 50 years old

in environmental and social conditions different from those experienced by most urbanised, modern humans. Such environmental conditions were famously referred to by the English psychologist John Bowlby as the *Environment of Evolutionary Adaptedness* (Bowlby 1969). Therefore even though cancer development may result from very ancient processes, its current and recurrent manifestations in various human populations—or among different individuals in the same population—may also be affected by the interaction between those biological processes and novel environmental conditions, including sociocultural factors that may affect lifestyle options. For instance, Table 1.1 shows a contrast in various reproductive variables that characterise two very different present human populations: African hunter-gatherers and urbanised Americans. The differences in such reproductive variables can be explained by various environmental factors that include availability of resources such as food but also cultural habits such as those affecting age at first birth or whether to lactate or not and how many children to have. The two syndromes, where hunter-gatherer women reach menarche at an earlier age, have their first birth younger, lactate their child for a longer period and have more children but fewer lifetime ovulations than American women (Table 2.1), also seem to be associated with a more elevated incidence of breast cancer in modern American women as compared with hunter-gatherer women (Eaton and Eaton 1999). In a review published by Alero Fregene and Lisa Newman in 2005, they showed that whilst the incidence of breast cancer in North America, northern Europe, Australia and New Zealand is about 95–100 cases/100,000, in most of Africa and eastern Asia it is much lower. In Africa, the incidence is 13.5/100,000 in middle Africa, 20.2 in eastern Africa, 24.8 in western Africa and 31.8 in southern Africa. This may suggest that, at least in part, cultural/environmental factors may affect cancer development. However, evidence also indicates that our genetics (see for instance the role of *BRCA1* and *BRCA2* mutations in predisposing to breast but also ovarian cancer development in some women, Nelson et al. 2005; Lux et al. 2006) may in fact interact with culture and other aspects of the environment to produce health outcomes that could facilitate or inhibit the development of cancer.

Such an interaction between genes and culture is suggested by Fregene and Newman's finding that sub-Saharan women do show some similarities with

African-American women in spite of broad cultural differences in their approach to fertility: in both cases age at diagnosis of cancer was about 10 years younger than in the case of American women of European descent, with stage of disease also being more advanced at diagnosis. In addition, although a longer period of lactation tends to decrease the probability of developing breast cancer, such effect is stronger for carriers of the *BRCA1* mutation than it is for women carrying the *BRCA2* mutation (Jernström et al. 2004).

The long-term persistence of cancer in a population immediately calls for the consideration of two broad levels of analysis in order to understand the influence of cancer on behaviour and how behaviour in turn may affect cancer development: a proximate level of causation that focuses on molecular, physiological and psychological mechanisms operating within the individual and a more distal level of causation focusing on longer term processes that also considers the social and cultural dimensions. Both proximate and distal levels of causation taken together are likely to better explain the recurrence of cancer through time and its specific relationships with behaviour and the mind.

Although throughout our evolution as a species cancer development may have been triggered by purely external factors, such as the ingestion of mutagenic substances with food, exposure to solar radiation, infection by carcinogenic viruses and bacteria and so on, other mechanisms are also plausible and not necessarily alternative. For instance, some cancers may result from spontaneous errors in the molecular genetic mechanisms of cell replication. Others may result from *epigenetic mechanisms* such as the silencing, through *methylation*, of genes that contribute to defence against cancer (Poulaki et al. 2005). There may be little evolved resistance against cancer-causing mutations if they manifest themselves at older ages (50+ years), simply because up until relatively recently it was unlikely that many human beings had reached such ages. In other words, genes that only express themselves in post-reproductive individuals tend to escape the "judgment" of natural selection which means that those genes can be transmitted from generation to generation without great impediments: a late-developing cancer may kill the organism, but it will do so after he or she has reproduced; hence, any mutation that causes such cancer may be passed on to the next generation. For cancer types that can develop at younger ages, one possible explanation for their endurance in human populations could be provided not only by the epigenetic mechanisms mentioned above but also by processes of *antagonistic coevolution* in which genes controlling the ability of cells to compete for limited resources used for reproduction may undergo mutations that lead the cell to uncontrolled reproduction and hence cancer (Crespi and Summers 2006). Alternatively, selection for high mutation rates, that might have been adaptive in our evolutionary past as our ancestors left Africa to rapidly spread across continents, settling in a great diversity of climates and habitats, may have had the unselected side effect of increasing cancer rates. This could explain for instance why cancer genes are unstable and highly prone to mutation (Lengauer et al. 1998) but also why 90 % of such cancer genes show mutations in cells of somatic tissues (mutations that cannot be transmitted to offspring) in contrast to 20 % in cells of the germline (such mutations can be transmitted to offspring) (Futreal et al. 2004). A balance between mutation (causing cancer but also novel adaptive traits

potentially useful in a changing environment) and selection (decreasing the ability of individuals to transmit the cancer-inducing mutation to their offspring) could provide an evolutionary explanation for the recurrence of this scourge in our species. Yet another view that frames cancer in an evolutionary perspective has been recently provided by Ewald and Swain Ewald (2013) who applied Dawkins' concept of the *extended phenotype* (Dawkins 1983) to cancer development. Does the understanding of these or other evolutionary processes concerning cancer matter? Yes, it does. As shown above for the case of breast cancer in women, the disruption of some adapted biological mechanisms may release the molecular processes that lead to cancer. Specific behavioural changes that restore the original adaptation may contribute, at least to some extent, to preventing the onset of cancer. If specific changes in behaviour are deemed inappropriate for cultural or personal reasons, such as protracting lactation for about 3 years, then alternative strategies may be sought that produce the same or a similar biological outcome of protecting the individual from developing cancer. In other words, evolutionary approaches may be put to good use for the design of better behavioural anticancer strategies.

In a recent review of evolutionary oncology, Mel Greaves (2007) points to the association of current cases of cancer in vertebrates with inbreeding, developmental anomalies, exposure to novel carcinogenic compounds and infectious agents or abnormal exposure to natural radiation (e.g. UV light) or hormones. Such diverse patterns of cancer causation could be broadly subsumed into the concept of *disruption of genetic and physiological coadaptations*. By a genetic and physiological coadaptation we mean the specific genetic and molecular structure of an organism that ensures adaptive functioning (survival and reproduction). Resilience of organisms to disruption of such coadaptations seems to have specific boundaries, beyond which the organism is unable to return to its functional dynamic. Microscopically, this may be translated into changes in molecular types (concentration, structure, rate of synthesis, clearance) of such magnitude that ordinary processes that ensure cell, tissue, organ and ultimately organism functioning are disrupted beyond repair. Such a situation could be produced by changes in the DNA that encodes for such molecules or by changes in the internal environment that affects production of those molecules and their mode of action. The internal environment and also the DNA, in turn, may be modified by external entities (viruses, bacteria, toxins, physical stressors such as temperature, radiation). In this context, the association of cancer with aging across taxa, including humans, is also affected by the accumulated effects over time of all the above factors. Stem cells, that retain their ability to replicate and differentiate into any tissue, would be particularly susceptible to disruption of such coadaptations, leading to cancer. The question relevant to this book is to what extent behaviour can also disrupt genetic and physiological coadaptations. Indeed, we will show that there are specific mechanisms that can link behaviour and cancer.

Whether they are the proximate result of environmental factors or a side effect of evolutionary processes, there is little doubt that most forms of cancer, as they manifest themselves in modern human populations, are not directly adaptive, and their ability to cause pain, disruption of normal life processes and premature death amply justifies the effort against them. The removal of the deadly effects of cancer from

Table 1.2 Percentage of cancer patient mortality contributed by the top nine most deadly types of cancer around the world

Males		Females		Total population	
Type	%	Type	%	Type	%
Lung	22.5	Breast	13.7	Lung	18.2
Liver	11.3	Lung	12.8	Stomach	9.7
Stomach	11.0	Colorectum	8.6	Liver	9.2
Colorectum	7.6	Cervix uteri	8.2	Colorectum	8.1
Oesophagus	6.5	Stomach	8.2	Breast	6.1
Prostate	6.1	Liver	6.5	Oesophagus	5.4
Leukaemia	3.4	Ovary	4.2	Cervix uteri	3.6
Bladder	2.7	Corpus uteri	2.2	Prostate	3.4
Non-Hodgkin's lymphoma	2.6	Thyroid	0.7	Bladder	2.0

Data are from the World Health Organization (2008)

human populations has been the chief spur for a considerable investment of funds, time and specialised expertise in research aimed at finding a cure for this group of diseases. The result of such effort is shown in improved and more effective therapies. However, in spite of the encouraging trends observed in economically developed countries such as the USA, where deaths from cancer have tended to decrease in recent times (Jemal et al. 2009), worldwide data from the World Health Organization (2009; data updated to 2004) indicate that cancer remains a major cause of loss of life around the world, accounting for 13 % of all deaths. In decreasing order of importance lung, stomach, liver, colorectal and breast cancers are currently the top causes of cancer mortality, explaining 70 % of cancer deaths worldwide. Incidence of cancer mortality is projected to increase around the world, with an estimated number of deaths of 12 million by 2030.

Specific cancer types also differ in prevalence between men and women populations, with women being mainly affected, in descending order of importance, by breast, lung, stomach, colorectal and cervical cancer, whereas men mainly suffering from lung, stomach, liver, colorectal, oesophagus and prostate cancer (World Health Organization 2009). In both women and men, the most prevalent forms of cancer are those affecting organs of the respiratory and the digestive system that are directly exposed to chemicals, viruses and microorganisms obtained from the external environment or organs associated with reproduction that are in close contact with steroids. It should also be noted that cancers of the skin that can be caused by exposure to UV radiation are relatively less common globally, perhaps reflecting the more ancient history of natural selection against those cancers (see Jemal et al. 2011 for an update with data from 2008 and for further updates visit the GLOBOCAN website of the WHO's International Agency for Research on Cancer; http://globocan.iarc.fr/Default.aspx).

As indicated above, different types of cancers contribute differently to patients' mortality rates. Table 1.2 summarises the values of mortality rates explained by various cancers in the overall world population and also for males and females. Again, the highest mortality rates are explained by cancers affecting organs of the

respiratory and digestive systems and, in women, organs associated with the reproductive function.

Although the bulk of the medical research effort has focused on the molecular and cellular biology of cancer, the past 20 years or so have also witnessed a remarkable increase in interest in the psychological, communicative and social aspects of cancer and disease. Indeed such interest is far older than 20 years, dating back to at least Galen (second century c.e.), who famously noted in his *De tumoribus praeter naturam* that "melancholic" women were more prone to developing cancer than women characterised as "sanguine" (cit. in Justice 1985). The relationship between cancer and behaviour is a complex one and also one that can be easily distorted by superficial generalisations and unsupported inferences. The secret hope that disease will just go away by sheer "mental force" is still pretty much alive, providing fertile ground for unscrupulous operators to manipulate vulnerable cancer patients and their families but also ground for an excessive scepticism among some medical professionals. Whether the activities of our nervous system, externally manifested in behaviours, can or cannot affect the onset and/or progression of cancer via specific molecular and cellular mechanisms should be a matter of proper scientific investigation, not a priori dismissal or acceptance. Moreover, the identification of such molecular, cellular and physiological mechanisms linking behaviour and disease is in fact the crux of the problem as, to quote Howard Friedman and Stephanie Booth-Kewley (1987: 541): "It is silly to postulate a psychological model of disease causation that is physiologically impossible".

A cell becomes cancerous when a single mutation (change in its genetic material, the DNA), or a series of accumulated mutations, allows it to undergo unrestricted reproduction (e.g. Peto et al. 1975). Epigenetic mechanisms have also been postulated, such as the silencing of apoptosis genes by methylation (see Poulaki et al. 2005). In a rare blend of scientific insight and literary skills, Mel Greaves (2000: 101) points out in his book *Cancer*: *The evolutionary legacy* that such "clonal escape" behaviour of cancerous cells is like a reversion to an ancestral state of unicellular life:

> The reproductive imperative becomes progressively uncoupled and insulated from rules for restraint or from penalty clauses. At the point of no return (invasive cancer), a dominant clone emerges with a mutinous character—stone deaf to social dialogue, divorced from its functional context, and with its historic genetic contract eroded. It is immortal and itinerant; free to do nothing but make more of itself. But this character is strangely *déjà vu*. It's back where it started. The obstacle race has been running backwards all along; a resurrection of the long-buried memory of unicellular selfishness. Doing what came naturally, long ago.

But more proximately, cancer simply results from the unconstrained activation of mechanisms of clonal expansion that are currently adaptive in multicellular organisms in the contexts of development and tissue repair. Although the DNA in our cells enjoys a degree of protection against such changes, mechanisms of protection are obviously not perfect. Indeed, from an evolutionary perspective perfect DNA protection is not even "desirable", as a genetic material that is unable to change cannot support processes of adaptive evolution: once adaptive evolution comes to a halt, sooner than later extinction is likely to follow. As mutant genes that can cause cancer

become expressed, a second line of defence for the organism is represented by a natural process of self-destruction of cells, known as *apoptosis*. When apoptosis fails, there is yet another line of defence that may prevent mutant cancerous cells to reproduce and spread, and that line of defence is represented by our immune system. We will see in the next chapter how behaviour can affect the rate of mutation, through the release of stress hormones for instance; how it can affect immunity and therefore rate of spread of cancer within the organism and also how it can affect apoptosis.

The realisation that the various systems that constitute our organism are functionally interconnected as a result of evolutionary processes has opened up new ways of studying disease and of finding effective therapies (e.g. Ewald and Swain Ewald 2012a, see also the various articles in Poiani 2012a). For as long as thoughts and feelings were considered in their more obvious external expressions, their origin in the brain was confined to the obscurity of a "black box"; and for as long as the immune system was studied in isolation to the endocrine and nervous systems, any link between behaviour and disease seemed rather fanciful, Galen notwithstanding. Bridging the gaps across disciplines was considered—and to some extent still is—a high-risk professional strategy as, unfortunately, professional success is still more likely to follow from narrow specialisation rather than multidisciplinarity. In spite of those constraints, a more integrative approach has been adopted in recent times, where the various mechanisms linking behaviour and disease are being elucidated, and that knowledge is being used in the design of more effective therapies. In the production of this book we followed such an integrative approach, based on the early biopsychological suggestions of George L. Engel (1977: 131) for the study of disease,

> We are now faced with the necessity and the challenge to broaden the approach to disease to include the psychosocial without sacrificing the enormous advantages of the biomedical approach

and the more recent evolutionary biosocial approach of Poiani (2010). We did so with a twist. The twist is represented by a specific analysis of the very many biological, social and communicative aspects of behaviour and their potential associations with cancer, down to as much detail as we could possibly master and, hopefully, the reader endure.

Our aim is to provide the reader, whatever his or her professional expertise, with enough background information to reach as deep an understanding as possible of the various factors and mechanisms that are relevant to the issue of "overcoming cancer". We strongly recommend against narrow-minded "black box" thinking: such as focusing on manifest behaviour like language and communication and treating the neurobiology, immunology and endocrinology of the sick individual as a mysterious "black box" or, conversely, plunging oneself into the molecular details of endocrine, neuronal and immune tissue activity whilst treating the social, cultural and communicative aspects of behaviour and their effects on physiology and cancer as a "black box". We hope that our two-way bottom-up and top-down approach will provide a better understanding—what Ronald Melzack refers to as "hybrid vigour" in research (Melzack 2008)—of the relationships between cancer and behaviour and perhaps inspire further developments in research and therapy.

Readers may have noticed that in the previous paragraph we wrote the expression "overcoming cancer" in quotes. The reason for this is that the expression contains a subtle ambiguity. In the manner of those paradoxical figures that can be seen as one object if observed in a specific way, or a very different one if observed through another perspective (see W.E. Hill's "My wife and my mother-in-law" illusion, or the ever-popular "cup and two faces" paradoxical figure), this book, Janus-like, has also two faces. One face is represented by the various ways in which an "overcoming" or otherwise threatening cancer may affect a person's thinking, behaviour, language and functioning in life, whereas the other is represented by the psychological and communicative strategies that can be adopted to help in the process of "overcoming" or at least attenuating cancer and its effects on our quality of life. As already mentioned, we approach both aspects of the cancer experience from a multidisciplinary standpoint, in an attempt to integrate the various levels of causation, from the molecular and physiological to the behavioural and social: How can behaviour affect physiological and cellular processes that lead to cancer or perhaps cancer regression and, vice versa, how can cellular and physiological processes affect the various aspects of behaviour under cancer? In this respect we will draw from both our professional expertise and also our personal experience in life in order to seek as profound an understanding of the various dimensions and complexities of behavioural oncology as possible.

Marisa Cordella is a professional linguist expert in discourse analysis and intercultural communication. In the past 15 years she has carried out research in the field of medical communication, studying the interaction between doctors and patients across various medical disciplines, including oncology. Marisa, as many others, has also experienced the loss of very dear people to cancer. Aldo Poiani is a professional biologist expert in animal behaviour, evolution and host–parasite interactions; he has studied issues relating to immunology, endocrinology, behaviour and disease in an evolutionary perspective. He has also survived Hodgkin's lymphoma, and therefore his understanding of the various behavioural, communicative and social aspects of cancer transcends the academic sphere. We are confident that these experiences have illuminated in a positive manner our professional work in the production of this book, helping us achieve a more thorough understanding of the various issues. We believe that the true complexity of the cancer experience can be better grasped with a mixture of technical knowledge, critical thinking and empathy.

Given our professional background, this book is likely to differ from others. Our objective has been to not only describe and critically review the current issues in behavioural oncology that have clearly emerged from recent medical, nursing and psychological research but also expand towards areas that may be regarded somewhat atypical by health practitioners (such as linguistic analysis and evolutionary medicine). With this we hope to provide an opportunity for the emergence of new ideas and practices that could be used for better interventions on cancer patients.

The book is organised into eight chapters that progress from the more molecular/physiological aspects of cancer and behaviour to the more social dimension. The last two chapters have a more practical focus considering, in turn, aspects of doctor–patient communication and complementary psychological therapies for cancer

patients. All chapters end with a summary section and a series of topics for discussion. With this we invite the reader to reflect on what it is known, think about what it is still unknown and perhaps propose novel and testable ideas that could expand our understanding of the behavioural, communicative and social aspects of cancer. Moreover, special emphasis will be given to thinking about novel therapeutical applications and providing a constructive critique of the current ones.

We start with Chap. 2 where we explore our current knowledge of the behaviour of sick animals and the major neuro-immuno-endocrinological mechanisms that are broadly common to both humans and other mammals in the expression of behaviours in illness. We will then address the more basic aspects of human behaviour as it is manifested in cancer patients more specifically, with emphasis on the experience of pain, chronic fatigue, sleep disorders, alteration of circadian rhythms and nausea. The chapter concludes with an analysis of how the activity of the senses varies under cancer.

We continue with Chap. 3, which focuses on some of the major psychological aspects of cancer, starting from psychological distress, potentially leading to demoralisation and eventually depression, and continuing with the emotional and cognitive experiences of cancer patients. Effects of cancer on dreams, sexual behaviour and personality will also be analysed, including variations across ages and sexes, to conclude with a focus on psychological morbidity in oncological context.

The existential dimension of cancer is the subject of the fourth chapter, where issues of religiosity and spirituality are addressed as they may affect cancer patients, including terminal patients.

Chapter 5 is next, in which the importance of coping strategies, especially those affected by social support, is analysed in detail. Here we also provide a detailed analysis of the use of the Internet by cancer patient support groups. But the social milieu is not always supportive of individuals with cancer, and so the issue of social stigma will also be analysed.

Chapter 6 explores the linguistic aspects of the cancer experience through the analysis of conversations and various forms of narratives. Special emphasis will be given to cross-cultural comparisons of the psychological, linguistic and communicative strategies used by cancer patients. The chapter concludes with a focus on cancer metaphors.

Communication between doctor and cancer patient is the topic of the next chapter, where various aspects of the communicative dynamics between patients, medical personnel and third parties are analysed. The chapter ends with a review of medical education and training in communication for both local doctors and international medical graduates.

The last chapter *Complementary psychological therapies* critically reviews the usefulness and limitations of various therapies such as art, relaxation, humour and many others that could help cancer patients during their treatment and in their remission period to reincorporate themselves to as normal a life as possible. Terminal cancer patients could also benefit from some of those therapies. The biological and psychological foundations of such therapies will be especially highlighted.

Chapter 2
The Behavioural Dimension of Cancer and Sickness

Animals behave in specific manners when they are sick, and some of those behaviours may be adaptive responses to disease, whereas others may be a more straightforward result of dysfunction. Conversely, behaviours such as stressful social interactions may affect the normal functioning of the organism contributing—alone or in conjunction with other factors—to disease development. We start this chapter by reviewing the biological aspects of the interface between behaviour and disease in animals, with special reference to cancer in humans, to then explore in more detail the relationship of human cancer with pain, fatigue, sleep disorders, nausea and the various senses.

Behaviour of Sick Animals

For a long time the behaviour of sick animals was considered an unremarkable subject of study. If an animal is debilitated by an infectious pathogen or a disease such as cancer, then it is no wonder that it may lack the energy to function and behave normally, leading, in the most extreme of cases, to loss of tissue mass and fluids that could ultimately result in death. This view was challenged by Neal Miller in the 1960s with his research on the sickness behaviour of animals injected with subseptic doses of endotoxin. Miller concluded that sickness behaviour is a result of an adaptive motivational state rather than an effect of physical debilitation (Miller 1964). The theme was further developed in the late 1980s by Benjamin L. Hart in his seminal work published in *Neuroscience and Biobehavioral Reviews*. There, Hart (1988: 123) formulated "the perspective that the behavior of sick animals and people is not a maladaptive response or the effect of debilitation, but rather an organized, evolved behavioral strategy" released by disease. The concept that fever in particular is an evolutionarily adaptive response to disease had also been developed by Matt Kluger in the 1970s (Kluger 1978). Of course, sickness behaviour can take extreme, even pathological, forms (Dantzer 2009), in which case the organism may indeed be

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Table 2.1 Sickness behaviours

Hypomotility (lethargy)
Asthenia (lack of energy)
Hyperthermia (fever)
Hypophagia (anorexia)
Adipsia (reduced drinking)
Hyperalgesia (increased sensitivity to pain)
Anhedonia (incapacity to experience pleasure)
Hypersomnia (increased time spent sleeping)
Emotional depression
Decreased interest in exploration
Decreased interest in social activities
Decreased sexual activity
Lack of grooming activity
Disruption of memory
Shortness of breath

Modified from Table 2 of Dunn et al. (2005); see also Fig. 2.1 for some additional behaviours

suffering from a deterioration of health; but in its more common manifestations, the set of behaviours associated with a sick state is a result of an evolved adaptation against some of the damaging effects of disease.

Sick animals and humans in particular tend to be lethargic in movements, behaviourally depressed, physically anorexic (that is, they lose body mass) and febrile. Such *sickness syndrome* is also accompanied by behaviours such as sleepiness, emotional depression, loss of appetite (hypophagia), reduction of drinking (adipsia), increased sensitivity to pain and lack of grooming, sexual activity and social interest among others (Hart 1988; Aubert 1999; Wagner et al. 2006; Dantzer 2009) (see Table 2.1). The sickness syndrome results from a re-organisation of the motivational priorities of the individual experiencing a drop in its usual healthy condition (Aubert 1999). Such re-organisation includes saving energy whilst also activating the immune system. That is, a sick animal will respond adaptively by shifting its usual physiological processes of homeostasis and associated behavioural repertoire to a mode that will allow it to better cope with the challenges imposed by disease. This has been shown experimentally in rats for instance, where sick animals kept under good housing conditions have been observed to interrupt behaviours typically associated with sickness, once confronted with an urgent priority such as retrieving scattered pups and improving insulation of the nest at lowered temperatures. Sickness behaviour is resumed once those urgent priorities have been attended to (Aubert 1999). That is, sickness behaviour is not necessarily evidence of an intrinsic debilitation of the organism but of a temporal rearrangement of the organism's physiological priorities.

Animals, including humans, affected by cancer may also manifest some or all of the behaviours that characterise the sickness syndrome (Cleeland et al. 2003, see also Kelley et al. 2003; Menéndez et al. 2003). That such behaviours can be adaptive is suggested, for instance, by the association of restriction of food ingestion,