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THE SPONTANEOUS REGRESSION OF CANCER

A review of cases from 1900 to 1987

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Abstract

The literature on the spontaneous regression of cancer is reviewed from 1966 to 1987 to update reviews by Everson & Cole and by Boyd. These authors reviewed all cases of spontaneous regression from 1900 to 1965. We then report the entire series from 1900 to 1987. We also attempted to determine what attributions for spontaneous regressions have been reported. Although almost half of the authors failed to speculate or specify a possible cause for the spontaneous regression, the remainder postulated responsible factors such as immunological or endocrine, surgical, necrosis, infection, or operative trauma. The only unorthodox treatment to appear in the literature was the psychological. We conclude that the literature on the spontaneous regression of cancer is still unable to provide unambiguous accounts of the mechanisms operating to affect these regressions.

Key words: Neoplasm, malignant, regression, spontaneous.

The spontaneous regression of cancer is said to occur when a malignant tumour partially or completely disappears without treatment or in the 'presence of therapy which is considered inadequate to exert a significant influence on neoplastic disease' (1). This definition makes clear that spontaneous regression applies to cases in which cancer is not necessarily cured and where the regression may not be complete or permanent (1, 2). Instead, it merely indicates that the tumour growth has stopped or reversed itself over some unspecified period of time. The definition also implies that the term spontaneous is, in fact, a misnomer (3). Obviously, some current unknown variables are involved in the initiation of regression.

In this paper we shall review the available medical literature on the spontaneous regression of cancer, including all cases from 1900 to 1987. With only minor modifications as outlined below, our definition of spontaneous regression will be in accordance with Everson and Cole. A special emphasis will be placed on locating possible causes hypothesized by authors for the occurrence of regressions.

Spontaneous regression

Spontaneous regression is viewed by many authors as a relatively rare occurrence; approximately 20 cases are reported in the world literature each year (4, 5). However, estimates of the 'true' rate vary widely. Bashford (cited in Rae, 6) argued that many cases are probably not reported and therefore estimated that the true rate was about one in every hundred thousand cases of cancer. Boyers (7) indicated that, due to the difficulties involved in establishing what criteria must be met in order for a specific case to constitute an instance of regression, the true frequency is probably closer to one in every eighty thousand cancer cases.

Franklin (8), however, has argued that spontaneous regressions are only reported in the rare case when a regression is both, 'dramatic and long-lasting'. Argued Franklin, 'less dramatic regression and fluctuation in the size of tumours, occur more frequently, but for many reasons they tend to get overlooked, and are almost never reported' (8). In the same vein, Smithers (9) argued that the adherence to rigid criteria may have delayed an understanding of spontaneous regression. Thus observers of the literature of spontaneous regression do not themselves agree on such fundamental issues as the frequency of its occurrence. The use of less rigid criteria for classifying the occurrence of spontaneous regression, however, could lead

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to a higher number of false positives with an attendant misleading increase in optimism in the general public.

An additional problem in determining the occurrence of a spontaneous regression exists due to the ambiguity in what is considered adequate treatment for a particular individual at a specific stage of a disease. In addition, Everson & Cole (1) specifically excluded leukemias and lymphomas, including Hodgkin's disease, from their review because of the natural changes in growth rates which these malignancies undergo.

Whatever the true incidence of spontaneous regression, numerous studies continue to appear in the literature every year. The two most comprehensive reviews of this literature appeared in 1966—one by Everson & Cole (1) and the other by Boyd (2). Everson and Cole's monograph included all cases from the literature, personal communications, and their own patients from 1900 to 1965, inclusive. Using their relatively stringent definition, they found 176 cases which met their criteria for spontaneous regression. Cases were rejected if the diagnosis of the disease was unconfirmed or if a treatment program was implemented which was capable of bringing about a regression. In addition to the omission of leukemias and lymphomas, these authors also excluded cases of retinoblastoma because histiological evidence confirming the presence of this cancer was rarely available.

Boyd's monograph detailed an additional 61 cases of spontaneous regression, including those of retinoblastoma, if the diagnosis was confirmed through the establishment of both a family history of the disease and histiological evidence. Although a number of other reviews of cases of regression are available (e.g. 302 cases (10)), these two reviews present the most detailed collection of case studies using standard criteria for selection. Our review will include the cases of Everson and Cole, and Boyd and bring these up to date to the end of 1987.

Criteria for inclusion

The accumulated world literature on the spontaneous regression of cancer from 1966 to 1987, inclusive, was reviewed and added to a compilation of those cases presented by Everson & Cole (1) and Boyd (2). We have accepted the criteria of Everson and Cole with the exception that we have included cases of lymphoma, leukemia and retinoblastoma. Without being too lax, we wished to reduce our misses while simultaneously preventing a high false-alarm rate. We can therefore provide a review of the literature from 1900 to 1987 with the exception that prior to 1966 no cases of lymphoma or leukemia were included by either Boyd or Everson and Cole. In addition, Everson and Cole omitted cases of retinoblastoma.

Both manual and computer searches through Index Medicus and CancerLit revealed a total or 1 199 articles pertaining to this subject. Of the 220 articles which were written in a language other than English, 52 could not be obtained either because of problems in finding a location, or obtaining the articles even if a location could be found. These 52 articles were typically in rarely referenced journals with poor international circulation. Additionally, 370 of the articles were animal studies and thus not relevant to our particular review. Of the remaining articles, 475 were omitted on the basis that they did not represent case histories or instances of spontaneous regression. These 475 articles fell in three groups: In the first group, 301 articles were general discussions of the nature or process of spontaneous regression, reviews of previously published works in the field, or were letters, editorials or research proposals addressing some aspect of cancer regression. A further 119 were articles which, although claiming a spontaneous regression of cancer, employed a standard, medical treatment procedure which could have been capable of regressing the cancer. The third group consisted of 55 references concerned with an experimental procedure or manipulation which was used to deliberately initiate or cause the regression.

Fourteen articles (11 referenced in Index Medicus, 3 obtained from reference lists in the obtained papers but not in the computer searches) are not included as individual case studies due to the fact that these 14 papers account for 6 136 cases of regression, the nature of which was unclear. These reviews lacked sufficient, histiologic evidence of both the presence and subsequent regression of individual neoplasms. The fourteen articles were: a) 2 110 cases of capillary hemangiomas (11); b) 2 084 cases of hemangioma, 83% of which were noted to regress (12); c) 596 cases of hydatiform mole regression (13); d) 505 cases of hemangioma (14); e) 233 cases of oral leukoplakias (15–18); f) 100 cases of hemangioma, 54% of which were noted to have regressed (19); g) 71 cases of basal carcinoma (20); h) 4 cancers with regression due to some unspecified psychological intervention (21); i) 18 unspecified types of cancer with regression due to 'non-somatic causes' ((22, 23); to establish the nature of the cancers in question and the psychological interventions presumed to have initiated the regressions, we attempted communication with Weinstock. This was unsuccessful.), and j) 486 cases of cutaneous melanoma (24). The lack of standardized reporting methods, and the inability to verify diagnosis and subsequent regression for such a large volume of cases prevents us from including these case reviews in our totals. Therefore, of the original 1 199 articles referenced in Index Medicus and CancerLit, 265 articles will be presented in this paper representing 504 case studies of the spontaneous regression of cancer.

The three reviews which are represented in this paper (Boyd, Everson & Cole; Index Medicus and CancerLit, 1966–1987), account for 741 separate case studies of the spontaneous regression of cancer. Table 1 presents the data from Everson & Cole (1) and Boyd (2). Over 63% (n = 111)

Table 1

Frequency of site of cancer, 1900–1965 (n = 237)

Cancer	Everson & Cole	Boyd
Hypernephroma	31	0
Neuroblastoma	29	3
Choriocarcinoma	19	0
Malignant melanoma	19	4
Bladder	13	0
Soft-tissue carcinoma	11	2
Sarcoma of the bone	8	1
Colon/rectal	7	1
Ovary	7	0
Testicular	7	1
Breast	6	15
Stomach	4	3
Unknown primary	4	0
Uterus	4	2
Liver	2	2
Larynx	1	0
Lung	1	4
Pancreas	1	0
Thyroid	1	4
Tongue	1	0
Vater's ampulla	0	1
Pharynx	0	1
Retinoblastoma	0	17
Total	176	61

of the cases presented by Everson and Cole were accounted for by 5 types of cancer; kidney (n = 31), neuroblastoma (n = 29), malignant melanoma (n = 19), choriocarcinoma (n = 19), and bladder (n = 13). A review of those cases identified by Boyd indicates that only 11% (n = 7) can be accounted for by these same 5 cancers.

A combination of all three of these data sets reveals that these same 5 cancers account for approximately 40% (n = 300) of all cases of spontaneous regression. If, however, cases of retinoblastoma, lymphoma, leukemia and breast cancer are now included, then these 9 cancers account for about 69% (n = 514) of all cases of spontaneous regression. Table 2 presents the frequency by site of spontaneous regressions from 1966 to 1987.

Of the 741 individuals who presented with a spontaneous regression of their cancer, approximately 60% were still alive more than one year after the regression occurred, while over 25% were still alive after 5 years. The lack of a standardized method of reporting such cases made it impossible to establish the length of regression for about 20% of the individuals.

Although conclusive evidence of the factor(s) actually responsible for the occurrence of a spontaneous regression is lacking, many of the studies reviewed proposed possible causative mechanisms. Unfortunately, Boyd only presented

Table 2

Frequency of site of cancer, 1966–1987 (n = 504)

Miscellaneous	(n = 211)	Genitourinary/breast	(n = 116)
Lymphoma	68	Hypernephroma	68
Leukemia	53	Breast	22
Neuroblastoma	41	Testis	16
Retinoblastoma	33	Bladder	4
Kaposi's sarcoma	7	Uretha	2
Hemangioendothelioma	2	Uterus	2
Hodgkin's disease	2	Endometrium	1
Angioendothelioma	1	Ovary	1
Lymphosarcoma	1		
Malignoma of sclera	1	Skin	(n = 84)
Sympathoblastoma	1	Malignant melanoma	69
Osteoblastoma	1	Bowen's disease (vulva)	6
Merkel's cell tumor	1	Basal cell carcinoma	4
		Bowen's disease (penis)	4
Primary unknown	(n = 10)	Epithelioma	1
Gastrointestinal	(n = 34)	Head and neck	(n = 13)
Colon/rectum	10	Adenoid cystic carcinoma	5
Stomach	10	Larynx	4
Liver	10	Pharynx	1
Pancreas	3	Other squamous cell carcinomas	3
Intestine	1		
Endocrine	(n = 2)	Respiratory	(n = 25)
Adrenal	1	Lung	18
Thyroid	1	Bronchial	7
Brain	(n = 4)	Soft-tissue and bone	(n = 5)
Glioma	2	Chondrosarcoma	1
Astrocytoma	1	Osteosarcoma	3
Intrasellar tumor	1	Sarcoma	1

a generalized discussion of possible factors, and thus the true frequency of each hypothesized mechanism is not known for those cases. Cole (3) was able to outline possible causative mechanisms for all of the cases presented in his 1966 study and his largest category by far (46%) was 'operative trauma." In our review of cases from 1966 to 1987, over 40% of the authors failed to specify any causative factors. It was possible, however, to determine the relative frequency of the remainder. By far the single largest category of 'causes' (referred to in over 20% of cases) was a general one labelled simply as 'host immunities'. Only two other causes were reported more than 5% of the time. These were 'cell maturation/differentiation' and 'necrosis'. Both 'infection' and 'operative trauma' were reported 4% of the time.

Discussion

A large number of authors who report spontaneous regressions do not discuss the possible reasons or mechanisms involved. This is understandable in light of the fact that the scientific literature generally provides little data to explicate this process. Publishing a case report in the literature requires that, unless certain, the author refrains from making plausible but unscientific guesses. On the other hand there are a subset of cases that either hint at the mechanisms involved or, where the authors felt that they had enough confidence, that outline speculations on the possible mechanisms involved. We will divide our discussion of possible mechanisms into those which are biological versus those that are primarily psychological.

Biological mechanisms. First, our review indicates, at least indirectly, what others have frequently reported, namely that the more common the tumour, the less likely one is to see cases of spontaneous regression (8, 25). For example, according to the National Cancer Institute's Surveillance, Epidemiology and End results program (1977–1981) (26), by far the most common cancers are lung, colon and rectum, and breast. Proportionally, however, they are underrepresented in the spontaneous regression literature. Franklin (8) has argued that perhaps there is greater susceptibility to common tumours against which there is little defence. With less frequently occurring tumours, however, there is greater resistance. Therefore, 'this makes the tumour less likely to develop, and if it should arise, there is a greater chance that it will be affected by host resistance factors' (8). Although this is an explanation that requires further research, it is one which posits host resistance as the site of action. Directly or indirectly, all biological explanations rely on this concept.

There are various factors which can affect host resistance. The majority of authors in our review who mentioned a possible cause did so by implicating either 'hormonal' or 'immunological' mechanisms. Thus, the onset of a pregnancy (e.g. (27)), severe dietary changes (e.g. (28)), or the cessation of ingestion of oral contraceptives (e.g. (29)) have each corresponded with the onset of a spontaneous regression. On the other hand, there are some cancers which obviously respond to hormonal manipulation. As Franklin (8) noted, breast and prostatic cancers which respond to endocrine changes are not viewed as regressing 'spontaneously.' It is possible that other tumours may also respond to changes in levels of hormonal environment, although this is not certain (1, 8).

Research in immunological functioning may soon be able to provide a better explanation of the mechanisms by which tumours develop or are impeded (4, 30, 31). Furthermore, current research in biotherapeutics may also provide improved treatment for some cancers (e.g. (32)). However, the exact immunological mechanisms for the 'spontaneous' regression of cancer are not understood and authors who use this explanation appear to do so more out of a willingness to provide some clue than out of any clear understanding of the mechanisms involved.

Many reports of spontaneous regression have implicated surgery or operative trauma as an element that can increase immunological resistance to tumour growth (e.g. (3)). A number of cases have been reported in which surgery conducted on the primary tumour or the metastases has led to regression in the remaining tumour mass. The removal of a portion of the tumour burden presumably allows the host immune system to destroy the remaining tumour (3, 8).

Necrosis or vascular insufficiency is another variable frequently reported as a cause for the regression of a tumour (33). Necrosis itself plays a role in controlling tumour growth and may lead not only to spontaneous regression but also to the stabilization of a tumour or even its progression (34). In the case of spontaneous regression, however, operative procedures or radiation either restrict blood and nutrient supplies to the tumour or else nutrients become insufficient leaving the tumour to die. Thus, although a therapeutic intervention was usually involved in such cases, the treatment which was utilized was considered to be inadequate to 'cure' the disease and hence the classification of these tumours as spontaneously regressed.

A number of cases of spontaneous regression have been directly attributed to infection (35). For example, there are reports of remission in leukemia which have been associated with pyogenic infections (36). An infectious disease incites an increased host immunity reaction which, in turn, may actually increase an individual's defenses against a tumour. Unfortunately, however, the exact nature of this process is also not well understood.

The very structure of this literature, relying as it does on case reports, makes it difficult to conclude with any certainty that the postulated variables are in fact responsible for a particular regression. It is only following the examination of a large series of cases that pursuing certain

causes in further research may seem prudent. Of those authors willing to speculate, immunological and endocrine functions appear to be the most consistently reported factors in the biological domain that are related to the spontaneous regression of cancer.

Psychological mechanisms. Of all possible mechanisms cited for a regression, the psychological is the only category which is not clearly biological. Only three authors are primarily responsible for the reports of regressions by psychological means in the scientific literature (Meares, Weinstock, Wolley-Hart) and only one of these (Meares) provided sufficient information to be able to include the cases in our tables. The cases come from papers published by Meares over a number of years, each of which includes one or more cases (37–42).

The reasons for this are unclear. It may be that no physician was willing to risk his/her reputation by reporting a case of spontaneous regression he/she felt was due to a psychological method. Interestingly, there is a large and popular literature on the healing effects on cancer of psychological techniques, therapies or treatments (e.g. (43–47)). Here, too, are many claims of regressions lasting for various periods of time. For example, Simonton et al. (43) indicate that, of 159 patients with 'medically incurable malignancy' treated by their method of intensive psychotherapy, 14 patients showed no evidence of disease and a further 12 showed a regression. Like almost all proponents of unproven remedies, the Simonton's provide no corroborating evidence for these figures.

Whereas psychological interventions may be beneficial for the psychosocial problems of cancer patients (e.g. (48)), the scientific literature indicates that there is no support for the claims that any of these techniques actually reject, contain or otherwise lead to the regression of cancer (e.g. (49)). While there are links between cancer and stress and cancer and the central nervous system mediated by the immune system, these are a long way from global personality variables and psychological treatments (e.g. (50–62)). The relationship between personality and psychopathological constructs and the onset or progression of cancer is itself far from clear. This literature is riddled with contradictions and unreplicable or interpretable results (63).

In summary, we are left to conclude that, although a great number of interesting and unusual cases continue to be published annually, there is still little conclusive data that explains the occurrence of spontaneous regression. Perhaps this is to be expected given the wide variety of neoplasms involved. Any explanations proffered are, of course, also dependent on the state of knowledge about the etiology of neoplasms generally. All one might hope for in that case, is that the continued publication of cases of spontaneous, or at least unidentified, regressions will prove to be of heuristic value.

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Request for reprints: Dr H. J. Stam, Department of Psychology, University of Calgary, Calgary, Alberta, Canada T2N 1N4. A bibliography of the papers culled from the world literature (1966–1987) and included in this review is available from the same address.

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