Although the great possibilities presented by the study of transmissible tumors of lower animals were fully revealed in 1894 by the systematic observations of Morau, it was not until 1901–2 that the work of Loeb in America and Jensen in Denmark attracted universal attention to this field. Especially in the last decade a great number of observations from a host of workers have produced a body of new data which is of first importance in oncology. From this extensive field it is my purpose to select for discussion certain problems which are of special interest to the general pathologist and to attempt to evaluate the new contributions by the old and established pathological criteria.

Prominent among the questions raised by the study of transmissible tumors of lower animals is whether these processes are genuine neoplasms. Considering that Virchow once said that no one even under torture could state exactly what a tumor is, this question must appear somewhat academic. Yet to the older pathologists, thoroughly saturated with the conviction that a neoplasm is viable only in its host, the demonstration of successful transfer to a new host naturally raised a lively scepticism. Transmissibility implied to them infectiousness and a granulomatous nature; the transplantable tumors must be shown to grow solely from the transplanted cells, after the manner of metastatic tumors, they must exhibit infiltrative growth, and

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produce metastases; the striking morphological resemblance to tumors was not universally regarded as decisive evidence. All these essential attributes of true malignant tumors have now been demonstrated for many of the transplantable growths in lower animals.

Personally, I consider the morphology alone as a sufficient guarantee of the neoplastic nature of many of these tumors. Doubts of its significance seem to result from a somewhat narrow conception of the very variable morphology of the cancer process, and similar doubts have arisen regarding the cancerous nature of certain human tumors of peculiar character. Thus Borst discusses the question whether primary liver cell carcinoma is a true carcinoma because the infiltrating cells are mechanically forced into the hepatic capillaries and do not actively grow into them as do some tumor cells. Yet it seems somewhat arbitrary to demand that hepatic carcinomas should behave exactly as do mammary carcinomas. The hepatic carcinomas have many peculiarities of their own, but all the essential characters of malignant tumors. Similar reservations may be made regarding carcinomas of the thyroid, stomach, adrenal, and other organs, in which morphological details are not cut to exactly the same pattern. Moreover, cancer morphology does not always appear at its full development, but is progressively unfolding its potentialities.

Analysis of the structure of human cancer reveals the following features, any one of which may dominate the picture or any combination of which may be exhibited.

1. Hyperplasia, surpassing that observed in other conditions occurring in the same organ. A sliding scale is here necessary owing to the great variations in the degree of hyperplasia resulting from inflammatory and functional changes in the different organs.

2. Atypical qualities of the cells. This feature is universally recognized as perhaps the most significant criterion of a malignant process, and when sufficiently pronounced may stamp the process as malignant in the absence of other changes. (Early carcinoma of larynx).
3. Loss of polarity. The regular alignment of cells in relation to one another and to basement membranes may early be lost in adeno-carcinomas, and is completely destroyed in diffuse carcinomas.

4. Desmoplastic properties. The capacity to excite the growth of connective tissue is very prominent in most carcinomas, but entirely lacking in others. Mammary fibrocarcinoma and embryonal carcinoma in children represent the two extremes.

5. Infiltrative growth is a late property of most carcinomas and the time of its appearance depends much on the accidents of position.

6. Metastases. As an objective sign the occurrence of secondary tumors is most convincing evidence of a lawless growth, but in benign metastasizing stroma they occur early, in an otherwise innocent tumor, and in some types of malignant hepatoma they never develop.

Judged by these main criteria, the tumors of lower animals take their places as malignant neoplasms, with certain peculiarities which are impressed upon them by their species.

Metastases were observed and experimentally produced by Morau, and Murray found them in 50 per cent of his mouse tumors. Their relative infrequency as compared with those of human tumors may be referred largely to the short duration, and the peculiarities of the circulation in small animals. Infiltrative growth is frequently observed under suitable conditions, especially when the tumor meets resisting structures. The degree of cellular hyperplasia may be so great that the tumor out-weighs the host, and in general it probably attains a larger relative volume than in human tumors. The atypical qualities of the cells are almost constantly pronounced, but while this feature has been emphasized by some, I have gained the impression that the variations in tumor morphology in lower animals are less violent and less extensive than in the human subject. Extensive overgrowth of connective tissue is also much less notable in the small animals for reasons which are not entirely clear.

I have extended this analysis to some length because the study
of lower animal tumors has forced the pathologist to relax to some extent certain rigid notions regarding what a tumor may do and how it may look.

The comparative studies have not, however, revealed any striking variations or new morphological properties beyond those occasionally exhibited by human tumors. The same fixity of form and clinical behavior reappear in the different examples of the same type of tumor, and this morphology is usually maintained throughout many generations of transplants. The structure may vary in different portions of the same tumor, and metastases may be more or less atypical than the original growth.

There is one important phenomenon exhibited by lower animal tumors, however, which is at variance with the rules deduced from human oncology. Certain transplanted tumors of mice and rats are said to excite a neoplastic process in the stroma of the host so that in the course of transplantation a stroma-born sarcoma may arise and even outgrow and eventually eliminate the original carcinoma. This phenomenon was first observed by Ehrlich and Apolant in the tenth generation of an adenocarcinoma of the mouse, and has been reported in both rats and mice by Loeb, Lewin, Bashford, Haaland, Russell, and others. The change appears to occur rather suddenly in the 8th to 10th or later generations. Russell fixed the usual period at the 55th to 60th day of propagation and states that it is independent of the number of transfers.

When once established it is usually progressive and both tumors persist together, or separate strains of sarcoma and carcinoma are obtained in subsequent transplants. The rate of growth of the mixed tumors is usually increased and the sarcoma is usually more active than the carcinoma, and is more frequently encountered in metastases (Haaland, Clunet).

The idea that the original growths were mixed tumors was abandoned for lack of any evidence. It is generally concluded that the sarcoma represents a neoplastic transformation of the stroma of the host caused by a stimulation of cells by the tumor epithelium. Both Haaland and Russell describe in detail the appearance of foci of overcellular stroma located in the centre
of carcinoma nodules, the increase in mitotic figures in the stroma cells, the survival of these altered cells in grafts, and their rapid increase until they overgrow the epithelial elements.

There is no counterpart in human pathology for this remarkable process. In no case has the stroma of a human tumor during vicissitudes of prolonged growth, inflammatory reaction from infection, recurrence after operation, or spontaneous metastases, taken on a neoplastic growth. It is true that very notable grades of reactive hyperplasia may be excited by the invasion of cancer cells. The most remarkable is probably seen in the bone metastases of prostatic carcinoma (v. Recklinghausen). Here there is very extensive overgrowth of bone following carcinomatous invasion, and histological study does not always succeed in separating tumor cells from multiplying osteoblasts in the new tissue. The process has been regarded by some as possibly sarcomatous, and secondary osteosarcomatous metastases are said to occur in the lung (Schmorl, Fischer-Defoy, Axhausen). Yet it is not clear that the osteoblasts take on true neoplastic properties, and the pulmonary nodules are generally regarded as osteoplastic carcinoma.

It is perhaps unreasonable to expect that there should be a human parallel, for human tumors do not experience quite the same insults as transplanted tumors receive. Yet it is a well established rule that the stroma never participates in the lawless growth of cancer cells except in mixed tumors, and it is distinctly anomalous that no trace of this sarcomatous transformation appears in man, when it is relatively common in mice. Under these circumstances it may reasonably be demanded that entirely demonstrative evidence should be furnished before its occurrence in mice can be accepted. Not having actively engaged in this study I am not in a position to deny the claim of the very competent investigators on whose evidence the interpretation rests, but it may be permissible to point out some of the difficulties in the way of accepting their interpretation that the stroma of mouse cancer becomes sarcomatous.

Human carcinoma never exhibits a sarcomatous transformation of its stroma, but its polyhedral cells frequently lose all
their epithelial characters and grow in spindle form. Epithelial tumors may from their inception appear like spindle cell sarcoma, as in the spindle cell basal-cell carcinomas of Krompecher, in spindle cell carcinoma of the thyroid, and in melanoma. It is becoming more and more apparent that many so-called sarcomas of the organs are in reality spindle cell carcinomas. Or the change to spindle cells may occur in one portion of a typical carcinoma as is frequently seen in tumors of the thyroid gland, and liver; or spindle epithelium may regularly appear with columnar, as in glandular adamantinoma. This tumor is notorious for its remarkable changes of cell form. In recurrences after operation on typical carcinomas, one frequently encounters pure spindle cell growths. I have traced an adamantinoma recurring after four operations, through the structures of adult acanthoma, plexiform epithelioma without squamous cells, spindle cell sarcoma, and finally round cell sarcoma. Recently I have found spindle cell perivascular sarcoma in a uterus, removed shortly after curettage, revealing typical adenoma.

Spindle tumor cells are so common in carcinoma that their occurrence in any carcinoma is very strong presumptive evidence that they are altered epithelium.

The intrinsic evidence relating to the transformation of the stroma is not entirely satisfactory. The change is sudden and coincident with an increased rate of growth of the tumor, as is the case with human tumors assuming the spindle cell form. A new tumor process, affecting the stroma, might be expected to develop more gradually. The new tumor process seems to outstrip and may eliminate the old. This observation, that an original carcinoma should yield its powers to the stroma cells and itself retire from the scene, seems highly paradoxical. The reverse process, viz., the elimination of spindle cells in the course of transplantation of a carcinoma has been observed by Apolant, but here the spindle cells were interpreted as altered epithelium. The fact reported by Haaland, that the carcinomatous element in the mixed tumor may be eliminated by heating to 44°C., is a suspicious circumstance and indicates that the spindle cells
are merely the more resistant as well as the more rapidly growing form of epithelium.

The crucial evidence is that presented by observations tracing the actual transformation of stroma cells into the spindle cell sarcoma. Not all authors have been able to convince themselves that the stroma really gives origin to the sarcoma but Russell and Haaland have traced this origin in detail. Haaland pictures peculiar halos of large pale cells surrounding epithelial groups. They are intermediate in form between stroma and tumor cells but Haaland derives them from the former. Yet the interpretation of transitional pictures is notoriously hazardous and few observers have been willing to trust it in this connection. Orth thought that Lewin's sarcoma represented granulation tissue. Through the kindness of Woglom, I have examined two cases purporting to show the sarcomatous transformation of stroma cells, but have been forced to draw from these sections the opposite conclusion, namely, that the spindle cells are derivatives of the epithelium. In view of all these difficulties it may be urged that further evidence is required before the sarcomatous transformation of mouse carcinoma can be accepted as proven. The writer would not deny its occurrence but merely asks for more evidence.

The experimental studies have not succeeded in defining what constitutes a tumor. They have shown indeed that perhaps the most essential property of tumor cells is the capacity for unlimited growth in a wide range of environments. A vast number of experiments in the transfer of normal or proliferating cells of embryonal or adult type has shown that these cells may multiply for a short period but soon differentiate and become quiescent, or atrophy and disappear. Transplanted tumor cells, however, continue to proliferate beyond the life-time of the host from which they were derived.

Yet just this conspicuous tumor character is exhibited in a notable degree by two diseases the exact nature of which has not been fully determined—the so-called infectious lymphosarcoma of dogs, and Rous' chicken sarcoma.
The infectious lymphosarcoma of dogs is transferred by coitus and also by other methods of contact. It affects primarily the genital organs, skin, kidneys and some other organs, and produces bulky primary tumors as well as miliary or massive metastases. The gross anatomy is typical of a malignant neoplasm, and the microscopical structure also is that of an extremely active, atypical, infiltrative, malignant tumor. It probably arises from the reticulum cell of the lymph-node. No micro-organisms are demonstrable in unulcerated tumors. Transplantation is successful only under the same conditions as govern the transfer of mouse tumors, i.e., the inoculation of living cells into closely related animals. Doubts regarding its true neoplastic nature arise from its evident relation to the lymphocytomas. Furthermore, it must be admitted that at some point in the history of its development a parasite may enter, though there is no evidence that a parasite persists throughout the disease. Bashford concluded that the tumor grafts grew in part at least from the host's cells like a granuloma. In serial transplants studied by Beebe and the writer it grew exclusively from the transferred cells. Bashford, on examining these sections, admitted that they differed from his own, and I am forced to conclude that he was dealing with infected material or that some other factor caused unnecessary inflammatory reaction about the grafts.

v. Dungern claims to have dealt the "coup de grace" to this tumor by showing, through the absence of agglutinins for dog's corpuscles, that the tumors lose their dog protein when transferred to foxes and accumulate only fox protein. His hare sarcoma, which had likewise certain granulomatous features, preserved its hare protein while growing in rabbits. I must decline to accept v. Dungern's conclusions, based as they are on such evidence as agglutination and hemolysis. The tumor can be seen to grow from the transplanted cells, and in comparison with such direct evidence the significance of antibodies for a specific protein is of quite subordinate value. Moreover it appears reasonable that a few dog tumor cells multiplying many thousand times in fox tissue should rapidly acquire the fox stamp. Beebe has shown that mammary carcinoma growing in
lymph-nodes acquires the nucleo-histon of lymph-nodes which is entirely missing in primary tumors of the breast.

I think therefore that the lymphosarcoma of dogs may safely be regarded as a disease sui generis, but essentially neoplastic. It may be complicated by a parasite, but the existence of this complication is not proven. Exactly the same uncertainties surround the origin and nature of human lymphosarcoma.

Rous' series of chicken sarcomas is one of the most interesting and obscure developments of experimental cancer research.

On morphology, one would accept his original tumor as a neoplasm. It presents excessive hyperplasia so far as may be judged by available standards. The atypical qualities of the cells are pronounced. There is infiltrative growth and metastases are ubiquitous. It was at first transferred with considerable difficulty and only to the same strain of chickens, but its viability gradually increased. Yet it violates the central criterion of tumor growth by being transmissible by means of tumor filtrate passed through a Berkfeld filter (No. 5 medium). The active agent will not, however, pass a Chamberland bougie F. Tumor tissue dried for some months remains active, but the resulting tumors are feeble. Exposure to 55°C. inactivates the agent. Cultures are negative.

On these data one must either discard the process from the group of neoplasms or alter the experimental criteria. The latter course seems the only logical choice.

In attempting to analyze the significance of this tumor we are handicapped because the principles of avian pathology are not well understood. One must hesitate to apply the standards of human pathology to the tissue reactions of the chicken. Exudative inflammation, functional and inflammatory overgrowth, metaplasia, etc., may follow somewhat different laws or standards in such widely separated species. Hence one may be in error in too closely identifying this chicken sarcoma with sarcomas in other animals. It may be an infectious granuloma with neoplastic morphology. But I do not think that either the granulomatous or the infectious nature is probable. The only infectious agent meeting the requirements would be an extremely
labile protozoön, of the actual presence of which there is no tangible evidence. It has been suggested as a last resort that fragments of cell nuclei capable of reproducing the cells may pass through a Berkfeld filter. While the data are still inadequate to force any conclusion, I have received the impression that Rous' sarcoma is a genuine neoplasm, occurring only in the chicken, and that the transmissible virus is of chemical, and possibly of ferment nature. The extensive series of transplants has probably intensified the action of any such chemical agent present in the original tumor, so that effects are now being produced with this tumor which were not possible with the spontaneous growth and which probably have no counterpart in any other process spontaneously occurring in Nature. At any rate, the principles deduced from this process must for the present be applied to this disease and to no other.

There are, however, some indications that chemical agents such as may be active in the chicken sarcoma, are also of influence in some human tumors. While many tumors, after their area of origin is defined, grow exclusively from their own resources, others grow by progressive inclusion of previously normal cells in the tumor sources. This principle comes to light in many different circumstances, and may be employed to account for diffusely spreading or multiple tumors of serous, mucous, or cutaneous surfaces, and in systemic tumors of lymph-nodes. In a lesser degree, it may account for collateral hyperplasia about the edges of some tumors. Paget's disease covering much of the chest and trunk would be explained thereby, and the pigment of melanoma is said to have excited the neoplastic proliferation of liver cells about hepatic metastases. So one may conceive that in the chicken tumor a chemical agent may exist which is remarkably effective in inducing neoplastic hyperplasia, and that either this agent alone or the cells that contain it may give rise to the tumor. It may here be recalled that the filtrate is much less effective than are the living tumor cells. Perhaps, also, chicken tissues are more responsive to such agents than are other animal cells.

It would be interesting to know if carcinoma in the chicken,
of which Ehrenreich has described several, is also transmissible by means of its filtrate.

In determining the effects of changes in the soil upon the growth of the tumor, experimental studies have yielded results of much practical value. Spontaneous variations in the growth of tumors are quite as well established clinically as in the experimental field, but the latter seems to have revealed some of the reasons. Racial differences in susceptibility have been practically identified with changes in the diet. The importance of carbohydrates in the nutrition of tumors has long been recognized among clinical writers and has been extensively discussed by Brault, Keating-Hart, and many others. The varying glycogen content of different human tumors suggests the caution that all tumors may not be especially dependent upon this class of food stuff, and the complexities of carbohydrate metabolism indicate that it may be difficult to secure satisfactory experimental evidence in the field of tumor diets. Nevertheless it appears that rats may be made refractory to the Buffalo sarcoma by a previous course of carbohydrate-free diet, that the course of this tumor once established is retarded by such diet, whereas it is accelerated by butyrates among the fats. Benedict saw the complete regression of large sarcomas in rats rendered completely diabetic by phloridzin. By substituting foods rich in lime, Sweet, Corson-White and Saxon rendered rats markedly insusceptible to sarcoma, and saw much acceleration in growth on return to normal diet. Contrary observations show that all tumors do not react to dietary changes, but the important feature of this work consists in the demonstration that the subject is susceptible of experimental study and opens up one of the most attractive fields in experimental cancer research.

Age is found to have no definite influence on the susceptibility to tumor grafts. Both young and old animals have proven suitable for experimental propagation, although most workers prefer quite young subjects. This rule, which could hardly have been established except by experimental studies, has an important bearing on our conceptions of the relation of age to tumor incidence. Cancer has been held to be a disease of old age and yet
young animals are the better soil for growing tumor cells. Hence it is not constitutional susceptibility but the effective action of inciting factors which belong to age. The problem of the inception of cancer is quite apart from the problem of its continuous growth. Experimental studies have had little concern with the histogenesis of tumors and only to a slight extent with their general etiology. In the transplantable tumors these important questions have suffered some neglect, but work in this field has been of great value in defining the influence of age, and separating the questions of inception from that of later growth of tumors.

Immunity: Although Lauder Brunton once said that immunity will eventually be found to be a function of the liver, the serologist will doubtless claim exclusive rights in this field. The pathologist may gladly yield this territory, pointing out that serology has signally failed to produce satisfactory evidence regarding the nature of resistance to tumor growth.

Animals in which a tumor has spontaneously regressed are often actively resistant to further implantation, but the resisting factors can rarely, if ever, be transferred to susceptible animals and specific antagonistic factors in the blood serum have not been satisfactorily demonstrated. On the contrary, it is rather clearly apparent that immunity to tumors is histioid and cellular and reveals itself in the reactive growth of connective tissue, phagocytosis, and lymphocytic attack on the tumor cells. A sufficient stroma reaction has been emphasized by many as the essential element in successful implantation, but abortive grafts are often found sharply enclosed in alien connective tissue. Phagocytosis is very frequently observed about grafts implanted in insusceptible animals, and many types of cells participate in this process.

Lymphocytosis and lymphocytic invasion are prominent in the reaction against grafts in resistant animals and about regressing tumors. There have been numerous observations pointing to the importance of lymphocytes, large and small, in the local and general reaction to tumor growth. Recently Murphy has shown that mouse tumors may grow in chicken embryos until the time when the production of lymphocytes becomes es-
tablished, that this time may be shortened by implanting spleen tissue in the embryo, and that tumors may grow in alien species whose lymphocytogenic function is paralyzed by the x-ray.

Most of these observations on the mechanism of tumor immunity must be valued as confirming conclusions previously drawn from human pathology. Schmidt has shown that vagrant tumor cells are not destroyed in the circulating blood, but are lodged in capillaries, coated with fibrin, inclosed by endothelial cells, and reduced to fibrous nodules. It is highly probable that many tumor cells are destroyed in the lymph nodes which drain tumor areas. The function of lymphocytes in limiting tumor growth is extensively illustrated in human material. In many instances they form the chief barrier against the initial downward invasion of epidermoid carcinoma. In many mammary carcinomas one may see islands of tumor cells in process of destruction by lymphocytes, and polyblasts figure prominently in the active fibrosis which incarcerates many tumor cells. It is a well founded principle of pathology that degenerating or alien tissue cells are removed by phagocytosis. Reactive fibrosis limits the growth of many tumors, more often in man than in lower animals, and may be interpreted as a form of cicatricial healing. It may reach very extensive proportions; thus, I have seen nearly the whole liver transformed into scar tissue in which were very scanty remnants of an original metastatic carcinoma. Lymphocytic activity and extensive fibrosis are features that belong to the so-called clinically resistant cases, and are generally wanting in the more rapidly progressing carcinomas. These processes seem to be particularly prominent in very old subjects in whom carcinoma is of slow progress and often takes a scirrhus form. Back of these well known histological signs of the mechanism of immunity are doubtless submerged constitutional and local influences, in the elucidation of which tumor serology doubtless faces a significant future.

On the therapeutic side, experimental cancer research still presents itself practically empty-handed. The exclusive and quite energetic pursuit of the principles of serum immunity has accomplished practically nothing, except to show that the malig-
nant tumor process can probably not be controlled by investigations along the lines which have proven effective in bacterial diseases. Vaccination by means of tumor derivatives has been practised on an enormous scale all over the world and has failed to justify itself. It has had some paradoxical success but has probably done more harm than good. No one has been able to improve upon Vidal's feeble showing for anticancer sera. Chemotherapy has never had any tangible basis in the tumor field, and from Weil's review it would seem to have even less claim to serious notice than has vaccination. Under these circumstances, I venture to raise the question whether it is not time to abandon this unprofitable territory and seek for help in entirely different directions. It may be that artificial alterations in the course of metabolic processes in the body, as suggested by recent dietary studies, may prove capable of influencing favorably the course of some malignant tumors. I freely confess the hope that the vegetable kingdom may be found to contain some agent that will specifically affect the cells of some tumors. It is the genius of vegetable products specifically and powerfully to affect different organs, tissues, and functions of the animal body, as exhibited by digitalis, strychnine, morphine, etc. Why should not some vegetable agent attack the delicately balanced nutrition of tumor cells? It would be extremely disconcerting, and even mortifying, if some vegetable alkaloid or glucoside were found to do more for cancer than all the theories of serum immunity, but the demonstration, if made, would have to be accepted.

At present, the only laurels in cancer therapy are being carried off by physical agents, x-ray and radium, and it seems to be only the difficulties of accessibility and dosage which stand in the way of the successful application of these agents to all localized and some generalized tumors. Cancer research should note that progress in the development of the x-ray is almost exclusively in the hands of elaborately equipped and far sighted business corporations, from whom one department of medicine receives orders when and how to proceed. This situation is not flattering to our dignity. With radium, the situation is somewhat different, since the study of the physics and therapeutics of
radium rests with Government organizations, universities, and private institutions endowed with a supply of this precious metal. It is not too much to hope that when the early stages of cancer are recognized as a proper field for the use of x-ray and radium, as has already occurred in the opinion of some competent authorities, much of the present fear of the disease, especially of the most deplorable post-operative recurrences, will be removed. In such an event, some of the present problems of cancer research will retain only an academic interest.

In the department of special etiology, experimental cancer research has secured some of its most significant results.

The chicken sarcomas are most suggestive from the point of view of their probable etiology and pathogenesis. Although there may be nothing like them in human pathology, they stand as a specific pathological entity and raise interesting questions regarding the etiology of other tumors. Borrel's consistent pursuit of animal parasites in tumors has enabled him and others to uncover the main factors in the causation of several tumors of lower animals and it is not impossible, although as yet unproved, that they may have counterparts in man. When Fibiger discovered a nematode worm in the gastric carcinoma of rats, and by a brilliant analysis identified and located this parasite in Nature and reproduced the disease experimentally, he established the existence of another specific disease of neoplastic character.

These contributions seem to me to point to the necessity of regarding all forms of neoplasms as specific diseases, connected only by the fact that they are neoplastic in greater or less degree, but differing in their etiology, clinical course, and therapeutic possibilities. In the same way, tuberculosis and bubonic plague are infectious diseases of inflammatory nature, but they are quite as closely related as Fibiger's gastric carcinoma of rats and pipe smokers' cancer of the lip. The habit of regarding cancer as a protean disease of uniform significance may well be abandoned in the interests of progress. When cancer research properly occupies itself in the study of the distinctive features of different cases of malignant disease, especially when it abandons the idea of a
universal cure for cancer, it will be in accord with sound pathological sense. It will then not be necessary to talk wisely to the public about the obscurities of cancer etiology, or speculate about why cells grow lawlessly. Concerning the ultimate nature of neoplastic overgrowth we shall never have more than a descriptive knowledge.

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