ADVANCES IN CANCER RESEARCH VOLUME 20

ADVANCES IN CANCER RESEARCH

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TUMOR CELL SURFACES: GENERAL ALTERATIONS DETECTED BY AGGLUTININS

Annette M. C. Rapin and Max M. Burger

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Department of Biochemistry, Biozentrum, University of Basel, Basel, Switzerland

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One of the most fundamental questions concerning malignant cells is: Why do they proliferate beyond normal limits and become invasive? This is the question posed in its most simple form by both layman and scientist.

Even though a host of researchers have been applying themselves to

this question for a number of years, it is still far from being answered. One concept that has emerged is that malignant cells show great alterations in their relationships, not only toward cells in the surrounding tissue which they may invade, but also toward each other. Kalckar (1965) has spoken of an altered "cell sociology" of malignant cells, stemming at least in part from their altered surface characteristics, and Pardee (1964) has pointed out that alteration of such membrane functions as transport could play an important part in control of growth.

Let us list briefly here some of the characteristics of tumor cells which are indicative of their having altered surface membranes:

A. In the organism

- 1. Tumor cells, whether they are part of a malignant or a benign tumor, multiply beyond normal limits, without being influenced by neighboring cells.
- 2. Most tumor cells do not cooperate with each other to form a tissue specialized for a certain function; individual cells thus become autonomous.
- 3. Although the chemical structure of cell surfaces is still poorly known, scrological studies have shown the antigenic makeup of tumor cells to differ in several respects from that of normal cells:
 - a. Appearance of tumor-specific transplantation antigens (TSTA) as well as virus-specific antigens on the surface of virally transformed cells (Klein, 1969),
 - b. Appearance in many cases of carcinoembryonic antigens or oncofetal antigens (Abelev, 1971; Gold and Freedman, 1965a,b),
 - c. Tumor cells are less differentiated than their normal counterparts; one manifestation of this is the decrease, or in some cases the total loss, of tissue-specific antigens (Weiler, 1959).
- B. In vitro. The possibility of transforming cells in vitro (by oncogenic viruses, or by chemical carcinogens) or the ability to select spontaneously transformed cells allows a comparison between normal and transformed cells originally stemming from the same tissue.
 - 1. A characteristic of untransformed tissue culture cells is their density-dependent inhibition of growth (DDI)¹ (Stoker and

¹ Abbreviations: cAMP, cyclic AMP = adenosine 3',5'-monophosphate; CMP, cytidine 5'-monophosphate; cGMP, cyclic GMP = guanosine 3',5'-monophosphate; cIMP, cyclic IMP = inosine 3',5'-monophosphate; cUMP, cyclic UMP = uridine 3',5'-monophosphate; DDI, density-dependent inhibition (usually of growth); diBcAMP, dibutyryl cAMP; DNP, dinitrophenol; Ig, immunoglobulin; PGE, prostaglandin E, RSV, Rous sarcoma virus; TAME, tosyl-arginine methyl ester (a protease inhibitor); TLCK, tosyl-lysyl-chloromethylketone (a protease inhibitor); TPCK, tosyl-phenylalanyl-chloromethylketone (a protease inhibitor). For abbreviations of lectin names, see Table I.

Rubin, 1967): At the stage where growing cells become confluent they cease dividing and do not continue to grow beyond the monolayer stage. Tumor cells escape this DDI of growth: contact does not prevent them from proliferating for a certain time, and they usually form disorganized multiple layers.

2. As first reported by Coman (1944) transformed cells are less adhesive toward each other and toward an external support. They can thus more easily grow in suspension, a property which in the organism would certainly be of advantage for their invasive and metastasizing growth (see also Abercombie and Ambrose, 1962).

3. Chemical and serological studies of surface membranes isolated from untransformed and from the corresponding transformed cells have shown a number of differences. Some of these studies will be

briefly reviewed in Section VI.

C. Increased agglutinability. Based on some previous reports by Easty et al. (1960), Aub and his collaborators reported in 1963 that a lipase preparation from wheat germ agglutinated tumor cells preferentially to normal cells. Until that time phytohemagglutinins or lectins, proteins usually isolated from plant seeds, had been used mainly for blood typing and for carbohydrate studies. This rested on Boyd's demonstration in 1945 (see Boyd, 1970) that the specificity of various agglutinins for different blood groups depends on a specific reaction between the lectin and the blood group carbohydrates located on the surface of erythrocytes, which was reminiscent of antigen-antibody reactions. The differential behavior of normal and tumor cells with an agglutinin was another indication for the existence of surface differences between these cells, and this agglutinin reaction has become a very useful tool for the study of cell surfaces. Attempts to find out how the transition from a normal and nonagglutinable cell to one that is transformed and agglutinable takes place, should yield insights into molecular alterations and rearrangements occurring with the transformation process. Studies along these lines have been going on for the last few years in our own and in several other laboratories. A few questions have been answered, but many more have been

This review will be concerned with studies on the agglutinin reaction, its use for the investigation of normal and of tumor cell surfaces, and with the process of transformation. It may not be amiss to stress at this point that this agglutination reaction is an artificial system since lectins are not present in the organism to interact with tumor cells. This should not detract from the usefulness of this reaction as a tool for the study of cell surfaces in general and of malignant transformation in particular.

It should be emphasized already at the beginning of this review that even though some correlations exist between changed surface characteristics of tumor cells and their neoplastic behavior, a causal relationship between these properties as well as an irrefutable proof of a direct involvement of the cell membrane in malignancy have yet to be conclusively demonstrated.

II. Correlation between Lectin Binding, Agglutinability, and the Transformed State

Following Aub's initial observation (Aub et al., 1963) that a crude wheat germ lipase preparation selectively agglutinated malignant cells, the active principle from such preparations, the protein wheat germ agglutinin (WGA), was isolated (Burger and Goldberg, 1967). This lectin did indeed agglutinate leukemia cells and cells transformed by the tumorigenic polyoma virus better than the corresponding untransformed cells. From carbohydrate inhibition studies it was concluded that the reactive group at the cell surface, probably a glycoprotein, contained β -1,4-di-N-acetylglucosamine (Burger and Goldberg, 1967). These initial studies opened the way to a large number of investigations in many laboratories, using various plant agglutinins, such as concanavalin A (Inbar and Sachs, 1969), soybean agglutinin (Sela et al., 1970), and a number of others (see Table I).

A review of these studies, which we shall present here, and which is summarized in Table I, leads to the conclusion that in general there is indeed a good correlation between tumorigenicity of cells and their ability to be agglutinated by plant lectins; like all generalizations, however, this one suffers a number of exceptions, and these will also be discussed here.

A. AGGLUTINATION STUDIES

Long before the discovery of the affinity of agglutinins for tumor cells, plant lectins were known to agglutinate red blood cells and Boyd found in 1945 (see Boyd, 1970) that they could be used for blood group typing [for historical review about this discovery, and for description of a number of lectins and their carbohydrate specificity, see Boyd (1970), Lis and Sharon (1973) and Sharon and Lis (1972)]. The ability of many lectins to agglutinate red blood cells from certain species shows that they are not exclusively tumor-specific (it should, however, be remarked in passing that human erythrocytes are not typical somatic cells). Aub himself followed his initial observations on the agglutinability of tumor cells (Aub *et al.*, 1963) by a series of tests on normal and tumor cells, and he found that although tumor cells consistently gave a

higher degree of agglutination than normal cells, the latter were often also agglutinated to a slight degree (Aub et al., 1965a,b).

Although in the great majority of cases it is tumor cells and transformed cells that are agglutinated by lectins, a few cases have also been reported where the normal cells, not their transformed counterparts, agglutinated better. This, however, seems so far to be a particularity of the interaction of lentil (*Lens culinaris*) agglutinin with normal liver cells and with rat hepatoma cells (but not with other types of cells, Borek *et al.*, 1973; see also Section III,B,4).

In several instances no difference in agglutinability could be observed between normal and tumor cells (see Table I). In these cases transformation has not changed the availability of a certain receptor site; this, however, does not preclude modification of sites on the same cells for other lectins. An example of this phenomenon is the agglutinability of normal lymphocytes as well as lymphoma cells by the red kidney bean agglutinin PHA (Phaseolus vulgaris agglutinin), whereas concanavalin A (con A) agglutinates the lymphoma cells only (Inbar et al., 1973a). It should also be kept in mind that different types of cells differ in their surface components and consequently in their ability to interact with different lectins [L1210 leukemia cells,2 for instance, are very well agglutinated by WGA but not by Con A (Burger, 1973), whereas Yoshida sarcoma cells are agglutinated by Con A and by PHA, but poorly by WGA (Tomita et al., 1972b); cells from a rat ascites hepatoma are agglutinable by WGA, but practically not by Con A (D. F. Smith et al., 1973)].

Interesting are also the cases where cells have become agglutinable after infection with nononcogenic viruses: Infection of baby hamster kidney (BHK) cells by Newcastle disease virus resulted in a thinning of the cell coat and agglutinability of the cells by Con A and WGA; a viral mutant which did not produce this thinning of the cell coat did not render the infected cells agglutinable (Poste and Reeve, 1972). Becht et al. (1972) showed that cells infected with a variety of nononcogenic enveloped RNA viruses, such as Sindbis, vesicular stomatitis, or influenza virus, became agglutinable by Con A concomitantly with the appearance of hemagglutinin on their cell surface; cells infected in

² Description of cells mentioned in this review: 3T3, mouse embryo fibroblasts; Py3T3, polyoma virus-transformed 3T3 cells; SV3T3, simian virus 40 transformed 3T3; RSV3T3, Rous sarcoma virus-transformed 3T3; BHK (or BHK21), baby hamster kidney fibroblasts; CHO, Chinese hamster ovary cells (a dedifferentiated, epithelioid cell line); HeLa, a line derived from a human epithelioid carcinoma (cervical); KB, a line derived from a human carcinoma (oral), epithelioid; L cells, mouse fibroblasts; L1210, mouse leukemia cells; NIL, hamster fibroblasts.

Membrane-bound Con A

mobility (70)

Lymphoma cells agglutinated better than normal lymphocytes (37)

control are less agglutinable

TABLE I*

Some Agglutinins and Their Properties
(Numbers in parentheses correspond to references at bottom of table. Complete bibliographical references will be found at end of review.) Part A

Asjn Tojn Tipel Tipel	A Sets	oir line O g g		star urg	Agglutination	o i dili di oo	di , Agj mu m m plar of init
Lectin and source	Isolation and purification	Specific	Normal cells (other than erythrocytes)	Normal cells + proteases or in mitosis	Transformed cells	References	Other properties
Concanavalin (Con	Concanavalin (Con Isolation, purifica- cc-p-Glucopyrano- A) from lack tion, and char-sides, cc-p-man-	α-D-Glucopyranc- sides. α-p-man-	0, but binds to	oine ily-i ctli ctli	s pi nph s ag	5, 6, 8, 10, 12, 14-	5, 6, 8, 10, 12, 14- Isolation of cell mem- 16, 19, 20, 25- brane receptor (3, 4,
bean (Canavalia	acterization (1, 17, 22, 38, 59,	nopyranosides,	1979 1979 1978		thi lyi gari ipho	28, 33, 36, 40, 42, 45, 48-50.	72, 86) Agglutination is temper-
trait trait trait mon	60, 74, 75, 82)	osides (a-meth-	di.			54, 61, 68, 71,	ature dependent (35,
este de la de la	(74, 75)	erally used (29)	Some lines +,		Some lines +,	26, 45, 47, 66, 67	Agglutination depends
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in (nó	(46, 82)	n display	N SO		sitive mutants	55, 65	Con A is mitogenic (43,
one of the last of	Con A not a gly-	th lls he	To		agglutinable only at permis-	t h	44, 56, 64) Stimulation of lympho-
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Fetal cells are agglutin- able (24, 48, 51, 73, 84)	Cells infected with some	viruses are agglutin-	able (7, 11, 63, 76, 89)	Č .		restores growth con-		Agglutination depends on		(9, 23, 88) Con A			mobility (30, 87)	T		injection are more		vasive (18)	Ö	hamster egg fertiliza-		B		cell division (57)	Bones (Continued)
6, 12			85	TOD IN A OF RE	SV. Lahara and Ed	32			S2 Want of all	81 I Jodanisky st		19. Vesely stal.				A2' punited and H						Et best lichardes 188		but mondad to	nna liewot , £8 H) regardf , 68
Normal, confluent cells + protein	synthesis inhib- itors are agelu-	tinable	Normal cells	treated with	urea are agglu-	CHO cells con-	verted to fibro-	blasts are less	agglutinable	2fr Noomer and Bruket (fall)		48. Micobion (1871)	AS MORCOAL (1977)			AP Mother (1821)	44: Macklot 4 al. (1973)	Carlos Siesens (2013)			(C) Kapeller and Bolismaki (1972)	30. Kaneko w uf. (1943)	38. Jones and Johns (1916)	Cast of the terms	(000) As a maint set (470) As a sadad Set
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* Parts A-F of Table I appear on pages 6-17.

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 - 3. Akedo et al. (1972) Allan et al. (1972)
- Arndt-Jovin and Berg (1971)

0

- Baker and Humphreys (1972)
 - Ben-Bassat et al. (1971) Becht et al. (1972)
- Berlin and Ukena (1972)
- 10. Biquard and Vigier (1972a,b) Birdwell and Strauss (1973)
 - Burger and Martin (1972) 12. Borek et al. (1973) 13.
- 15. Cline and Livingston (1971) Burger and Noonan (1970) 14.
 - 16. Culp and Black (1972)
- 17. Cunningham et al. (1972)
 - de Micco et al. (1973) 8
 - de Petris et al. (1973) De Salle et al. (1972) 19. 20.
- 22. Edelman et al. (1972) Eckhart et al. (1971)
- 23. Edelman et al. (1973) 24. Francois et al. (1972)
 - 25. Friberg et al. (1971) 26. Friberg et al. (1972)
- Furmanski et al. (1972) Glick and Buck (1973)
 - Goldstein et al. (1965) Gunther et al. (1973)

33. Inbar and Sachs (1969) 31. Hadden et al. (1972) 32. Hsie et al. (1971)

61. Ozanne and Sambrook (1971a) 62. Ozanne and Sambrook (1971b)

Poste and Reeve (1972) 64. Powell and Leon (1970)

- 34. Inbar et al. (1969)
- Inbar et al. (1971) 35.
- Jones and Johns (1916) Inbar et al. (1973a) Inbar et al. (1972a) 36. 37. 38.

Salzberg and Raskas (1972) Schnebli and Burger (1972)

Salzberg and Green (1972)

Renger (1972)

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- Kapeller and Doljanski (1972) Kaneko et al. (1973) 40. 39.
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Steinberg and Gepner (1973)

Shoham and Sachs (1972) D. F Smith et al. (1973)

Shinitzky et al. (1973) Salzberg et al. (1973)

Sumner and Howell (1936)

Sumner (1919)

- Mackler et al. (1972) Mallucci (1971) 14. 15.
- Moore and Temin (1971) McKenzie et al. (1972) Moscona (1971) 17. 16. 18

78. van der Noordaa et al. (1972)

 Veselý et al. (1972)
 Vlodavsky et al. (1972) Vlodavsky et al. (1973)

Wang et al. (1971)

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77. Turner and Burger (1974)

Tevethia et al. (1972)

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- Novogrodsky and Katchalski (1971) Noonan et al. (1973b) Noonan et al. (1973a) 54. 55. .99

Weston and Hendricks (1972)

85.

Weiser (1972) Weber (1973)

Yahara and Edelman (1972)

88. Yin et al. (1972)

Wray and Walborg (1971)

- 58. Oikawa et al. (1973) O'Dell (1972)
- Olson and Liener (1967a) Olson and Liener (1967b)

89. Zarling and Tevethia (1971)

TABLE I (Continued) Part B

				Agg	Agglutination		
Lectin and source	Isolation and purification	Specific	Normal cells (other than erythrocytes)	Normal cells + proteases or in mitosis	Transformed cells	36 30 References	Other properties
Soybean agglutinin (SBA), from soy- bean (Glycine max)	Isolation, purification, and characterization (2, 5-9, 11, 17) MW of tetramer: 110,000 (6, 8, 11, 17) Is a glycoprotein (8, 17) Four isolectins	N-Acetyl-D-ga- lactosamine (D- galactose is a weaker inhib- itor) (10)	0, but binds to normal cells 0	+ +	Transformed ham- ter embryo cells agglutinated only after pro- tease treatment	3, 12, 13 12	Agglutination is not temperature dependent (3, 4) Agglutination does not depend on cellular ATP content (16) Is not mitogenic (4) Normal lymphone cells are aggruphynthese and lymphone cells are aggruphynthese cells are aggruphinated to same
ban altos.l	isolated (7)	olking/P rout/lidge	alley Israeo V. meda vedato) (aggretotals cos	fantyold allen espandoug al- abolim miro	beninotanin's alon_\		degree (4) Trypsinization of erythrocytes increases binding and agglutinability (1.15)
				yang.	RSportuation		Membrane-bound lectin has relatively low mobility (14)
Key to references for Table I, 1. Gordon et al. (1972a) 2. Gordon et al. (1972b) 3. Inbar et al. (1971) 4. Inbar et al. (1973a) 5. Liener and Pallansch (1952)	s for Table I,B: 1972a) 1972b) 771) 773a)	7.7 8.7 9.1 10.1	7. Lis et al. (1966a) 8. Lis et al. (1966b) 9. Lis et al. (1969) 10. Lis et al. (1970) 11. Pallansch and Liener (1953)	r (1953)	13. 14. 15. 16.	 Sela et al. (1971) Shinitaky et al. (1973) Vlodavsky et al. (1972) Vlodavsky et al. (1972) Wada et al. (1973) Wada et al. (1973) 	1973) 1972)

TABLE I (Continued)
Part C

				Agg	Agglutination		Mentalatively low
Lectin and source	Isolation and purification	Specific	Normal cells (other than erythrocytes)	Normal cells + proteases or in mitosis	Transformed cells	References	Other properties
Wheat germ agglutini (WGA), from wheat germ (Triticum vulgaris)	Isolation, purifica- Di-N-acetylchi- tion, and charac- tobibse; N-ace terization (1, 2, tylghucosamin 8, 10, 25, 27, ovomucoid (2, 30-32, 40) 8, 10, 16, 45)	Di-N-acetylchi- tobibse; N-ace- tylglucosamine; ovomucoid (2, 8, 10, 16, 45)	0, but binds to normal cells	+	+ Percent (Liber cannel)	3-5, 8-10, 13, 14, 17, 19, 22-24, 29, 35, 36, 39 41, 46, 48	Isolation of cell membrane receptor (7, 21, 44, 50) Agelutination not temperature dependent
nfministerm medecel succession (ASSA) succession succes	Crystallization (30, 32) MW of dimer: 35,000 (2, 31) Not a glycopro-	ega o tybeb. W. o) solested to a composite a composit	Some lines +,	+ +	Some lines +, some lines - Temperature-sen- sitive mutants agglutinable	4, 15, 26, 28 11, 12, 33, 38	(19, 20) Agglutination not dependent on cellular ATP content (47) Nonmitogenic (20)
fun titoe!	tein (1, 2, 31) Three isolectins 'separated (2, 27)	untrough not solides	Marrial della (Application of the Change Cha	Europid elso reasetord +	only at permissive temperature Cells having regained growth control are less	. 36, 39	Normal lymphocytes and lymphoma cells agglu- tinated to same degree (20) Membrane-bound lectin
			Normal, confluent cells + protein synthesis inhib- itor are agglu- tinable	boundario O) I	agglutinable	9	has relatively low mobility (43)