jected towards the end of pregnancy, two aborted and six delivered sickly litters. Many of these baby mice were eaten by their mothers but a few survived and showed patchy defects of the fur similar to those brought about by direct injection. The most regular damage of the aborted foetus, as revealed by histological examination, were scattered necrotic foci in the liver without leucocytic infiltration.

In a further experiment, 10 females in the middle of pregnancy were subcutaneously injected with half slope agar cultures and killed 24 h later. In six of them abortion had already begun by that time. Cultures were performed from the liver and spleen of the females on the one hand, and from the embryos on the other. No *Haemophilus* was found in the organs of the mother animals nor was there any subplacentar hemorrhage. The abortion could not, therefore, be related to maternal injury. Out of 66 embryos, 13 yielded a rich confluent growth, 8 showed isolated colonies and 45 were sterile. The distribution of these findings is given in table No. II.

Table II
Presence of *Haemophilus* in embryos of pregnant mice infected 24 h before examination

Serial No. of female	Embryos			
	Total number	Confluent growth	Isolated colonies	Sterile
1	7	7	–	–
2*	6	–	1	5
3*	4	–	4	–
4	10	–	–	10
5*	7	–	–	7
6*	8	6	2	–
7*	6	–	–	6
8	7	–	–	7
9	4	–	–	4
10*	7	–	1	6
Total	66	13	8	45

* Abortion had already started before examination.

The bacteriological findings of the placentas, cultured separately, did not differ from those of the corresponding foetus. Experiments with intravenous injections of 1/10 slope agar cultures gave even more impressive results. After 24 h, the organs of all the females were sterile whilst the Haemophilus could be recovered from the embryos in most of the litters.

In conjunction with our previous results, these findings may be interpreted as follows: The embryos are killed by the liberated endotoxins to which they are more sensitive than the adult animals. If bacteria penetrate through the placental barrier into the foetus, they establish a foothold in the impaired embryonic and placentar tissue, whilst they are quickly destroyed in the adult animal by cellular defence mechanisms. Humoral antibodies would have acted equally in both, mother and foetus, since they pass unhindered through the placenta in rodents, apes and man[1].

The susceptibility of infants to *H. influenzae* infections cannot, therefore, be explained solely by a lack of humoral immunity. The primary cause appears to be a particularly high vulnerability of the immature organism to the endotoxins of Haemophilus which seem to paralyze its cellular defence. The exact nature of this process still requires elucidation. It would be even more interesting to investigate, whether a difference in vulnerability, similar to that between foetus and mother, pertains also to other immature tissues within a mature organism, i.e. in tumor-bearing animals. Should this be confirmed, the Haemophilus toxin might be considered for trial as an anticancerous agent.

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Zusammenfassung

Mäuse zeigen in den ersten zwei Wochen ihres Lebens, im Vergleich mit erwachsenen Tieren, eine ähnlich erhöhte Empfindlichkeit gegenüber Allgemeininfektion mit *Haemophilus influenzae* wie menschliche Kleinkinder in den ersten zwei Jahren. Diese Erscheinung beruht vermutlich in erster Linie auf einer besonders hohen Empfindlichkeit des infantilen Organismus gegenüber den Endotoxinen, welche die zellulären Abwehrfunktionen hemmen. Noch empfindlicher sind Föten in der Mitte der Gestationsperiode, die sämtlich abgetötet werden, während das Muttertier keinerlei Schädigung erleidet.

Es wird angeregt, zu untersuchen, ob eine ähnliche Verschiedenheit der Empfindlichkeit zwischen den unreifen Zellen von malignen Tumoren und den reifen ihrer Träger besteht.

Der Rucktanz als wesentlicher Bestandteil der Bienentänze

Vor einigen Jahren berichteten wir über richtungsweisende Bienentänze bei Futterplätzen in Stocknähe[1]. Es stellte sich heraus, dass bei einer Entfernung des Futterplatzes von zum Beispiel 30 m sowohl Sicheltänze (BALTZER, TSCHUMI) als Acht- und Schwänzeltänze (v. FRISCH) nebst Übergangstanzformen ausgeführt wurden. Dazu kamen noch regellos eingestreute Rundtänze und ungerichtete Sicheln. Trotz der starken Variation schienen die Tänze, wie dies auch TSCHUMI[2] feststellte, eine ausgesprochen richtungsweisende Funktion zu erfüllen. Letztere kann deshalb unseres Erachtens nur auf den im Jahre 1950 von uns beschriebenen «Rucktänzen» beruhen, die unter anderem auch die «Wendepunkte» BALTZERS[3] markieren. *Nur diese Rucktanzstrecken können durch Länge und Richtung die Lage der Futterquelle anzeigen*, entsprechend den von v. FRISCH entdeckten Regeln. Die zwischen den Rucktänzen eingeschalteten Laufstrecken haben, ebenso wie die Rundtänze und die ungerichteten Sicheln, eine aktivierende Bedeutung. Ihr Auftreten ist einerseits durch örtliche Umstände auf der Wabe bedingt, andererseits durch das Temperament des Volkes. Letzteres kann verschieden sein, und zwar sowohl *vorübergehend* durch Einflüsse, wie Wetter, Qualität der Futterquelle, Stärke und Entwicklungszustand

[1] J. H. MASON, T. DALLING, and W. S. GORDON, J. Path. Bact. *33*, 783 (1930).

[1] G. HEIN, Exper. *6*, 142 (1950).

[2] P. TSCHUMI, Rev. suisse Zool. *57*, 584 (1950).

[3] F. BALTZER, 12. Jber. Schweiz. Ges. Vererbungsforsch. *27*, 197 (1952).

des Volkes, Alter der Königin, als auch *bleibend* verschieden durch erbliche Unterschiede zwischen den Rassen oder einzelnen Völkern.

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Summary

Bee dances indicating the position of feeding places situated at short distances from the hive show a great variability. They have only one element in common which seems suitable to this purpose: the "pull-dance" (Rucktanz), described by us earlier.

Method for the Quantitative Evaluation of Platelet Function in Blood Coagulation

The role of platelets in blood coagulation has been characterized in connection with the identification of several active agents supplied by the platelets themselves: (1) platelet thromboplastic factor[1]; (2) platelet factor 1, presenting Ac-globulin activity[2]; (3) platelet factor 2, which accelerates the conversion of fibrinogen into thrombin[2]; (4) platelet factor 3, or antiheparinic factor, which is probably similar or identical with the thromboplastic factor[3].

Quantitative or qualitative alterations of platelets may be revealed by the incomplete transformation of prothrombin into thrombin (positivity of the prothrombin consumption test). This test is, however, positive also in various other conditions, and its limited specificity is often the cause of diagnostic difficulties.

For the quantitative evaluation of platelet function as a whole, a method has been worked out which is based on the functional study of isolated platelets in a clotting system. The principle of the method is the following: all factors are kept constant, except the factor to be determined, i.e. platelet functional activity, according to this scheme:

Constant
Prothrombin
Antihemophilic globulin and allied factors
Ac-globulin or proaccelerin
Factor VII or proconvertin or SPCA
Calcium
Platelet number (300,000/cu.mm)

Variable
Platelet functional activity

The substrate containing all factors to be kept constant is obtained by preparing a strictly platelet-free plasma (high speed centrifugation, cold, siliconed glassware and needles[4]). Calcium is supplied in known amounts.

The most important point of the method is the preparation of the platelet suspension. In previous research, exact quantitative tests have not been employed[1], partly because the preparation of non-agglutinated platelet suspensions containing a fixed amount of platelets presents some technical difficulties. To avoid agglutination and to perform an accurate count of the platelets, it is indispensable to use throughout: (*a*) arquad coated needles; (*b*) siliconed coated glassware; (*c*) sequestrene as anticoagulant; (*d*) triton and sodium acetate for preserving the platelets. The details of this technique are described by STEFANINI *et al.*[2] and have been followed exactly. Differential centrifugation and repeated washings of platelets have to be carried out rapidly.

By employing such methods, it is possible to prepare suspensions of non-agglutinated platelets and to bring their concentration to 300,000/cu.mm with sufficient accuracy. The test for the quantitative evaluation of platelet function in blood coagulation is carried out as follows; 0·1 cm³ of the platelet suspension is added to 0·1 cm³ of platelet-free plasma. One tenth cm³ of $CaCl_2$ 0·025 *M* is added to this mixture and the clotting time at 37°C is recorded. By using serial dilutions of the platelet suspension, a standard curve is obtained which makes it possible to evaluate the percentages of platelet activity. The recalcification time of the platelet-free plasma, to which the undiluted platelet suspension has been added, is considered as 100%. If saline is added instead of platelet suspension, the values of the recalcification time refer to 0% platelet activity, and should be more than 6–8 min at least. The percentage of reduction of the clotting time is converted into percentage of platelet functional activity (Fig. 1).

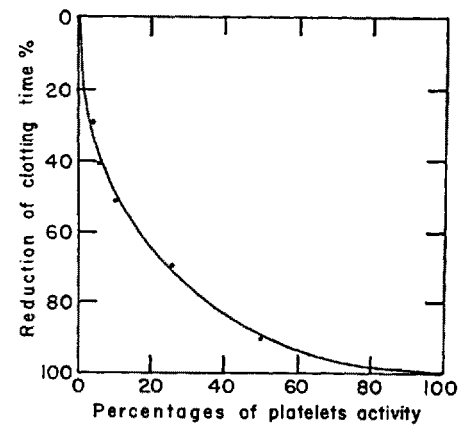

Fig. 1.–Standard curve for the evaluation of the percentages of platelets activity from the reduction of clotting time.

The same test might be carried out by using a two-stage method, as already observed by one of us[3]. High concentrations of purified prothrombin (3000 units/cm³), platelet-free plasma and optimal calcium amounts are used for this purpose. Platelet suspensions may be prepared according to the same procedure.

Preliminary observations carried out by using this method[4] have shown that variations of platelet func-

[1] M. STEFANINI, Amer. J. Med. *14*, 64 (1953).

[2] A. G. WARE, J. L. FAHEY, and W. H. SEEGERS, Amer. J. Physiol. *154*, 140 (1948).

[3] S. VAN CREVELD and M. M. P. PAULSSEN, Lancet *2*, 242 (1951).

[4] J. BERNARD, J. L. BEAUMONT, and M. CL. CHARREYRON, Rev. Hématol. *8*, 20 (1953). – J. P. SOULIER and M. J. LARRIEU, J. Lab. Clin. Med. *41*, 849 (1953).

[1] J. BERNARD, J. L. BEAUMONT, and M. CL. CHARREYRON, Rev. Hématol. *8*, 20 (1953). – J. P. SOULIER and M. J. LARRIEU, J. Lab. Clin. Med. *41*, 849 (1953). – A. J. QUICK, W. F. STAPP, and C. V. HUSSEY, J. Lab. Clin. Med. *39*, 142 (1952). – L. N. SUSSMAN, N. WALD, and R. L. ROSENTHAL, Blood *7*, 1100 (1952).

[2] M. STEFANINI, W. DAMESHEK, J. B. CHATTERJEA, E. ADELSON, and I. B. MEDNICOFF, Blood *8*, 26 (1953). – M. STEFANINI and W. DAMESHEK, New England J. Med. *248*, 797 (1953).

[3] S. A. JOHNSON and P. DE NICOLA, Unpublished observations.

[4] P. DE NICOLA, P. ROSTI, and C. CARCUPINO, Proceed. IV. Congr. European Soc. Hematol., Amsterdam, Sept. 8–12, 1953.