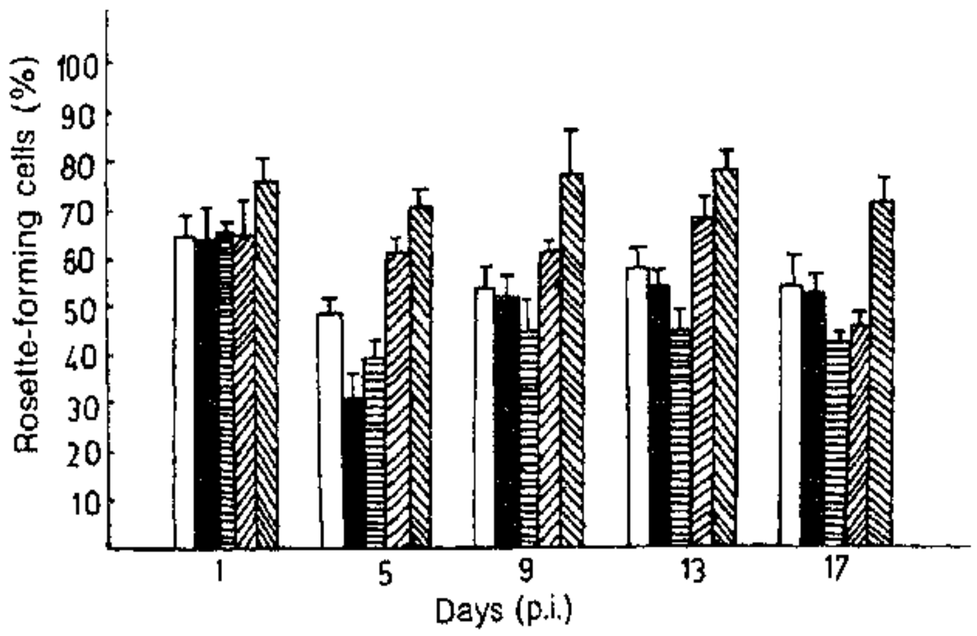

Figure 2. Pattern of attachment of opsonized sheep erythrocytes by peritoneal macrophages on different days after inoculation of TS1 (□); TS2 (■). JEV and TS1 (▤); JEV (▨) or uninoculated control (▩). Mean value ± SD presented.

functions. Figure 2 shows that following Ts_1 cell inoculation 64±6% of macrophages attached EA on day 1 which slightly decreased on day 5 (48±2.6%) and then recovered. A sharp decline in the capacity of macrophages to attach EA (31±7.0%) was observed on day 5 after Ts_2 administration which increased thereafter to 52±1.5% but remained below the control values throughout the study period. In control mice 70–78% of macrophages showed attachment of EA.

In another experiment the combined effect of JEV and Ts_1 on Fc-receptor-mediated attachment of EA by peritoneal macrophages was studied (Figure 2). In mice given JEV and suppressor cells simultaneously, the binding of EA by macrophages was reduced significantly compared to that of mice receiving JEV alone or Ts_1 cells alone on day 5. The values remained low throughout the study period in comparison with controls.

Effect of SF on Fc-receptor-mediated attachment by macrophages

Groups of mice inoculated with SF (1:50) i.p. were sacrificed at different periods and the capacity of macrophage to bind opsonized SRBC through Fc receptor was studied. Figure 3 shows that in the normal control group the percentage of rosette forming Mϕ was 78±6% while that in SF inoculated groups was reduced throughout the study period with the mean value of 49±5%.

The effect on attachment of opsonized SRBC by Mϕ on different days after simultaneous i.p. inoculation of SF (1:50) and JEV was studied. A sharp decline in the proportion of cells binding to opsonized SRBC was seen 11 to 13 days p.i. being 18 to 20%. The Fc-receptor-mediated attachment values recovered gradually (Figure 3).

In vitro effect of SF on Fc-receptor-mediated attachment

In vitro effect of SF on the macrophages to suppress

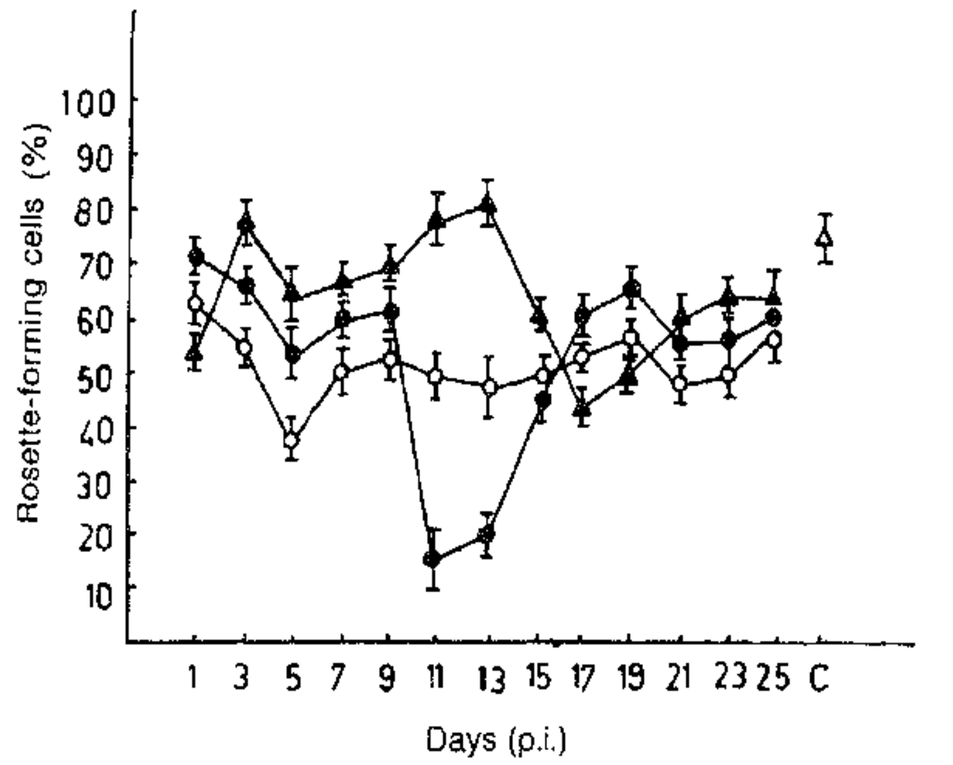

Figure 3. Pattern of attachment of EA on peritoneal macrophages on different days after inoculation of SF and JEV given at the same time (●), or SF (○), or JEV (▲), or uninoculated control (△). Mean value ± SD presented.

their binding capacity for the opsonized SRBC was studied. The Mϕ cell sheet was incubated with different dilutions of SF for 1 h at 37°C and then washed. Table 1 shows that the rosette-forming capacity of Mϕ remained unaltered as there was no difference in the binding capacity of control (84.3±0.98%) and different dilutions of SF-treated macrophages.

Discussion

The results show that during JEV infection, the Fc-receptor-mediated attachment of EA to mouse macrophages was depressed. The macrophages lost their ability to bind EA markedly on day 18 p.i. At later periods this recovered slightly but remained significantly lower than the controls.

The depression of Fc-receptor functions of macrophages can be caused by different mechanisms, viz. immobilization of the receptors[12,13]; selective internalization of macrophage membrane[14]; production of antibody to macrophage surface by host[15]; decreased contents of cyclic AMP-dependent protein kinase[5]; killing or functional damage of macrophages by the

Table 1. *In vitro* effect of SF on Fc-receptor mediated attachment.

Group	Dilution	% positive* ± SD
Normal	—	84.3±0.98
SF	1:20	81.3±3.74
SF	1:50	79.7±0.7
SF	1:100	82.3±1.5

Mϕ monolayers prepared from normal mouse peritoneal fluid were incubated with different dilution of SF for 1 h at 37°C and then assayed for Fc-receptor-mediated binding capacity.
Mean ± SD presented.
*Macrophages with 3 or more than 3 SRBC's attached on it were considered positive.

virus-induced cytotoxic factor[16] etc. While investigating the cause of depressed Fc-receptor-mediated attachment in JEV infection it was realized that the period of alteration of the EA binding coincides with the appearance of supressor T cells in such mice[7].

We have observed earlier that JEV infection in mice generates two suppressor pathways; one mediates suppression of antibody production and the other delayed type hypersensitivity immune response[6]. Maximum suppressor activity was noticed between day 18 and day 20 p.i. The first generation of JEV-induced suppressor T cells (Ts_1) produce a suppressor factor. SF recruits a second generation of Ts_2 cells, which transmits suppressor signal through production of SF_2[8]. Therefore, an attempt was made to investigate the effect of suppressor T cells or their products on the Fc-receptor functions of macrophages. We have observed the lowest values of EA attachment by macrophages on day 18 following JEV infection. At later periods the values increased slightly but continued to be less than those in uninfected control mice. Further, the treatment of mice with JEV-induced Ts_1 cells had slight effect while the Ts_2 cells produced a significant decline in Fc-receptor-mediated attachment of EA by macrophages on day 5 p.i. (Figure 2). Similarly when JEV and SF were given together the attachment values showed depression earlier than those of mice given JEV alone. Thus suppressor cells induced by JEV appear to regulate the Fc-receptor-mediated attachment of EA on macrophages through suppressor factor and may be a mechanism for prevention of virus infection of macrophages. Lymphokines are known to activate macrophages and enhance their functional capabilities, including the Fc-receptor functions[3]. It is interesting to note that of the two antigenically close flaviviruses, the JEV and the dengue, the soluble T cell products depress the macrophage functions by two different mechanisms. In DV infection it is mediated by a cytotoxic factor which is a T cell product and kills the macrophages[16]. On the other hand in JEV infection the SF (a non-cytotoxic product of T cell) brings about the same effect through functional disturbance of the macrophage. Suppressor factors generated in different models have been shown to bind to the surface of macrophage and transmit the signal to the target cells[17,18]. In JEV model also, macrophages have been shown to be obligatory for transmission of signal between Ts_1 and Ts_2 cells by the SF[8]. During this process, the Fc-receptor functions probably get disturbed. Thus, suppressor cells may control Fc-receptor-mediated attachment and help in prevention of virus infection to macrophages.

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A note on tunable transconductance of a branching molecular wire

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It is shown that the Griffith boundary conditions on the wavefunction of an electron moving coherently on a molecular network of one-dimensional conductors (delocalized bonds) lead to a branching resistance at the node that can be tuned resonantly. The latter provides a mechanism for regulating the transfer of an extraneous electron over a 'molecular wire' connecting two components of a supramolecular device.

IT is well known that the quantum chemistry of conjugated and aromatic molecules is well described by the simple 'network model' in which the molecule is replaced by a network of straight lines (bonds) connecting neighbouring nodes (atoms), providing pathways for the motion of 'free electrons[1,2]. The potentials are effectively replaced by the boundary conditions on the wavefunction, namely that the latter should be single-valued and the sum of its outward derivatives should add to zero at each mode[3]. This network model, sometimes also referred to as the free electron model, leads to resonance energy, bond order and excitation energy values to within a few per cent of those calculated by the method of molecular orbitals.

Early applications of this simple network model were concerned primarily with the energetic aspect of the problem. Recently, the emphasis has shifted to another aspect, namely, that of charge transport by these conjugated chains leading to the concept of the 'molecular wire', e.g. bispyridine polyenes[4]. For the purpose of this note we can fix our attention on the archetypal linearly conjugated trans-polyacetylene $(CH)_n$. Here there is a covalent backbone of σ-bonds resulting from the planar sp^2-hybridization, while the π-orbitals overlap laterally to give a nominally half-filled band of delocalized π-electrons. This metallic state, of course, assumes complete resonance *sans* broken symmetry. The second possibility is that of dimerization that can be visualized chemically as bond alternation (alternating single and double bonds) resulting in a broken symmetry. Physically this is the Peierls state with a gap opening at the Fermi-level (i.e. a semiconductor). Now, an extraneous electron injected into the chain can propagate as in a doped semiconductor, or more interestingly, it can lead to a charged (spinless) or neutral (spin 1/2) soliton (i.e. soliton doping). Thus, in all the three cases we can have a mobile charged object that translates along the chain and is subject to particle quantum mechanics. Hence a molecular wire.

Let us consider the transconductance of a branched molecular wire due to these charged particles. Figure 1*a* shows a hypothetical conjugated chain (1, 2) branched (bifurcated) at the node (3) with (3,4) as the side-chain. Figure 1*b* shows the equivalent network for this branched molecular wire. Let the charged particle be injected at the terminal 1 at energy $E=\hbar^2k^2/2$ m with k as the wave-vector. It is partially reflected (amplitude reflection coefficient r), partially transmitted (t) with the different partial waves as shown in Figure 1*b*. All distances are measured away from the node (3) as the origin. Thus we have the Griffith boundary conditions[3]

$$\psi_1(0)=\psi_2(0)=\psi_3(0), \qquad \text{(single valuedness)}$$

$$\sum_i \frac{\delta\psi_1}{\delta x_i}\bigg|_{\text{node}}=0, \qquad \text{(Kirchhoff law)} \tag{1}$$

with

$$\psi_1=\exp(-ikx_1)+r\exp(ikx_1)$$
$$\psi_2=2s\cos k(l-x_4)$$
$$\psi_3=t\exp(ikx_2). \tag{2}$$

Solving for the transmission coefficient $T\equiv|t|^2$, we get

$$T\equiv|t|^2=1/(1+\tfrac{1}{4}\tan^2 kl). \tag{3}$$

It is convenient to introduce transconductance as given by the Landauer formula[5]

$$G=\left(\frac{e^2}{2\pi\hbar}\right)\frac{T}{1-T}=\left(\frac{2e^2}{\pi\hbar}\right)\cot^2 kl. \tag{4}$$

For eq. (4) to hold we have to assume that the molecular wire is terminated at the two ends 1 and 2 into electroactive reservoirs where the particle can be

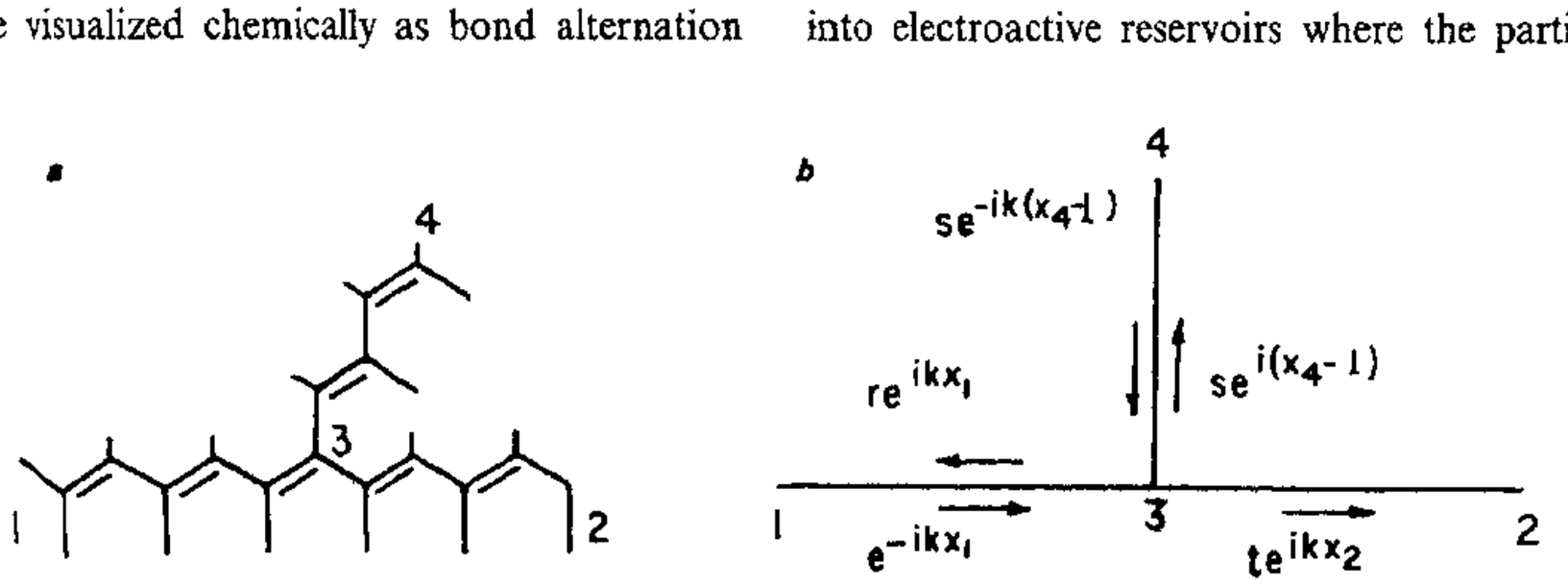

Figure 1. *a*, Hypothetical branched conjugated chain. *b*, Equivalent network showing partial wave amplitudes.

injected or removed reversibly. The expression (3) is, of course, valid generally and is central to our discussion.

As is readily seen, the transmission is resonantly tuned by the side-chain length l. Most importantly, the electron transfer can be blocked for certain tuned values of the side chain length, i.e. for $\cot kl = 0$. This suggests the intriguing possibility of a critical regulation of electron transfer between sites of reduction and oxidation in a supramolecular structure. It has been suggested that long-distance electron transfers are involved in the enzyme catalysis of biological activity and, indeed, specific pathways for electron transfers have been proposed[7]. What we are suggesting here is that in such cases a critical regulation, or control of the action, can be achieved by a relatively small molecule acting as a side-chain of suitable length located on the pathway.

Finally, for this purely quantum-interferential control to be effective, a long coherence length is required at physiological temperatures. This favours the solitonic mode of charge transport along the conjugated chain.

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Occurrence of twinning and parallel growth in zircons of the Palampur granitoids, northwestern Himalaya

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Well-developed geniculate and less regular types of twinning and two types of parallel growth have been observed in zircons of the Palampur granitoids. The crystallization histories of such crystals have been deduced on the basis of morphological features. These primary growths are considered to be of magmatic origin, and have formed at a late stage in the crystallization history of zircons.

ZIRCONS sometimes show various kinds of primary and secondary growth features. The primary growths are in the form of parallel, necked and twinned crystals. Such growths are quite rare in zircons and are considered to be characteristic of magmatic granitoids[1,2]. Secondary growths occur in the form of enhanced overgrowths, outgrowths and multiple growths. These growths are relatively common in zircons and are characteristic of anatectic or metasomatic granitoids[2-6].

During investigations on Palampur granitoids[7], well-developed twinning and parallel growths have been noticed in the zircons; these are recorded for the first time from the Himalayan granitoids. It may be mentioned that such growth features are extremely rare in the literature.

Two types of twinned zircons were observed in the rocks under study. These are: (i) the geniculate or elbow type of twinning (Figure 1*a*, *b*) with (101) or (011) as composition plane. These twins are quite comparable with British Museum (Natural History) twinned zircons as reproduced in Figure 2:9 of Jocelyn and Pidgeon[1]; (ii) these crystals are of a less regular type of twins (Figure 1*c*–*e*). In this case, the zircons are no longer symmetrical about the composition plane which itself penetrates partly into the crystal. These examples of twins are comparable with Figure 1:6–9 of Jocelyn and Pidgeon[1].

The morphological outlines of the above described zircons indicate that in the case of geniculate twins, initially the crystals had separate growth histories, and later on they joined together to form geniculate twins. On the other hand, in the case of less regular twins, it seems that both the crystals had joined together at an earlier stage of crystallization, and they then grew separately to form twins. These twinned crystals therefore, are likely to have formed at a late stage in their crystallization history.

Zircons of the Palampur granitoids exhibit two types of parallel growth: (i) composite parallel grown zircons lying on face 100 and united along (100) composition plane (Figure 1*f*–*h*). Such zircons show parallel or nearly parallel extinction, as the C-axes of both the crystals are parallel; (ii) composite parallel grown zircons lying on prismatic face 110 or 100 and showing a tendency to join along a pyramidal face (Figure 1*i*). The plane along which such crystals are united is somewhat irregular and inclined (Figure 1*j*). The parallel growths have also been noted by other workers[1,2,8,9]. Poldervaart and Eckelmann[3] named such zircons as 'aggregate crystals'.

The presence of different type of morphological features, like inclusions, cracks, concentric zones, etc. in both the portions of composite zircons indicates that initially both the crystals had grown separately, and subsequently they joined together resulting in parallel growths. Jocelyn and Pidgeon[1] stated that the rarity of