

Heated red blood cells shed vesicles to indicate distress

When a human or animal are exposed to elevated temperature, the body temperature rises. A small increase in the body temperature may result in health problems that include a headache, dizziness, or fainting.

Red blood cells are very sensitive to heat. In 1865, Max Schultze used a light microscope to see changes of blood cells at increased temperatures. He examined a tiny droplet of live blood received from humans by fingerpick. At the temperature of 37°C, most of the red blood cells are oval biconcave disks of about 7 micrometers in diameter. At elevated temperatures, the red blood cells change from the biconcave discs to the spheres with small spicules. The transformed red blood cell were called echinocyte. He observed that many red blood cells shed vesicles. More than 100 years later, these observations were confirmed by electron microscopy, and recently by a high-resolution light microscope (Fig. 1).

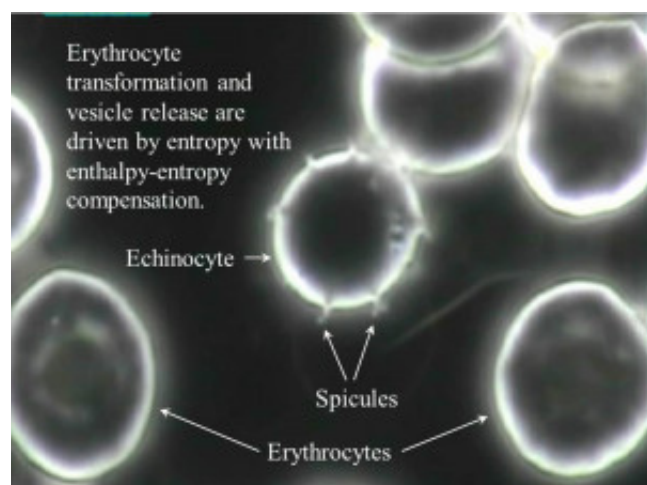


Fig. 1. Red blood cell-echinocyte transformation after exposure to heat. Vesicles are ejected from spicules.

The process of shedding vesicles is termed “vesiculation”. To analyze the relationship between temperature, rate of vesiculation, and state of the cells, we use thermodynamics. The thermodynamics describes heat and temperature and their relation to the properties of the system. In 1873 Willard Gibbs, introduced the equation relating energy at a stable temperature and pressure:

$$\Delta G = \Delta H - T\Delta S, (1)$$

where ΔH is a total amount absorbed or release heat at constant pressure, called enthalpy, T is

absolute temperature, and ΔS is a change in entropy. The entropy is a measure of disorder. The energy equivalent of this disorder is the product of T and ΔS .

During the physical exercise or the exposure to heat, the body absorbs the heat (ΔH), and accompanied change in energy of disorder is measured as $T\Delta S$. Sometimes, the energy associated with the change of entropy, $T\Delta S$, correlates with the change in enthalpy, ΔH , keeping the Gibbs free energy, ΔG , small compared to both ΔH and $T\Delta S$. If ΔG is small Equation 1 becomes as follows:

$$\Delta H \approx T\Delta S \quad (2)$$

Thus, ΔH linearly depends on ΔS . This phenomenon is known as an enthalpy-entropy compensation.

The process of red blood cell transformation at elevated temperatures is accompanied by a large number of physical and biochemical changes within the cell and cell membrane. The change in the rate of vesiculation, the change in membrane composition and ion permeability, the formation of abnormal hemoglobin, and decrease of membrane resistance to a pressure is a limited list of changes.

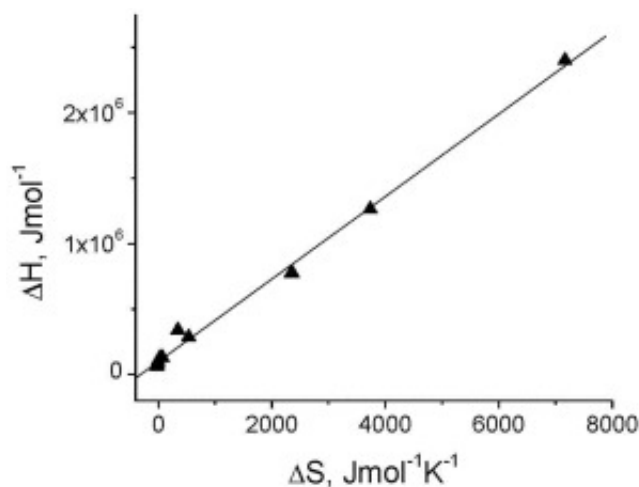


Fig. 2. Enthalpy - entropy correlation for red blood cell transformation.

An enthalpy change (ΔH) for vesiculation of human and rat erythrocytes was calculated together with ΔH related to other physical and biochemical changes. When ΔH values were plotted as a function of entropy change (ΔS), the data were fitted with a single line indicating the enthalpy-entropy compensation (Fig. 2). However, the suggestion that vesiculation, ion permeability, the formation of abnormal hemoglobin, and osmotic resistance of human and rats follow the same

mechanism is hard to accept.

Nevertheless, all processes include both water and cell membranes. Therefore, it is concluded that the ΔH compensation is based on the physical state of water molecules in the cell membrane. The small value of ΔG indicates that processes in the live cell are very economical. They keep the free energy at a minimum.

Because the decreased stability of red blood cells reduces their capability of oxygen transport, impairs endurance, and causes fatigue, the number of vesicles increases due to heat stress and exercise may be recognized for its diagnostic value of human health and performance.

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Publication

[Thermodynamic evaluation of vesicles shed by erythrocytes at elevated temperatures.](#)

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