

# Smart Cell Signal™ and Essential Hypertension

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People with raised blood pressure are at a greater risk of heart failure, kidney failure, stroke, dementia and loss of vision. Essential hypertension remains the biggest cause of death and infirmity in developed societies.

It is known from the Lewington data [1] that for about every 2 mm Hg of blood pressure reduction, there is about a 7 per cent reduction in coronary heart disease and about a 10 per cent reduction in stroke.

Nevertheless current treatments to reduce the adverse effects of high blood pressure do not remove the most common cause of pressure elevation in the circulation, essential hypertension.

It is known that essential hypertension is directly associated with the percentage of loss of skin capillaries [2-6] and that the loss is related to age [7]. Human skin holds more blood than any other organ and has around thirty capillary loops per square millimetre.

Rarefaction of these capillaries is known to commence in the third decade of life and by age 70 more than 40 per cent of the capillaries may be permanently lost. It has been shown that women at age 80 may have no capillary loops at all in the forearm skin [7].

In human capillaries, hydrodynamic pressures generated by plasma flow around red blood cells squeeze the cells, as narrow elongated shapes, one at a time into the capillaries.

Blood capillaries have no compliance and, given a constant red blood cell loading, any loss of the number of capillaries will inevitably cause a permanent pressure rise in the arteries.

It follows that where there is rarefaction of capillaries there will be a loss of arterial elasticity as a result of the elevated pressure.

Such a structural and functional change in arterial blood vessels has been described at the earliest stages in hypertension and acts not only as a marker for essential hypertension but also a risk factor for accelerated disease development. Studies demonstrate that impairment in the mechanical properties of large arteries represents an independent risk factor for future cardiovascular events.

Humans have about 3 to 4 million eccrine sweat glands spread throughout the skin. The glands supply continuous normal insensible perspiration via ducts to the skin surface.

Insensible perspiration is an aqueous liquid, taken from circulating serum, containing electrolytes, principally sodium, waste products and antimicrobial peptides [8-15]. As this liquid passes out through the sweat ducts, the transmembrane regulators in the duct wall make an adjustment to the electrolytes proportionate to the speed of passage through the duct.

The concentration of sodium in delivered perspiration is typically 25 per cent less than that in serum. The antimicrobial peptides, acting together with the balance of electrolytes [16], reduce the microbial load on the skin and prevent entry of microbes into the sweat ducts.

Human sweat glands and ducts have the ability to instantly switch over from insensible perspiration to the output of copious sweat in response to heat or exercise. Cooling copious sweat passes through the sweat ducts at seven times the rate of insensible perspiration. At this output, in a well-conditioned person, the transmembrane regulator of the duct removes all of the electrolytes. This effect is well known as the observation by athletes that there is no salt taste in their sweat when they are fully fit.

Copious cooling sweat does not have the balance of electrolytes that are present in perspiration. When the sweat glands are switched to copious cooling output there is no protection against the entry of normal skin microbes into the sweat ducts. This does not matter, since in copious cooling sweating the force of output of the copious sweat is sufficient to keep the ducts clear.

Populations of many societies are not fully fit, but typically have long periods of time with little physical activity, interspersed with short periods of exertion and also stress. This pattern results in the sweat glands starting to switch from insensible perspiration output to copious sweat output, but not fully completing the switch. The sweat ducts become habituated to a slightly increased output of perspiration, which is, as a result, deficient in electrolytes [17].

Without the proper levels of electrolytes the antimicrobial peptides are ineffective [16], allowing normal skin microbes to enter the sweat duct. The immune reaction to the entry of microbes into the duct blocks the duct at a point near to the sweat gland itself. Perspiration output under pressure from the sweat gland now ruptures the duct and redundant perspiration spreads into the surrounding skin, destroying any blood capillaries in its path. This is the cause of the rarefaction of capillaries, cumulatively increasing with age.

For ease of understanding their function, the eccrine sweat ducts may be described as Habituating and Velocity Associated Reabsorptive Ducts (HAVARDS).

Over the last few thousands of years, the HAVARDS of humans in the temperate zones were conditioned to proper habituation by sweating induced by many hours of physical work each day. This physical work is no longer common and for many people HAVARDS can only be conditioned by high ambient temperature. Essential hypertension is currently likely to be more prevalent in areas of increasing geographical latitude, and more prevalent in winter than in summer [18].

It is anticipated that people living in the temperate zones will never be persuaded to undertake regular daily sweat-inducing exercise. Instead there has to be medical intervention to restore the proper habituation of HAVARDS.

It is known that blood capillaries can regenerate, a process known as angiogenesis [19]. Preventing the destruction of capillaries by redundant perspiration from ruptured ducts should allow for angiogenesis of capillaries.

If a surplus of electrolytes could be created in the body then there would be no need for the protective conservation of electrolytes by the sweat ducts. If this surplus could be maintained then the sweat ducts would remain open, protected by antimicrobial peptides.

Adding electrolytes to body water, e.g. to the water in the digestive tract, results in surplus electrolytes being immediately dispersed and eliminated via the kidneys. For success, it would be necessary to create an apparent surplus that could not be eliminated.

An ion eXtra™ capsule can achieve this apparent surplus. The capsule seals small amounts of electrolyte, (e.g. sodium, potassium, magnesium and chloride) within a coating. The coating is permeable to gas but not to liquid. The solid electrolytes within the capsule change the immediate environment of the epithelial cells, rearranging the interfacial water molecules so that they appear to be ordered in the same manner as if the electrolytes were dispersed in water.

This re-ordering of water molecules is sufficient to open the ion channel gating [20], although no ions can pass through the channel as they are held to the capsule. However opening ion channels initiates cell-to-cell signalling throughout the body [21], with the body metabolism adjusting to an apparent surplus of electrolyte. This is the Smart Cell Signal™ effect.

In summary, a surplus would normally activate ion transport to remove the surplus, but the electrolytes cannot dissolve because of the coating, and so the electrolytes cannot be removed. The apparent surplus continues whilst the capsule brushes past the epithelial cells achieving the objective of creating an apparent constant surplus of electrolyte. The consequent receipt of signals indicating a surplus thus prevents the inappropriate conservation of electrolyte by the sweat duct transmembrane regulator.

The electrolytes sealed in the capsule pass through the body unchanged by being used, providing the desired effect for 24 hours or a little longer.

Full angiogenesis of skin capillaries by ion eXtra™ over 14 to 30 days allows for a lasting reduction in blood pressure to the level of a typical twenty-one year old, in the absence of significant atheroma or major organ disease.

It will be seen from the above description that research into the cause of essential hypertension cannot be carried out on laboratory animals, since their physiology is different from humans in that they do not have HAVARDS. There is one other mammal that does have HAVARDS, producing copious sweat for thermoregulation, and that is the horse. The horse is also the only other mammal to suffer from primary hypertension.

The Equiwinner™ dermal patch for horses uses ActiveSignal™ Smart Cell Signal™ technology to reverse essential hypertension in horses, and thus end exercise-induced pulmonary haemorrhage (EIPH). EIPH is as prevalent in horses as primary hypertension is in humans. Equiwinner™ has been on sale worldwide since June 2004, thousands horses successfully treated.

ActiveSignal™ products have comprehensive patents or patents pending in 55 countries, covering all aspects of the products.

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