REVIEW ARTICLE

# Is There a Role for Diaphoresis Therapy for Advanced Chronic Kidney Disease Patients?

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Diaphoresis therapy to remove water and solutes for the treatment of advanced chronic kidney disease (CKD) and chronic dialysis patients is an inadequately characterized treatment that was first reported over 50 years ago. Intensive diaphoresis, induced by heat treatment with saunas (dry heat) or hot baths (wet heat), can substantially increase cutaneous losses of water, urea, sodium, potassium, chloride, lactate, and possibly other solutes. How effectively diaphoresis therapy might remove many uremic toxins is not known. Diaphoresis therapy is not sufficiently effective to replace dialysis treatments, but theoretically it might be used to delay the start of chronic dialysis, supplement infrequent dialysis therapy, or augment chronic dialysis treatment perhaps especially for dialysis patients with excessive salt and water intake. Diaphoresis might be helpful for managing edema resistant states. Because it is inexpensive, diaphoresis may be particularly valuable in lower income countries where some patients may need to pay for dialysis. Diaphoresis might enhance some aspects of dietary treatment. The short-term and long-term effectiveness, safety, and patient acceptance of diaphoretic therapy need to be more carefully investigated.
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## Introduction

IT HAS BEEN known for many years that people lose solutes as well as water in sweat. Studies examining losses during diaphoresis (sweating) in both healthy and sick people, including those with kidney or heart failure, indicate that diaphoresis losses of water and certain solutes can be substantial.[1–3] When chronic dialysis therapy first became available, the costs for dialysis treatment and the gap between the numbers of patients in need of dialysis and the availability of this treatment led researchers to search for alternative therapies that might mitigate a patient's need for dialysis treatment. Hence, studies were conducted to examine whether diaphoresis could substitute for or be used to augment the effectiveness of hemodialysis treatment.[1,2] The use of diaphoresis to treat kidney failure did not become widespread, partly because hemodialysis and peritoneal dialysis treatments became more efficient and readily available in the developed countries and because some of the limitations of diaphoresis treatment became more evident. Diaphoresis was shown to not be sufficiently effective to completely substitute for chronic hemodialysis or peritoneal dialysis treatment.

Several developments have led to renewed interest in diaphoresis therapy. The emergence of chronic dialysis therapy in developing countries has led to increased interest in and demand for this dialysis treatment.[4] It is currently estimated that more than 2 million people in need of renal replacement therapy do not have access to standard dialysis therapy, and this is particularly common in low-income countries.[4] It is likely that this number may stay the same or increase in the foreseeable future. End-stage kidney disease (ESKD) patients in low-income countries frequently must pay all or part of their dialysis treatment costs. These financial demands as well as the current interest in infrequent dialysis treatment for new-onset ESKD patients[5] and the not uncommon need to start dialysis therapy urgently have led the authors to question whether diaphoresis therapy might delay or reduce the need for chronic dialysis in selected ESKD patients. This review addresses these issues. We will start with a brief review of sweat gland structure and function. More extensive reviews of sweat gland development, structure, and function have been published recently.[6,7]

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## Morphologic and Physiological Characteristics of Sweat Glands

There are 2 main types of sweat glands: eccrine glands and apocrine glands.[6,8] Eccrine glands in humans are found in the skin almost throughout the body except perhaps for few or none being present on the lips, auditory canal, and glans penis.[6,7,9] There are about 1.6 to 5 million eccrine glands in humans.[7] The number of eccrine sweat glands is roughly proportional to the body surface area. Eccrine glands are efficiently regulated to maintain the body temperature using the heat of water evaporation. Apocrine glands appear to be phylogenetic remnants of scent glands and are located almost exclusively in axillae and external genitalia.[10] Apocrine glands do not have thermoregulatory properties. A third type of gland, called an apoeccrine gland, has been described that appears to have mixed characteristics of both eccrine and apocrine glands.[6-8] In this review, we will focus on the properties of eccrine glands.

The human eccrine gland is a tubular structure about 3 to 5 mm in length. There is a coiled secretory portion of the duct (secretory coil) that connects to a rather straight reabsorptive duct (sweat duct) that empties to the outside of the skin. The secretory coil contains 3 types of cells, clear cells, dark cells, and myoepithelial cells. Stem cells may also be present. Myoepithelial cells have a mechanical or structural function. Dark cells contain comparatively few mitochondria and membrane villi. Clear cells contain many mitochondria and membranous villi and are thought to perform the major secretory activity of the eccrine gland. It has been suggested that clear cells are mainly responsible for secreting water, electrolytes, and inorganic compounds, whereas dark cells contribute to the secretion of larger organic molecules such as glycoproteins.[8] The sweat duct contains 2 layers of cells. The sweat duct lumen is lined by a suprabasal (luminal) cell layer with a basal cell layer situated above these cells.[6]

The secretory coil secretes sodium, potassium, and chloride, other macrominerals, many organic compounds and small amounts of trace elements, vitamins, and other solutes (Tables 1 and 2). Water excretion by the secretory coil is facilitated by aquaporin-5. The sweat duct partially reabsorbs a number of minerals secreted by the secretory coil. Ion channels, pumps, and co-transporters are present in clear cells and in dark cells.[6] In the secretory coil, these include NKCC1 ($Na^+$-$K^+$-$Cl^-$ co-transporter 1), $Na^+$-$K^+$-ATPase ($Na^+$-$K^+$ pump), NHE1 ($Na^+/H^+$ exchanger), CFTR (cystic fibrosis transmembrane conductance regulator), acquaporin-5. In the sweat duct, these include ENaC (epithelial sodium channel), $Na^+$-$K^+$-ATPase. NHE1, CFTR, and NKCC1.[6] Surprisingly, we were unable to find published papers of the microscopic or ultrastructural anatomy of sweat glands of adult CKD or ESKD patients, except for 1 report published in the era before dialysis therapy. This study described some smaller (atrophic) sweat glands in these patients.[23] Because many of the patients in this latter study died with untreated uremia, these pathologic findings may not be relevant for today's CKD and ESKD patients.

**Table 1.** Typical Composition of Sweat in Healthy People and Maintenance Hemodialysis Patients

| Clinical Conditions | Healthy Normals | | | MHD Patients | |
|---|---|---|---|---|---|
| Inducer | Heat | Exercise + Heat | Pilocarpine | Heat | Pilocarpine |
| Major cations | | | | | |
| Na (mmol/L) | 25.4 ± 18.0[11] | 42 ± 21[12] | 46.1 ± 24.5[13] | 38.4[1,*] | 34.2 ± 17.1[13] |
| K (mmol/L) | 4.35 ± 0.4[11] | 10.8 ± 2.6[12] | 11.5 ± 4.7[13] | 24.2[1,*] | 14.9 ± 6.6[13] |
| Ca (mmol/L) | 0.73 ± 0.11[11] | 1.01 ± 0.63[12] | 0.45 ± 0.08[13] | – | 0.90 ± 0.39[13] |
| Mg (mmol/L) | 0.13 ± 0.03[11] | 0.21 ± 0.16[12] | 0.10 ± 0.09[13] | – | 0.31 ± 0.11[13] |
| Major inorganic anions | | | | | |
| Cl (mmol/L) | 11.1 ± 2.3[11] | – | 45.6 ± 24.5[13] | 28.2[1] | 33.9 ± 17.2[13] |
| Phosphate (μmol/L) | – | – | 57 ± 33[13] | – | 80 ± 30[13] |
| Sulfate (μmol/L) | – | – | 105 ± 6[14] | – | 404 ± 43 [14,†] |
| Organic compounds | | | | | |
| Urea (mmol/L) | – | 22.2 ± 8.0[15] | 13.9 ± 6.0[13] | 11.7 ± 10.0[16] | 66.0 ± 31.1[13] |
| Creatinine (mmol/L) | | 0.031 ± 0.017[15] | | 0.105 ± 0.088[16] | |
| Lactate (mmol/L) | 6.84 ± 0.90[11] | – | – | – | – |
| Uric acid (mmol/L) | | 0.025 ± 0.007[15] | | 0.038 ± 0.014[16] | |
| Total amino acids (mmol/L) | – | 7.79 ± 1.85 [17,†] | – | – | – |
| Vitamins | | | | | |
| Vitamin B1 (thiamin, mg/dL) | – | 0.130 ± 0.008[18] | – | – | – |
| Vitamin C (mg/dL) | – | 0.02 ± 0.01[18] | – | – | – |
| Trace elements | | | | | |
| Zinc (μmol/L) | – | 7.54 ± 6.47[12] | – | – | – |
| Copper (μmol/L) | – | 4.21 ± 2.52[12] | – | – | – |

MHD, maintenance hemodialysis.
All data are expressed as mean and standard deviation except for † citations 14 and 17 which give the variance as standard error.
*Mean values retrieved from a figure in the article.

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