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# High-Resolution Model of Human Skin Appendages for Electromagnetic Dosimetry at Millimeter Waves 

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(Regular Paper)
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#### Abstract

The purpose of this work is to evaluate local power and electric field distributions inside high-resolution 3D anatomical CAD models of cutaneous appendages at 60 GHz upcoming for $5 \mathrm{G} / 6 \mathrm{G}$. The microscale resolution models of cutaneous nerves, lymphatics, pilosebaceous unit, microvasculature, eccrine sweat glands, Meissner corpuscle and Pacinian corpuscle were designed based on morphometric data and microscopic images. The Maxwell's and Hanai's mixture equations were employed to retrieve the complex permittivity of cutaneous appendages based on their free water content. The cutaneous appendages were exposed to a uniform plane wave. The finite element method was used to compute the power loss density $(\boldsymbol{P L D})$ and internal electric field $(\boldsymbol{E})$. The results indicate that the maximal $\boldsymbol{P} \boldsymbol{L} \boldsymbol{D}$ in cutaneous appendages occurred for $\boldsymbol{E}$ polarized along their longest dimension. Higher peak $\boldsymbol{P L D}$ was observed in high water content cutaneous appendages such as acrosyringium (45\%), epidermal axon (37.9\%), Pacinian corpuscle ( $32.5 \%$ ), blood capillary ( $30.6 \%$ ), lymph capillary ( $20 \%$ ) and arrector pili muscle ( $13.5 \%$ ) compared to the surrounding skin. Higher $\boldsymbol{E}$ was detected in low water content hair with the peak $\boldsymbol{E} 45.1 \%$ greater than in surrounding skin. The interfaces of the cutaneous appendages also manifested a boost (23.8\%-60.9\%) in $\boldsymbol{P L} \boldsymbol{D}$ compared to the surrounding skin. Detailed physical interpretation of observed phenomena is provided in the article. These results provide an insight into microscale power deposition in skin at frequencies upcoming for wireless communications.


INDEX TERMS Dosimetry, finite element method, human skin, millimeter waves.

## I. INTRODUCTION

In recent years, the prevalence of smartphones along with their data-intensive applications have spurred a phenomenal growth of mobile data traffic. According to the International Telecommunication Union (ITU), the exponential surge of mobile data traffic will reach 5 zettabyte/month by 2030 [1] whilst the number of devices connected to internet worldwide will
exceed 75.4 billion by 2025 [2]. Evidently, the conventional microwave spectrum cannot serve this booming demand for higher data rates. Thus, the wireless industry is propelled towards underutilized millimeter wave (mmWave) bands. Since the launch of fifth generation (5G) mobile communications in the first quarter of 2020, mmWave spectra have been deployed to provide ultra-low latency ( $\approx 1 \mathrm{~ms}$ ) and ultra-high data rates
$\underset{\text { MHz to THz Community }}{\text { MTT- }}$
(several GB/s). The small cell infrastructure in the 5 G era and beyond also targets to provide seamless coverage, ubiquitous connections and spectral efficiency. In particular, unlicensed allocations have been made in V-band for the development of 5G [3] and next generation Wi-Fi standard (e.g., WiGig) [4]. The mmWave spectrum is also critical for highly anticipated delay sensitive and high-speed communications such as virtual and augmented reality, IoT, autonomous vehicles, smart wearables, real-time telemedicine, remote surgery, and smart factory [2], [5], [6].

The systematic and continuous push towards the emerging 5G/6G mobile communications will make human exposure to mmWave radiation ubiquitous worldwide. As a consequence, development of tissue models accounting for local power deposition in cutaneous layers has become paramount for accurate dosimetry at these frequencies.

The anthropomorphic computational models (Virtual Family, Visible Human, etc.) have yielded a wealth of scientific knowledge on microwave dosimetry [7], [8]. However, the millimeter resolution of the voxel-based models is not enough to resolve skin substructures [9]. Early investigators resorted to theoretical calculations to evaluate power absorption within homogeneous [10] and planar stratified semi-infinite models [11] of human skin. For body-centric antenna and propagation measurements generic homogeneous skin-equivalent semi-solid [12] and solid [13] models were also introduced.

Human skin is neither homogeneous nor isotropic, rather it is a highly complex heterogeneous structure. Alekseev and Ziskin pioneered the numerical investigation of selective absorption of mmWave energy by cutaneous blood vessels, hair and sweat ducts as the complex permittivity of such skin appendages differ appreciably from the mean value for human skin [14], [15]. Their finite-difference-time-domain (FDTD) analysis at 42.25 GHz revealed that the specific absorption rate (SAR) inside the cutaneous blood vessels is substantially higher $(\approx 40 \%)$ than that of dermis and is a function of orientation of the incident electromagnetic (EM) field. Two-dimensional (2D) models of sweat duct and hair are considered in their work. Horizontal plexus of cutaneous blood vessels is modeled in three dimensions (3D) without taking into account the associated capillary loops. Another step towards understanding the interaction of mmWave with human skin was spearheaded by Feldman et al., who promulgated the hypothesis that helical portion of the sweat duct in epidermis might act as an array of low $\boldsymbol{Q}$ helical antennas between 200 and 450 GHz [16]-[18]. Although significant progress has been made in the field of skin dosimetry at mmWaves, understanding of the power deposition in cutaneous layers at micro-scale level requires additional investigation.

In this paper, we report high-resolution 3D models of a complete set of human skin sub-structures (blood vessels, nerve fibers, lymphatic vessels, eccrine sweat glands, sebaceous glands, hair, arrector pili muscle, Pacinian corpuscle, and Meissner corpuscle) developed based on anatomical data. These models are used for the first-time for local micro-scale resolution dosimetry at 60 GHz to quantitatively analyze the
electric field $(\boldsymbol{E})$ and power loss density ( $P L D$ ) distributions in the skin sub-structures. Different polarizations are considered to resolve finer details and analyze hot-spot formation.

## II. MATERIALS AND METHODS <br> A. GEOMETRIC MODEL OF INTEGUMENTARY SYSTEM

Skin model consisted of three distinct layers: stratum corneum, epidermis and dermis with a typical thickness of $11 \mu \mathrm{~m}, 120 \mu \mathrm{~m}$ and 1.1 mm , respectively. The thickness of stratum corneum, epidermis and dermis in general vary between $8.08-13.66 \mu \mathrm{~m}, 31.2-189.2 \mu \mathrm{~m}$ and $0.46-1.9 \mathrm{~mm}$, respectively [19], [20]. A 3D sinusoidal surface (1) was used to model the height ( $h$ ) of the junction between epidermis and dermis [Fig. 1(h)]:

$$
\begin{equation*}
h= \pm A \cos \left(\frac{2 \pi x}{S}\right) \cos \left(\frac{2 \pi y}{S}\right) \tag{1}
\end{equation*}
$$

where, $\mathrm{S}=0.21 \mathrm{~mm}$ and $\mathrm{A}=30 \mu \mathrm{~m}$ are span and amplitude, respectively. The distribution density of dermal papillae was $\approx 40$ per $1 \mathrm{~mm}^{2}$ [21].

## 1) CUTANEOUS INNERVATION

The skin contains a dense network of nerve fibers as shown in Fig 1(a) [22]. The horizontally arranged small nerve bundles in the subepidermal neural plexus were represented by a cylinder with a typical diameter and length of $10 \mu \mathrm{~m}$ [23] and 0.2 mm , respectively [Fig. 1(a)]. The length of the superficial neural plexus in general varies between 42-100 mm per $\mathrm{mm}^{2}$ [24]. The plexus was positioned at a typical depth of 0.2 mm from the skin surface. However, it can be as close as 0.1 mm from the surface of the skin [25]. Three C-fibers with a typical diameter of $1.5 \mu \mathrm{~m}$ and one $A \delta$ fiber with a diameter of $3 \mu \mathrm{~m}$ arborized from plexus at a distance of $25 \mu \mathrm{~m}$ [26] from dermo-epidermal junction and meandered towards vital layers of epidermis [Fig. 1(a)]. The variation of diameter of $A \delta$ fibers is generally between $1-6 \mu \mathrm{~m}$ whereas the diameter of C -fibers can be as low as $0.2 \mu \mathrm{~m}$ [27]. Moreover, the spatial density of epidermal nerve fibers in general varies between 0.5-8.5 per $0.01 \mathrm{~mm}^{2}$ [24], [28]. The depth of intraepidermal nerve fibers from stratum corneum was selected to be $0.4 \mu \mathrm{~m}$ [29] and $4.75 \mu \mathrm{~m}$, respectively.

## 2) CUTANEOUS LYMPHATICS

The cutaneous lymphatic network originates in the dermal papillae as blind ended tubes which drain into two horizontally running channels [Fig. 1(b)] [30]. The diameter of the lumen of lymphatic capillary was $25 \mu \mathrm{~m}$. However, it can be as low as $15 \mu \mathrm{~m}$ [31]. The diameter of vessels in superficial and deep plexus was $50 \mu \mathrm{~m}$ and $100 \mu \mathrm{~m}$, respectively. The variation in diameter of superficial and deep plexus is in general between $50-100 \mu \mathrm{~m}$ [32]. The lymphatic capillary was located at a depth of $50 \mu \mathrm{~m}$ from dermo-epidermal junction [33]. However, it can be as close as $25 \mu \mathrm{~m}$ from the junction [33]. The superficial plexus was located at a depth of $280 \mu \mathrm{~m}$ from the dermo-epidermal junction whereas deep lymphatic plexus


FIGURE 1. Geometric model: (a) cutaneous nerves microscopic image [22] and 3D model; (b) cutaneous lymphatic microscopic image [30] and 3D model; (c) pilosebaceous unit microscopic image [36] and 3D model; (d) cutaneous microvasculature microscopic image [48] and 3D model; (e) eccrine sweat gland microscopic image [36] and 3D model; (f) Meissner corpuscle microscopic image [57] and 3D model; (g) Pacinian corpuscle microscopic image [59] and 3D model; (h) Stratum corneum and epidermis 3D model. Reprinted with the permissions from Elsevier.
was located at a depth of approximately $900 \mu \mathrm{~m}$ from the skin surface. The depth of the superficial plexus from the dermoepidermal junction generally varies between $50-300 \mu \mathrm{~m}$ [34]. Moreover, the vertical length of the lymphatic capillary was 0.245 mm whereas the horizontal length of lymphatic vessels was 0.2 mm [30]. Lastly, the mean lymphatic vessel density in papillary and reticular dermis was $18.1 \pm 5.3$ per $\mathrm{mm}^{2}$ and $8.9 \pm 1.2$ per $\mathrm{mm}^{2}$, respectively [35].

## 3) PILOSEBACEOUS UNITS

The pilosebaceous unit comprised of hair follicle, hair, sebaceous gland and arrector pili muscle as displayed in Fig. 1(c) [36]. The vellus hair follicle was represented by a cone with a typical top diameter of $86 \mu \mathrm{~m}$ and a length of 0.64 mm [37]. The density of hair follicles at different body sites varies between 0.14 and 2.92 per $\mathrm{mm}^{2}$ [38]. The portion of hair anchored in hair follicle was roughly 0.6 mm
long while the portion of the hair emerging out of skin was about 0.3 mm in length. The top and bottom diameter of hair cone were approximately $36 \mu \mathrm{~m}$ and $54 \mu \mathrm{~m}$. The variation of diameter of the hair is generally between 16-80 $\mu \mathrm{m}$ [39], [40]. The hair protruded from the skin at an angle of $30^{\circ}$ [41].

The arrector pili muscle associated with hair follicle was modeled by a meandered cylinder with a typical diameter of $60 \mu \mathrm{~m}$ that spanned between epidermis and hair follicle. The diameter of arrector pili muscle generally varies between 10-140 $\mu \mathrm{m}$ [42]. It was connected to hair follicle at a depth of roughly 0.4 mm and had a total length of about 0.54 mm .

The sebaceous glands were saccular and ovoid with a typical diameter and length of $120 \mu \mathrm{~m}$ and $125 \mu \mathrm{~m}$, respectively [43], [44]. The sebaceous glands were linked to the lumen of hair follicle via a small duct at a depth of roughly 0.16 mm [45]. In most parts of human skin, the density of sebaceous glands is less than 1 per $\mathrm{mm}^{2}$ [44]. Lastly, microscopy data issued from [46], [47] were used to model the capillaries of hair follicle and sebaceous glands as shown in Fig. 1(c).

## 4) CUTANEOUS MICROVASCULATURE

The cutaneous microvasculature was organized into two main interconnected networks of blood vessels as shown in Fig. 1(d) [48]. The lumen of arteries and veins of the superficial plexus were modeled with cylinders with a typical diameter of $10 \mu \mathrm{~m}$ and $12.5 \mu \mathrm{~m}$ and were located at a typical depth of 0.35 mm from the skin surface. The diameter of the lumen of arteries and veins in the superficial plexus generally varies between $7.5-12 \mu \mathrm{~m}$ and $10-15 \mu \mathrm{~m}$ [49]. The range of variation of the depth of superficial plexus from the skin surface is $0.2-0.4 \mathrm{~mm}$ [14], [50]. The length of the vessels was fixed at 0.2 mm .

The arterial limb, venous limb and the intrapapillary loop that constituted the endothelial tube of the capillary had a typical diameter of $6.25 \mu \mathrm{~m}, 8 \mu \mathrm{~m}$ and $4.25 \mu \mathrm{~m}$, respectively. The diameter of the endothelial tube of the arterial limb, venous limb and intrapapillary loop in general varies between 5$7.5 \mu \mathrm{~m}, 6-10 \mu \mathrm{~m}$ and $3.5-6 \mu \mathrm{~m}$, respectively [49]. The length and width of the capillary loop was about $250 \mu \mathrm{~m}$ and $45 \mu \mathrm{~m}$. Generally, the length and width of the capillary loop varies between $23.3-270 \mu \mathrm{~m}$ and $32-46.1 \mu \mathrm{~m}$ [51]. The number of capillary loops varies from $20-150$ per $\mathrm{mm}^{2}$, which in average corresponded to one capillary loop per $0.04 \mathrm{~mm}^{2}$ [52].

The lumens of arteries and veins of the deep dermal plexus were also modeled with cylinders with a typical diameter of $25 \mu \mathrm{~m}$ and $30 \mu \mathrm{~m}$. The outside diameter of vessels in deep dermal plexus in general varies between $40-50 \mu \mathrm{~m}$ [48]. The deep dermal plexus was positioned at a depth of 1 mm from the skin surface [53]. However, the deep plexus can be as deep as 1.5 mm from the skin surface [53]. Two vertical tributaries connected the upper plexus to lower one. The depth dependent distribution of cutaneous blood vessels was used to model arterioles and venules ( 2 per $\mathrm{mm}^{2}$ ) [54].

## 5) ECCRINE SWEAT GLANDS

Eccrine sweat glands are widely distributed in skin (0.13-3.6 per $\mathrm{mm}^{2}$ ) [47], [55]. It was composed of an excretory duct and a secretory portion [36] (Fig. 1(e)). The inner typical diameter of the convoluted secretory duct embedded deep into dermis was set to $40 \mu \mathrm{~m}$. However, the diameter of the lumen of the secretory duct can be as low as $30 \mu \mathrm{~m}$ [56]. Moreover, the overall diameter of the secretory portion was roughly fixed at $600 \mu \mathrm{~m}$. The variation of overall diameter of the secretory portion is in general between $500-700 \mu \mathrm{~m}$. The total length and inner diameter of the straight excretory duct emerging from the secretory portion were roughly 1 mm and $20 \mu \mathrm{~m}$. The length of straight duct can be up to 2 mm whereas the diameter of its lumen can be as low as $10 \mu \mathrm{~m}$ [56]. The distal helical segment (acrosyringium) of the excretory duct consisted of three turns whereas the axial pitch, major and minor radius of the helix were $60 \mu \mathrm{~m}, 120 \mu \mathrm{~m}$ and $20 \mu \mathrm{~m}$, respectively. The minor radius of the helix can be up to $60 \mu \mathrm{~m}$ [56]. Lastly, in the absence of the quantitative data regarding blood supply, microscopy images [47] were used to sketch the capillaries (diameter $=6.5 \mu \mathrm{~m}$ ) [49] wrapped around the secretory portion. One of the capillaries from the secretory portion also spiraled along the straight duct.

## 6) MEISSNER CORPUSCLES

Meissner corpuscle was modeled by a tapered cylindrical structure with the typical dimensions of $50 \mu \mathrm{~m} \times 100 \mu \mathrm{~m}$. The width and length of Meissner corpuscle can vary between $30-75 \mu \mathrm{~m}$ and $50-150 \mu \mathrm{~m}$, respectively [57] [Fig. 1(f)]. The diameter of the spiraling nerve fiber associated with Meissner corpuscle was about $3 \mu \mathrm{~m}$. They were localized between epidermal ridges at a depth of roughly 0.1 mm from the skin surface and are numerous within skin (20-50 per $\mathrm{mm}^{2}$ ) [58].

## 7) PACINIAN CORPUSCLES

An ellipsoid with the typical dimensions of $1 \mathrm{~mm} \times 0.5 \mathrm{~mm} \times$ 0.5 mm was used to model Pacinian corpuscle [59] [Fig. 1(g)]. The dimensions of the Pacinian corpuscle can be as large as $2.5 \mathrm{~mm} \times 0.75 \mathrm{~mm}$ [60]. It was located roughly at a depth of 1 mm from the skin surface. The distribution density of the Pacinian corpuscles was reported to be $5-10 / \mathrm{cm}^{2}$ [61].

## B. ELECTROMAGNETIC PROPERTIES

The complex permittivity of stratum corneum [62], epidermis and dermis [62], blood [63], arrector pili muscle [63] and hair [15] at 60 GHz are summarized in Table 1. Owing to lack of experimental data on the electromagnetic properties of rest of the skin appendages, the mixture equations were employed to derive the complex permittivity based on their distinctive water content. The complex permittivity of pure water at skin temperature ( $33{ }^{\circ} \mathrm{C}$ ) [64] was evaluated by using Ellison model [65]. The permittivity ( $\approx 2.5$ ) of proteins [62] was used to model the volume fraction of dry components.
tABLE 1. Water Content and Complex Permittivity at $\mathbf{6 0} \mathbf{~ G H z}$

| Compartment | Water <br> Content | Bound <br> Water | Complex <br> Permittivity |
| :---: | :---: | :---: | :---: |
| Stratum corneum [62] | - | - | $3.5-0.51 \mathrm{j}$ |
| Epidermis and <br> Dermis [62] | - | - | $8.17-11.27 \mathrm{j}$ |
| Blood [63] | - | - | $12.3-16.4 \mathrm{j}$ |
| Arrector pili <br> muscle [63] | - | - | $12.9-15.8 \mathrm{j}$ |
| Hair [15] | - | - | $2.6-0.1 \mathrm{j}$ |
| Water [65] | - | - | $15.2-23.3 \mathrm{j}$ |
| Lymph [67] | $96.6 \%$ | $1.1 \%$ | $14.84-22.3 \mathrm{j}$ |
| Axoplasm [68] | $84.74 \%$ | $4.9 \%$ | $13.04-18.46 \mathrm{j}$ |
| Remak bundles [73] | $71.56 \%$ | $9.13 \%$ | $11.2-14.5 \mathrm{j}$ |
| Sweat [69] | $97.79 \%$ | $0.7 \%$ | $15.02-22.7 \mathrm{j}$ |
| Sebaceous glands [70] | $13.2 \%$ | $22.5 \%$ | $4.4-1.7 \mathrm{j}$ |
| Humid Hair [71] | $0.1 \%$ | $32.1 \%$ | $3.34-0.3 \mathrm{j}$ |
| Pacinian corpuscle [60] | $91.1 \%$ | $2.86 \%$ | $13.99-20.51 \mathrm{j}$ |
| Meissner corpuscle | $70.8 \%$ | $9.4 \%$ | $11.1-14.2 \mathrm{j}$ |
| lumen [73] |  |  |  |

## 1) WATER

The mass fraction of the biomolecules and water in various skin appendages was converted to the volume fraction by using a mass density of $1.35 \mathrm{~g} / \mathrm{cm}^{3}$ [66] and $1 \mathrm{~g} / \mathrm{cm}^{3}$, respectively (Table 1). The selected values for axoplasm (Squid giant axon) are close to the data reported for small axons (90$91 \%$ ) [72]. Moreover, since the major portion of Meissner corpuscle's lumen and Remak bundles is occupied by Schwann cells, the water content of Schwann cells ( $\approx 75 \%$ ) [73] was used as a first approximation.

## 2) BOUND WATER

In this work, $0.35 \mathrm{~g}(0.2-0.65 \mathrm{~g})$ of bound water per g of biomolecules was assumed to evaluate the volume fraction of water bound to polar groups and charged sites on the surface of biomolecules [74], [75] (Table 1).

## 3) MIXTURE MODEL

The mixture equations were applied in two steps. First, the Maxwell's mixture equation (2) [76] was used to enumerate the effective complex permittivity of hydrated biomolecules $\left(\varepsilon_{e}^{*}\right)$.

$$
\begin{equation*}
\frac{\varepsilon_{e}^{*}-\varepsilon_{b}^{*}}{\varepsilon_{e}^{*}+\varepsilon_{b}^{*}}=p \frac{\varepsilon_{p}^{*}-\varepsilon_{b}^{*}}{\varepsilon_{p}^{*}-2 \varepsilon_{b}^{*}} \tag{2}
\end{equation*}
$$

where $p$ is the volume fraction taken by the biomolecule, $\varepsilon_{p}^{*}$ and $\varepsilon_{b}^{*}$ are the complex permittivity of the biomolecule and bound water shell, respectively. Since, the Maxwell's mixture equation only gives best accuracy for dilute suspensions
( $p \approx 30 \%$ ) [74], in the second step Hanai's mixture equation (valid up to $\phi=0.8$ ) [77], [78] was utilized to extract the complex permittivity of the skin appendages $\left(\varepsilon^{*}\right)$ :

$$
\begin{equation*}
1-\phi=\frac{\varepsilon^{*}-\varepsilon_{e}^{*}}{\varepsilon_{w}^{*}-\varepsilon_{e}^{*}}\left(\frac{\varepsilon_{w}^{*}}{\varepsilon^{*}}\right)^{1 / 3} \tag{3}
\end{equation*}
$$

where $\phi$ is the volume fraction occupied by the hydrated biomolecules and $\varepsilon_{w}^{*}$ is the complex permittivity of the free water molecules.

## C. NUMERICAL METHOD

The full-wave electromagnetic simulations were performed using finite element method implemented in COMSOL Multiphysics (RF module). The source was a TE and TM plane wave $\left(10 \mathrm{~W} / \mathrm{m}^{2}\right.$ at 60 GHz$)$. The periodic boundary conditions (Floquet periodicity) were applied in the $x$ and $y$ directions to simulate an infinite structure. A free space padding of 0.25 mm was inserted between the periodic port and the skin model (in case of the hair follicle, a padding of 0.35 mm was added). The outgoing waves were absorbed by 8 -layered perfectly matched layer. The computational volume $(0.2 \mathrm{~mm}$ $\times 0.2 \mathrm{~mm} \times 1.1 \mathrm{~mm}$ ) in majority of simulations was discretized into roughly $3 \times 10^{6}-5 \times 10^{6}$ tetrahedral Lagrange quadratic mesh elements. The minimum and maximum mesh cell size was within $0.05-30 \mu \mathrm{~m}$ range. The numerical simulations were performed on a high-performance work station equipped with 36-core dual CPU of Intel Xeon(R) Gold 6140 (2.3-3.7 GHz) with 768 GB RAM. This numerical approach was validated on canonical analytical model in our previous study [79]. Lastly, the numerical simulations were performed on isolated skin appendages since simulating a complete skin model containing all skin appendages would require computational resources exceeding the capabilities of the highperformance workstations used in this study.

## D. DOSIMETRY METRICS

PLD was used to assess the local power loss per unit volume in the skin substructures $\left(P L D=\frac{1}{2} \sigma_{e}|E|^{2}\right.$ where $\sigma_{e}=$ $\left.\sigma_{s}+\omega \epsilon^{\prime \prime}\right)$. The maximum electric field ( $E_{\max }$ ) and maximum power loss density $\left(P L D_{\max }\right)$ represent the highest values of $E$ and $P L D$ in the skin appendages. For comparison, the $E_{\max }$ and $P L D_{\max }$ were then normalized with respect to $E$ and $P L D$ in the surrounding skin (defined as the uniform $E$ and $P L D$ in skin sufficiently far from the local distortions created by skin appendages and located at the same depth). Note that, although skin at microscale level is comprised of individual cells, recent microdosimetry study at the level of single skin cell model revealed that $P L D$ distribution within cellular compartments is nearly uniform at 60 GHz [84]. Lastly, average electric field $\left(E_{\text {avg }}\right)$ and average power loss density $\left(P L D_{\text {avg }}\right)$ were evaluated as $E$ and $P L D$ averaged over the volume of skin substructure.

## III. RESULTS

Table 2 summarizes the $E_{\max }$ and $P L D_{\max }$ in various skin appendages compared to surrounding skin and skin surface. In

TABLE 2. Local Dosimetry Metrics Within Skin Appendages at Typical Depths for Incident Power Density of $10 \mathbf{W} / \mathbf{m}^{\mathbf{2}}$

| Skin appendages | Compared to background skin |  | Compared to skin surface |  | Maximal values |  | Mean values |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | $\boldsymbol{E}_{\text {max }}$ | $P L D_{\text {max }}$ | $\boldsymbol{E}_{\text {max }}$ | $P L D_{\text {max }}$ | $\begin{aligned} & \boldsymbol{E}_{\max } \\ & (\mathrm{V} / \mathrm{m}) \end{aligned}$ | $\begin{aligned} & P L D_{\max } \\ & \left(\mathrm{kW} / \mathrm{m}^{3}\right) \end{aligned}$ | $\begin{gathered} E_{\text {avg }} \\ (\mathrm{V} / \mathrm{m}) \end{gathered}$ | $\begin{gathered} P L D_{\mathrm{avg}} \\ \left(\mathrm{~kW} / \mathrm{m}^{3}\right) \end{gathered}$ |
| Acrosyringium | $\searrow 5 \%$ | $\nearrow 45 \%$ | $\searrow 9 \%$ | $\nearrow 97.2 \%$ | 34.6 | 45.2 | 26.6 | 27.5 |
| Axoplasm | $\searrow 0.8 \%$ | $\nearrow 37.9 \%$ | $\searrow 2.8 \%$ | $\nearrow 97.1 \%$ | 37.1 | 42.5 | 27.4 | 23.7 |
| Pacinian corpuscle | $\searrow 9.66 \%$ | $\nearrow 32.5 \%$ | $\searrow 88.2 \%$ | $\searrow 44.1 \%$ | 4.53 | 0.69 | 2.6 | 0.25 |
| Blood capillary | $\searrow 0.5 \%$ | $\nearrow 30.6 \%$ | $\searrow 18.1 \%$ | $\nearrow 95.4 \%$ | 31.4 | 26.9 | 19.5 | 10.7 |
| Meissner corpuscle's axon | $\searrow 10.4 \%$ | $\nearrow 23.9 \%$ | $\searrow 31.2 \%$ | $\nearrow 94.2 \%$ | 26.2 | 21.2 | 23.1 | 16.5 |
| Lymphatic capillary | $\searrow 20.7 \%$ | $\nearrow 20 \%$ | $\searrow 39.2 \%$ | $\nearrow 93.8 \%$ | 23.3 | 20.2 | 15.8 | 9.5 |
| Arrector pili muscle | $\searrow 9.2 \%$ | $\nearrow 13.5 \%$ | $\searrow 24 \%$ | $\nearrow 94 \%$ | 29.1 | 22.3 | 17.5 | 8.6 |
| Sebaceous gland | $\nearrow 36.96 \%$ | $\searrow 61.8 \%$ | $\searrow 4.02 \%$ | $\nearrow 67.6 \%$ | 36.8 | 3.9 | 30.4 | 2.6 |
| Hair | $\nearrow 45.05 \%$ | $\searrow 97.1 \%$ | $\nearrow 41.6 \%$ | $\searrow 43.04 \%$ | 65.7 | 0.71 | 33 | 0.2 |

* $\searrow$ and $\nearrow$ denote decrease and increase, respectively, compared to background skin or skin surface.


FIGURE 2. Cutaneous nerves: (a), (c) $E$ (arrows in the figure only show the $E$ field direction and not the amplitude); (b), (d) PLD distribution.
addition, $E_{\text {avg }}$ and $P L D_{\text {avg }}$ within different skin appendages is also presented. Note that the data in Table 2 correspond to the polarization (TE or TM) resulting in $P L D_{\max }$ within the skin appendages.

## A. NERVES

Fig. 2 illustrates $E$ and $P L D$ distributions in cutaneous nerves. The maximum $P L D\left(42.5 \mathrm{~kW} / \mathrm{m}^{3}\right)$ in the horizontal segment of the epidermal axoplasm when incident $E$ was oriented parallel to it was roughly $37.9 \%$ higher than in the surrounding skin [Fig. 2(b)]. This local enhancement in $P L D$ was primarily due to the higher conductivity of axoplasm as the induced $E(37.1 \mathrm{~V} / \mathrm{m})$ inside the horizontal axoplasm approached the incident $E(37.4 \mathrm{~V} / \mathrm{m})$ at axoplasm/epidermal interface due to continuity of the tangential components of $E$ [Fig. 2(a)]. Contrarily, in case of orientation of $E$ normal to the lateral surface of the axoplasm, the maximum $P L D\left(24 \mathrm{~kW} / \mathrm{m}^{3}\right)$ inside the axoplasm was about $7.4 \%$ lower than in the surrounding skin [Fig. 2(d)]. This was in accordance with boundary conditions
on normal $E$ at axoplasm/skin interface, which required a reduction in the internal $E(27.9 \mathrm{~V} / \mathrm{m})$ relative to the incident $E(37.1 \mathrm{~V} / \mathrm{m})$ by a factor of roughly the relative permittivity of axoplasm [Fig. 2(c)]. It is worthwhile to mention that the maximum PLD inside the axoplasm was $97.1 \%$ higher than at the surface of stratum corneum for the former polarization. On the contrary, the peak $E(37.1 \mathrm{~V} / \mathrm{m})$ in the axoplasm was $2.8 \%$ lower than at the skin surface for the polarization shown in Fig. 2(a).

For $E$ perpendicular to the vertical section of the axoplasm, the maximal $P L D\left(40.05 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and $E(46.1 \mathrm{~V} / \mathrm{m})$ localized at axoplasm/skin interface was up to $35.1 \%$ and $37.1 \%$ higher than maximal $P L D$ and $E$ induced inside axoplasm for both polarizations [Fig. 2(a, b)]. This was the direct consequence of the Gauss's law ( $\varepsilon_{1} E_{1}^{\perp}-\varepsilon_{2} E_{2}^{\perp}=\rho_{s}$ ), which led to accumulation of surface charges at the dielectric discontinuities. Although the thicker $A \delta$ fiber produced greater distortions in $E$ and $P L D$ than thinner C-fiber, the average $E$ and $P L D$ in $A \delta$ and C-fiber differed by less than $1 \%$. This suggests that power loss inside various nerve fibers in skin is almost independent of their diameter. Moreover, for both polarizations, $E$ (29 $\Rightarrow 22 \mathrm{~V} / \mathrm{m})$ and the corresponding $P L D\left(27 \Rightarrow 15 \mathrm{~kW} / \mathrm{m}^{3}\right)$ decreased monotonically by $24.1 \%$ and $44.4 \%$ in the straight section of the central axoplasm (100 $\mu \mathrm{m}$ long). The $E$ and $P L D$ inside the skin model at the same depth was $24.1 \%$ and $6.25 \%$ higher than that of the axoplasm for both polarizations. The swifter decay of $E$ in axoplasm was due to its higher permittivity and conductivity compared to the background skin.

Lastly, the peak PLD ( $15.09 \mathrm{~kW} / \mathrm{m}^{3}$ ) in horizontal plexus was also $22.3 \%$ higher than in the surrounding skin for $E$ parallel to its longest dimension [Fig. 2(b)]. In contrast, for $E$ polarized normal to the lateral surface of the plexus, the peak $P L D\left(11.3 \mathrm{~kW} / \mathrm{m}^{3}\right)$ in the plexus was $6.9 \%$ lower than that in the surrounding skin [Fig. 2(d)]. The relatively lower increase in plexus in comparison to epidermal axons was mainly due to lower conductivity of Remak bundles. It is worthwhile to


FIGURE 3. Cutaneous lymphatic: (a), (c) $E$ distribution; (b), (d) PLD distribution.
mention that both $E$ and $P L D$ in the cross sections of nerves transverse to the propagation direction were uniform as the dimensions of nerves were an insignificant fraction of wavelength ( $0.003 \lambda$ ) in skin at 60 GHz .

## B. LYMPH

Fig. 3 displays $E$ and $P L D$ in the longitudinal cross section of cutaneous lymphatic vessels in papillary dermis. The maxima of the $P L D\left(20.2 \mathrm{~kW} / \mathrm{m}^{3}\right)$ in the terminal portion of the openended lymphatic capillary were $20 \%$ higher than undistorted $P L D$ in surrounding skin for the polarizations shown in Fig. 3 (b, d), respectively. The $E$ in the capillary ( $23.3 \mathrm{~V} / \mathrm{m}$ ) was $20.7 \%$ weaker than the surrounding skin as $E$ was normal to lateral surface of the capillary for both polarizations. Notice that the peak $P L D$ in the lymphatic capillary was $93.8 \%$ higher than at the skin surface whereas maximal $E$ was $39.2 \%$ lower.

Conversely, for $E$ polarized along the axis of the horizontal plexuses located in papillary and reticular dermis, $P L D$ in superficial $\left(9.75 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and deep plexus $\left(1.1 \mathrm{~kW} / \mathrm{m}^{3}\right)$ was $51.5 \%$ and $51.8 \%$ higher than in surrounding skin [Fig. 3(b)]. The corresponding $E$ in the superficial $(16.2 \mathrm{~V} / \mathrm{m})$ and deep plexus ( $5.5 \mathrm{~V} / \mathrm{m}$ ) was almost similar (difference $=2 \%$ ) to the surrounding skin due to the continuity of the tangential component of $E$. Moreover, for the polarization shown in Fig. 3(d), $P L D$ in superficial ( $4.36 \mathrm{~kW} / \mathrm{m}^{3}$ ) and deep dermal plexus $\left(0.54 \mathrm{~kW} / \mathrm{m}^{3}\right)$ was $11.5 \%$ and $13.5 \%$ lower than that in surrounding skin. This is due to $33.1 \%$ and $33.9 \%$ weak $E$ in superficial ( $10.8 \mathrm{~V} / \mathrm{m}$ ) and deep plexus ( $3.8 \mathrm{~V} / \mathrm{m}$ ) from surrounding skin as $E$ was normal to the vessels. Note that $E$ was $0.8 \%$ weaker in the thicker vessels. The aforementioned results suggest only a marginal effect of vessel diameter on induced $E$.


FIGURE 4. Pilosebaceous unit: (a), (c) E distribution; (b), (d) PLD distribution.

For polarizations in Fig. 3(b, d), the exponential decay of $P L D$ from the capillary $\left(20.2 \mathrm{~kW} / \mathrm{m}^{3}\right)$ to the distal end of lymphatic vessel ( $0.8 \mathrm{~kW} / \mathrm{m}^{3}, 0.6 \mathrm{~kW} / \mathrm{m}^{3}$ ) was $96 \%-97 \%$. The monotonous decay in corresponding $E$ from capillary $(23.3 \mathrm{~V} / \mathrm{m})$ to posterior end $(4.6 \mathrm{~V} / \mathrm{m}, 4.03 \mathrm{~V} / \mathrm{m})$ of the lymph vessel was $80.2 \%-82.7 \%$. Notice that the peak $E(40 \mathrm{~V} / \mathrm{m})$ and PLD $\left(31 \mathrm{~kW} / \mathrm{m}^{3}\right)$ at the interface of the terminal capillary with skin were $29 \%$ and $50 \%$ higher than in surrounding skin [Fig. 3(a, b)]. Lastly, since the diameter of lymphatic vessels was much smaller than the wavelength ( $\lambda / 20$ ), $E$ and $P L D$ inside the lymphatic vessels were almost uniform in the $E-H$ plane.

## C. PILOSEBACEOUS UNIT

The details of local $E$ and $P L D$ through a central plane of pilosebaceous unit are shown in Fig. 4. The analysis of Fig. 4(a, c) revealed that the peak $E$ was not located at the outer boundary of hair or skin. Rather, the site of maximal $E$ ( $62.9 \mathrm{~V} / \mathrm{m}-65.7 \mathrm{~V} / \mathrm{m}$ ) was inside the portion of hair that was embedded in epidermis. This can be attributed to low permittivity and conductivity of hair due to its lower water content. The maximum induced $E$ inside the hair ( $65.7 \mathrm{~V} / \mathrm{m}$ ) and sebaceous gland $(36.8 \mathrm{~V} / \mathrm{m})$ was $45.05 \%$ and $36.96 \%$ higher than surrounding skin for the orientation of $E$ perpendicular to the base of the sebaceous gland [Fig. 4(a)]. Contrarily, for the polarization of $E$ parallel to the base of the sebaceous gland, the peak $E$ in hair ( $62.9 \mathrm{~V} / \mathrm{m}$ ) and sebaceous gland ( $29.4 \mathrm{~V} / \mathrm{m}$ ) was $40.5 \%$ and $15.7 \%$ higher than in surrounding skin [Fig. 4(b)]. Note that for both polarizations the peak $E$ in hair was $39.2 \%-41.6 \%$ higher than at the skin surface whereas the peak $E$ in the sebaceous glands was $4.02 \%$ and $22.9 \%$ lower for the polarization shown in Fig. 4(a, c), respectively.

Despite the high levels of $E$, the corresponding $P L D$ in hair $\left(0.71 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and sebaceous gland ( $3.9 \mathrm{~kW} / \mathrm{m}^{3}$ ) was roughly $97.1 \%$ and $61.8 \%$ lower compared to the surrounding skin for the former polarization whereas for the latter one $P L D$ in hair $\left(0.65 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and sebaceous gland $\left(0.25 \mathrm{~kW} / \mathrm{m}^{3}\right)$ was $97.5 \%$ and $78.7 \%$ lower. It is worthwhile to mention that for polarizations in Fig. $4(\mathrm{~b}, \mathrm{~d})$, the peak $P L D\left(0.71 \mathrm{~kW} / \mathrm{m}^{3}, 0.61 \mathrm{~kW} / \mathrm{m}^{3}\right)$ in hair was $43.04 \%$ and $47.4 \%$ lower than the skin surface whereas it was $67.6 \%$ and $49.7 \%$ higher in the sebaceous gland ( $3.9 \mathrm{~kW} / \mathrm{m}^{3}, 2.5 \mathrm{~kW} / \mathrm{m}^{3}$ ).

Aside from the higher $E$ inside the hair, the local spikes in $E(57.2 \mathrm{~V} / \mathrm{m})$ and $P L D\left(61.53 \mathrm{~kW} / \mathrm{m}^{3}\right)$ around the hair for the polarization shown in Fig. 4(c, d) were roughly 37.5\% and $60.9 \%$ greater compared to that in surrounding skin. On the other hand, for the polarization shown in Fig. 4(a, b), the local $E(16.01 \mathrm{~V} / \mathrm{m})$ and $P L D\left(4.82 \mathrm{~kW} / \mathrm{m}^{3}\right)$ in the immediate vicinity of the hair was $53.7 \%$ and $78.5 \%$ smaller than in the surrounding skin. These abrupt local enhancements and reductions were because hair with an electrically small diameter acted as an electric dipole with surface charges induced at its boundaries depending on the polarization.

In contrast, the high-water content appendages, such as arrector pili muscle and capillaries associated with sebaceous gland, exhibited the peak $E(28.9 \mathrm{~V} / \mathrm{m}$ and $24.8 \mathrm{~V} / \mathrm{m})$, which was $11.2 \%$ and $10.2 \%$ lower than in surrounding skin for the polarization shown in Fig. 4(a). Conversely, the corresponding $P L D$ in muscle $\left(21.9 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and capillaries $\left(16.83 \mathrm{~kW} / \mathrm{m}^{3}\right)$ was $9.6 \%$ and $14.7 \%$ more intense than in surrounding skin [Fig. 4(b)]. Furthermore, for the orientation of $E$ displayed in Fig. 4(c), the maximal $E$ was $9.2 \%$ lower in arrector pili muscle ( $29.1 \mathrm{~V} / \mathrm{m}$ ) and $16.1 \%$ higher in capillaries of sebaceous gland ( $33.1 \mathrm{~V} / \mathrm{m}$ ) compared to that in surrounding skin. The $P L D$ associated with the muscle $\left(22.3 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and capillaries ( $30.03 \mathrm{~kW} / \mathrm{m}^{3}$ ) in this case was $13.5 \%$ and $51.6 \%$ higher than that in surrounding skin. The notable enhancement of power absorption inside the capillaries for the latter polarization is explicable by charge accumulation at the interface of sebaceous gland due to the discontinuity of normal electric displacement vector $D$, which results in discontinuity in $E$ equal to the permittivity of sebaceous material. Notice that maximal $P L D\left(22 \mathrm{~kW} / \mathrm{m}^{3}\right)$ inside the arrector pili muscle was $94 \%$ higher than the skin surface whereas peak $E(29 \mathrm{~V} / \mathrm{m})$ was $24 \%$ lower for both polarizations.

For both polarizations shown in Fig. 4, the $E$ in arrector pili muscle and capillaries around the hair follicle attenuated by $48.3 \%(29 \Rightarrow 15 \mathrm{~V} / \mathrm{m})$ and $50 \%(20 \Rightarrow 10 \mathrm{~V} / \mathrm{m})$ over a length of $270 \mu \mathrm{~m}$ and $325 \mu \mathrm{~m}$, respectively. The higher conductivity of blood and muscle was the critical factor that led to this attenuation. The corresponding $P L D$ in the arrector pili muscle ( $22 \mathrm{~kW} / \mathrm{m}^{3}$ to $6 \mathrm{~kW} / \mathrm{m}^{3}$ ) and capillaries supplying the hair follicle $\left(11.5 \mathrm{~kW} / \mathrm{m}^{3}\right.$ to $\left.2.9 \mathrm{~kW} / \mathrm{m}^{3}\right)$ decreased by $72.7 \%$ and $74.8 \%$.

The variation of $E$ with depth $(640 \mu \mathrm{~m})$ in hair $(57 \Rightarrow$ $12 \mathrm{~V} / \mathrm{m}$ ) for both polarizations was $78.9 \%$. The $E$ in the skin $(9.2 \mathrm{~V} / \mathrm{m})$ near the bottom of hair root was $23.3 \%$ lower than that inside the hair. Thus, the low water content hair provides


FIGURE 5. Cutaneous microvasculature: (a), (c) $E$ distribution; (b), (d) PLD distribution.
a natural route for the penetration of 60 GHz signal to deeper more vulnerable regions of skin with less attenuation. The $P L D$ also followed a similar trend i.e., attenuation of $P L D$ in hair $\left(0.5 \Rightarrow 0.02 \mathrm{~kW} / \mathrm{m}^{3}\right)$ was $96 \%$ over a length of $640 \mu \mathrm{~m}$.

## D. CUTANEOUS MICROVASCULATURE

Fig. 5 shows $E$ and $P L D$ in an orthogonal cut through the section of the cutaneous microvasculature located above superficial plexus. The local peak ( $26.9 \mathrm{~kW} / \mathrm{m}^{3}$ ) of $P L D$ inside the leading segment of the capillary loop is the most striking feature of the results shown in Fig. 5(b) and was approximately $30.6 \%$ higher than the surrounding skin. This is explicable by the higher conductivity of blood in comparison to skin as $E$ in top segment of the capillary loop ( $31.4 \mathrm{~V} / \mathrm{m}$ ) was similar to the surrounding skin due to the conservation of tangential component of $E$ across the interface. Note that the peak $P L D$ inside the capillary loop was $95.4 \%$ higher than at the skin surface. Contrarily, the maximal $E(31.4 \mathrm{~V} / \mathrm{m})$ induced in the capillary loop was $18.1 \%$ lower than at the skin surface. The results also showed remarkably high (peak $=30.9 \%$ ) $P L D$ $\left(24.9 \mathrm{~kW} / \mathrm{m}^{3}\right)$ at the vertical boundaries of capillary loop/skin compared to the surrounding skin. This was because the step discontinuity in permittivity led to charge accumulation at the boundary and resulted in strong concentration (peak $=16.9 \%$ ) of $E(36.4 \mathrm{~V} / \mathrm{m})$ in comparison to surrounding skin. The aforementioned abrupt changes in $P L D$ highlight that pericytes and endothelial cells, which line the blood vessels, are exposed to higher PLD compared to surrounding skin cells. Lastly, the average $P L D\left(10.63 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and $E(19.61 \mathrm{~V} / \mathrm{m})$ in the arterial limb was $0.7 \%$ and $0.33 \%$ higher than in venous limb of the capillary suggesting a weak dependence on their diameter.

Similarly, compared to surrounding skin, $P L D$ was predominantly enhanced ( $31.4 \%$ and $31.5 \%$ ) in arteries of superficial ( $9.05 \mathrm{~kW} / \mathrm{m}^{3}$ ) and deep plexus ( $0.56 \mathrm{~kW} / \mathrm{m}^{3}$ ) [Fig. 5(b)]. Note that the difference between $E$ inside the superficial ( $18.19 \mathrm{~V} / \mathrm{m}$ ) and deep ( $4.53 \mathrm{~V} / \mathrm{m}$ ) plexus compared to surrounding skin was less than $0.1 \%$. For most part, $P L D$ and $E$ were uniform in the transverse cross-sections of the blood vessels except directly under the vertical joints that connected blood vessels with each other.

For the polarization shown in Fig. 5(d), it was clear that maximum $P L D$ in the capillary loop ( $18.7 \mathrm{~kW} / \mathrm{m}^{3}$ ) was roughly $5 \%$ lower than surrounding skin. This was because the boundary conditions in this orientation required $|E|$ to alter inversely with the permittivity of the corresponding domains. In other words, peak $E$ inside the capillary loop $(25.51 \mathrm{~V} / \mathrm{m})$ was $19.2 \%$ lower than surrounding skin. Moreover, in contrast to the polarization shown in Fig. 5(b), PLD on the surface of the vertical segments of the capillary loop $\left(12.1 \mathrm{~kW} / \mathrm{m}^{3}\right)$ was $29.6 \%$ lower than the surrounding skin [Fig. (c)]. This was due to formation of electric dipole around the capillary loop with a minimum $E(25.4 \mathrm{~V} / \mathrm{m})$ at the lateral sides $(16.1 \%$ lower than in surrounding skin).

Likewise, due to weak $E$ coupling, the maximal $P L D$ inside the arteries $\left(5.9 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and veins $\left(5.3 \mathrm{~kW} / \mathrm{m}^{3}\right)$ of superficial plexus was diminished by $5.5 \%$ and $5.36 \%$ compared to the surrounding skin [Fig. 5(c, d)]. Similarly, the maximal PLD in arteries $\left(0.37 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and veins $\left(0.3 \mathrm{~kW} / \mathrm{m}^{3}\right)$ of deep plexus was $7.3 \%$ and $6.5 \%$ lower than in surrounding skin. The corresponding reduction of $E$ in superficial ( $19.4 \mathrm{~V} / \mathrm{m}$ ) and deep ( $3.7 \mathrm{~V} / \mathrm{m}$ ) plexus was observed to be $19.4 \%$ and $20.2 \%$ in comparison to surrounding skin.

Lastly, $P L D\left(4.8 \Rightarrow 0.4 \mathrm{~kW} / \mathrm{m}^{3}\right)$ inside arteries connecting superficial plexus to deep dermal plexus exhibited a general decline ( $92 \%$ ) along the propagation path $(0.58 \mathrm{~mm})$ for both polarizations (data omitted for brevity). This was due to a rapid decay $(71 \%)$ of $E(13.3 \Rightarrow 3.8 \mathrm{~V} / \mathrm{m})$ in highly lossy blood. The results were similar for both polarizations because $E$ was directed normal to the lateral surface of blood vessels.

## E. ECCRINE SWEAT GLANDS

Fig. 6 shows that the precise location of the maximal $P L D$ $\left(45.2 \mathrm{~kW} / \mathrm{m}^{3}\right)$ occurred in the portion of acrosyringium, which was parallel to the applied $E$, and was roughly $45 \%$ higher than that in surrounding skin for both polarizations. The local enhancement in power dissipation was due to a higher conductivity of sweat in comparison to surrounding skin. The corresponding peak $E(34.6 \mathrm{~V} / \mathrm{m})$ approached $E(5 \%$ difference) in surrounding skin due to requirements imposed on tangential $E$ to be continuous across the interface. Note that for both polarizations, the maximal $P L D$ in the acrosyringium ( $45.2 \mathrm{~kW} / \mathrm{m}^{3}$ ) was $97.2 \%$ higher than at the skin surface. Contrarily, the peak $E$ induced in acrosyringium ( $34.6 \mathrm{~V} / \mathrm{m}$ ) was $9 \%$ lower than at the skin surface.

Similarly, for the polarization displayed in Fig. 6(d), the maximal $P L D\left(9.3 \mathrm{~kW} / \mathrm{m}^{3}\right)$ inside the spiraling blood vessel was $3.2 \%$ higher than in surrounding skin. Contrarily, for the


FIGURE 6. Eccrine sweat gland: (a), (c) E distribution; (b), (d) PLD distribution.
polarization shown in Fig. 6(b), the peak PLD $\left(8.8 \mathrm{~kW} / \mathrm{m}^{3}\right)$ inside the blood vessel was $2.94 \%$ lower than in surrounding skin. The higher power loss in the former case was because of stronger $E(18.4 \mathrm{~V} / \mathrm{m})$ due to the slanting profile of the blood vessel with respect to the applied $E(21.9 \mathrm{~V} / \mathrm{m})$ [Fig. 6(c)]. Stated differently, the $E(17.9 \mathrm{~V} / \mathrm{m})$ was relatively less strong in Fig 6(b) as the blood vessel presents a less slant profile to the applied $E(21.9 \mathrm{~V} / \mathrm{m})$. Thus, for normal incidence, higher power loss occurs in the blood vessels which transverse the skin at an oblique angle in comparison to vertical vessels.

In the straight duct, there was a progressive reduction of $P L D\left(15 \Rightarrow 0.2 \mathrm{~kW} / \mathrm{m}^{3}\right.$ i.e., $\left.98.7 \%\right)$ and $E(20 \Rightarrow 2.3 \mathrm{~V} / \mathrm{m}$ i.e., $88 \%$ ) over a length of 1 mm for both polarizations due to highly dissipative sweat. Similarly, the $E(18 \Rightarrow 4.4 \mathrm{~V} / \mathrm{m})$ and PLD $\left(9 \Rightarrow 0.5 \mathrm{~kW} / \mathrm{m}^{3}\right)$ inside the blood vessels $(660 \mu \mathrm{~m}$ long) spiraling along the sweat duct also decreased by $75 \%$ and $94 \%$.

It is also worthwhile to mention that at the midpoint of the straight duct $P L D\left(1.73 \mathrm{~kW} / \mathrm{m}^{3}\right)$ was $7 \%$ lower than in surrounding skin for both orientations of $E$. Similarly, $E$ $(6.7 \mathrm{~V} / \mathrm{m})$ was $32.1 \%$ lower than surrounding skin owning to higher attenuation inside the sweat. Contrarily, at the same depth, $P L D\left(1.88 \mathrm{~kW} / \mathrm{m}^{3}\right)$ inside blood vessels was $1 \%$ higher than in surrounding skin. The key factor responsible for this was the higher conductivity of blood vs skin and it led to $16 \%$ lower $E(8.4 \mathrm{~V} / \mathrm{m})$ in blood vessel compared to surrounding skin. Note that $P L D$ and $E$ were uniform in the transverse sections of the straight duct and associated blood vessels.
Lastly, for the polarization case in Fig 6(a, b), the average $P L D\left(0.178 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and $E(2.06 \mathrm{~V} / \mathrm{m})$ inside the secretory coil located at the end of the dermis was $19.5 \%$ and $24.3 \%$ lower than that in its associated blood vessels. A different picture emerged from Fig. 6(c, d) where secretory coil's average $P L D\left(0.247 \mathrm{~kW} / \mathrm{m}^{3}\right)$ was $13.3 \%$ higher than that of blood vessels. This was primarily because the orientation of


FIGURE 7. Meissner corpuscle: (a), (c) E distribution; (b), (d) PLD distribution.
the secretory coils was relatively parallel to applied $E$ in the latter case and led to comparatively stronger coupling. In other words, average $E$ inside the blood vessels ( $2.69 \mathrm{~V} / \mathrm{m}$ ) was $10.4 \%$ higher than that of the secretory coil.

Despite the high conductivity of sweat, the peaks in $P L D$ were also localized in the zones immediately around the portion of duct normal to applied $E$ for both polarizations (data omitted for brevity). For instance, the PLD ( $42 \mathrm{~kW} / \mathrm{m}^{3}$ ) near the interface of the top segment of the sweat duct was $50 \%$ higher than inside the same duct $\left(21 \mathrm{~kW} / \mathrm{m}^{3}\right)$. This is because of the existence of the charge distributions at such interfaces which is necessary to satisfy the charge conservation ( $\sigma_{1} E_{1}^{\perp}-\sigma_{2} E_{2}^{\perp}=j \omega \rho_{s}$ ) and Gauss's law. Consequently, $E(47 \mathrm{~V} / \mathrm{m})$ at the aforementioned interface was also $50 \%$ higher compared to internal $E$ of the duct. Due to dissipation of energy along the boundary of sweat duct, the cells lining the duct would be subjected to a higher intensity mmWave radiation than cells inside them.

## F. MEISSNER CORPUSCLE

Fig. 7(a) depicts that the PLD maxima ( $19.01 \mathrm{~kW} / \mathrm{m}^{3}$ ) inside the frontal hemispherical lumen of Meissner corpuscle was $1.7 \%$ greater than in surrounding skin because of its higher conductivity. Opposite was true for $E(28.3 \mathrm{~V} / \mathrm{m})$ which was $10.1 \%$ lower than in surrounding skin. Due to geometrical symmetry of Meissner corpuscle, the polarization dependence of $P L D$ and $E$ was weak $(0.01 \%)$ [Fig. 7(c, d)]. Note that the maximal $P L D$ inside the lumen of Meissner corpuscle was $93.4 \%$ higher than the skin surface whereas maximal $E$ ( $28.32 \mathrm{~V} / \mathrm{m}$ ) inside it was $25.8 \%$ lower.

Moreover, $P L D\left(19.01 \mathrm{~kW} / \mathrm{m}^{3}\right.$ to $\left.12.7 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and $E$ ( $28 \mathrm{~V} / \mathrm{m}$ to $23 \mathrm{~V} / \mathrm{m}$ ) tapered off by $33 \%$ and $18 \%$ along the propagation route inside the Meissner corpuscle for both


FIGURE 8. Pacinian corpuscle: (a), (c) E distribution; (b), (d) PLD distribution.
polarizations. A closer examination of Fig. 7(d) revealed that the peak $P L D\left(21.2 \mathrm{~kW} / \mathrm{m}^{3}\right)$ inside the axon of Meissner corpuscle was roughly $22.9 \%$ higher compared to its lumen. This was because $E(26.2 \mathrm{~V} / \mathrm{m})$ parallel to the axon interface remained continuous in conformity with the boundary conditions for tangential $E$. However, in spite of the high conductivity of axoplasm, the maximal $P L D\left(16.91 \mathrm{~kW} / \mathrm{m}^{3}\right)$ inside it in Fig. 7(b) was only $0.8 \%$ higher than in surrounding lumen of Meissner corpuscle. This was due to the reduction of induced $E(23.4 \mathrm{~V} / \mathrm{m}$ versus $26.6 \mathrm{~V} / \mathrm{m})$ by $11.9 \%$ inside the axoplasm in accordance with boundary conditions on the normal component of incident $E$. Furthermore, Fig. 7(a, b) show that the highest $E(34.7 \mathrm{~V} / \mathrm{m})$ and $P L D\left(22.6 \mathrm{~kW} / \mathrm{m}^{3}\right)$ around the fringes of the Meissner corpuscle were $12.7 \%$ and $23.8 \%$ higher than unperturbed values in skin. Contrarily, the peak $E$ ( $26.8 \mathrm{~V} / \mathrm{m}$ ) and $P L D\left(13.5 \mathrm{~kW} / \mathrm{m}^{3}\right)$ along the top boundaries of the Meissner corpuscle in Fig. 7(c, d) were $9.7 \%$ and $18.5 \%$ lower compared to surrounding skin because of formation of an electric dipole around it. Finally, in the transverse sections of the Meissner corpuscle, the $E$ and $P L D$ were not uniform due to the presence of the axon.

## G. PACINIAN CORPUSCLE

Fig. 8(a) shows that the highest $P L D\left(0.69 \mathrm{~kW} / \mathrm{m}^{3}\right)$ was localized at the front surface of the Pacinian corpuscle and was roughly $32.5 \%$ higher than the surrounding skin mainly due to significantly higher conductivity of the Pacinian corpuscle. The peak $E(4.53 \mathrm{~V} / \mathrm{m})$ at the frontal surface corresponded closely ( $9.66 \%$ difference) to that in surrounding skin due the continuity of the tangential components of the incident $E$. Alternatively, for the polarization displayed in Fig. 8(d), the peak $P L D\left(0.575 \mathrm{~kW} / \mathrm{m}^{3}\right)$ was only $20.5 \%$ higher than the surrounding skin. The absorption was higher in the former case because incident $E$ was parallel to the longest dimension of the corpuscle, which resulted in stronger $E$. Conversely,
the maximal $E(4.1 \mathrm{~V} / \mathrm{m})$ in Fig. 8(c) was $16.9 \%$ lower than in surrounding skin. Notice that the maximal PLD in the Pacinian corpuscle was $44.1 \%$ and $54.04 \%$ lower than at the skin surface for the polarizations displayed in Fig. 8(b, d), respectively. Similarly, maximal $E$ induced in the Pacinian corpuscle was also $88.2 \%$ and $89.3 \%$ lower than at the skin surface [Fig. 8(a, c)].

Moreover, $P L D$ and $E$ underwent severe attenuation inside the Pacinian corpuscle due to its higher water content. The reduction in $P L D\left(0.06 \mathrm{~kW} / \mathrm{m}^{3}\right.$ [Fig. $\left.8(\mathrm{~b})\right]$ and $0.08 \mathrm{~kW} / \mathrm{m}^{3}$ [Fig. 8(d)]) and $E(1.3 \mathrm{~V} / \mathrm{m}$ [Fig. 8(a)] and $1.6 \mathrm{~V} / \mathrm{m}$ [Fig. 8(c)]) exceeded $88 \%$ and $65 \%$ at its trailing surface for both polarizations. Lastly, the peak $P L D$ and $E$ due the dielectric discontinuity of the Pacinian corpuscle was always along the axis parallel to the incident $E$. For instance, the peak $P L D\left(0.36 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and $E(4.35 \mathrm{~V} / \mathrm{m})$ for the polarization in Fig. 8(a, b) along the major axis of the Pacinian corpuscle was roughly $52.4 \%$ and $31.05 \%$ higher than in surrounding skin. On the contrary, for the orientation of $E$ displayed in Fig. 8(c, d), the peak $P L D\left(0.097 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and $E(2.28 \mathrm{~V} / \mathrm{m})$ was approximately $36.5 \%$ and $20.3 \%$ lower than in surrounding skin.

## IV. CONCLUSION AND DISCUSSION

In this study, high-resolution 3D models of a complete set of human skin sub-structures (blood vessels, nerve fibers, lymphatic vessels, eccrine sweat glands, sebaceous glands, hair, arrector pili muscle, Pacinian corpuscle, and Meissner corpuscle) were developed based on anatomical data. These models were used to investigate local power distribution in human skin appendages at 60 GHz .

The maximum $P L D\left(45.2 \mathrm{~kW} / \mathrm{m}^{3}\right)$ was observed in the portion of the acrosyringium parallel to the incident $E$ and was about $45 \%$ higher than surrounding skin, mainly due to high water content of sweat. The epidermal axons and Pacinian corpuscle were the skin substructures with second ( $37.9 \%$ i.e., $42.5 \mathrm{~kW} / \mathrm{m}^{3}$ ) and third ( $32.5 \%$ i.e., $0.69 \mathrm{~kW} / \mathrm{m}^{3}$ ) highest $P L D$ compared to the surrounding skin whereas the PLD in blood capillary, axon of Meissner corpuscle, lymphatic capillary and arrector pili muscle was $30.6 \%\left(26.9 \mathrm{~kW} / \mathrm{m}^{3}\right), 23.3 \%$ $\left(21.2 \mathrm{~kW} / \mathrm{m}^{3}\right), 20 \%\left(20.2 \mathrm{~kW} / \mathrm{m}^{3}\right)$ and $13.5 \%\left(22.3 \mathrm{~kW} / \mathrm{m}^{3}\right)$ higher than surrounding skin. Note that some reports suggested that the pain-relieving effect of mmWaves could pass through nerve endings of skin [80]. Our results demonstrate relatively high exposure levels in $A \delta$ and C fibers known to be involved in pain detection.

Although, the maximum $E$ inside hair ( $65.7 \mathrm{~V} / \mathrm{m}$ ) and sebaceous gland ( $36.8 \mathrm{~V} / \mathrm{m}$ ) was $45.05 \%$ and $36.96 \%$ higher than in surrounding skin, the corresponding $P L D\left(0.71 \mathrm{~kW} / \mathrm{m}^{3}\right.$ and $3.9 \mathrm{~kW} / \mathrm{m}^{3}$ ) was $97.1 \%$ and $61.8 \%$ lower due to their lower water content. Moreover, the higher $E$ detected inside hair suggests that hair provides a natural route for the penetration of 60 GHz signal to deeper regions of skin.

The heterogeneous skin model also revealed strikingly higher ( $23.8 \%-60.9 \%$ ) local $P L D\left(22.6-61.53 \mathrm{~kW} / \mathrm{m}^{3}\right)$ at skin appendages-skin interfaces compared to the surrounding homogeneous skin due to the dielectric discontinuities. This
suggests that cells lining (epithelial, endothelial, pericytes, etc.) the different skin appendages are exposed to higher electromagnetic power and potentially subject to higher local heating (thermal analysis is out of the scope of this study and constitutes one of its perspectives).

This study is one of the very first attempts towards understanding of microscale power deposition in skin at mmWave frequencies. Generic models of the skin sub-structures based on the electron microscopy images provide an insight into micro-scale absorption mechanisms in cutaneous layers and reveal the potential capability of mmWaves to selectively target specific skin appendages such as nerves or capillaries. Further refinement of such models based on stochastic approach and recent advancements in non-invasive imaging techniques (e.g., optical coherence tomography [18]) would allow to assess interindividual variability. Additionally, the swift progress towards accurately mapping skin water content at microscale and nanoscale level using Raman microspectroscopy [81], [82] and low-loss electron energy-loss spectroscopy [83] would further improve the dielectric dispersive models of skin appendages and help to elucidate specific absorption sites. Lastly, an experimental direct or indirect validation of the reported results could provide further insight into numerical results, the accuracy of which is directly related to the realism of the model physical properties as well as to the precision of computational technique.

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