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NUMERICAL INVESTIGATION OF THE PLASMA FORMATION IN SKIN TISSUE BY NANOSECOND Nd: YAG LASER PULSE

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Abstract. A numerical analysis is performed to investigate the comparative contribution of the mechanisms responsible for electron gain and losses in laser-induced breakdown of the skin and underlying tissues. In this regard we adopted a simple theoretical formulation relying on the numerical solution of a rate equation that describes the growth of the electron density due to the joined effect of multiphoton, cascade and chromophore ionization processes. Here, the rate also includes the effect of electron loss due to diffusion and recombination processes. The analysis considered skin tissue irradiated by a Nd:YAG laser radiation in the $200 - 550$ nm wavelength range with 6 ns pulse duration full-width half-maximum (FWHM).

1. INTRODUCTION

Since its first discovery in the 1980s (Anderson et al. 1981, Nakagawa et al. 1985), laser-induced skin tissue breakdown has been extensively studied due to its potential and promising applications in different fields, such as molecular and cellular biophysics and bioengineering. As the pulsed laser technologies continued to advance, the fundamental knowledge of laser-skin interaction mechanisms has become of vital importance in the development of modern medicine. Parallel to the investigations related to medical progress in the field, laser-induced breakdown (LIB) phenomena has been extensively studied by several researchers to determine theoretically, as well as experimentally, breakdown thresholds and free electrons density distribution of the formed plasma. Although the breakdown of skin tissues induced by nanosecond laser pulses has attracted active attention, complete and detailed modeling of the mechanism involved in this phenomenon is still an open question.

In the present work, a modified model previously developed by Hoseinimotlag et al. 2014 based on the solution of the free electron density rate equation is proposed to numerically investigate laser-driven plasma formation in skin tissue induced by a Gaussian pulse with a full width at half the maximum (FWHM) of 6 ns at wavelengths in the range 200 nm to 550 nm. The model takes into account the generation of free electrons due to the combined effect of multiphoton, cascade and chromophore ionization processes. These processes are opposed by the loss of electrons within the interaction region through diffusion out of the focal volume and recombination. Thus, in this work, the computations focus on studying the separate contribution of each of the gain and loss processes.

2. THEORETICAL MODEL

The skin tissue is a composite structure containing the inclusions of different type and dimension (blood vessels, nerve endings, sweat glands, and hair follicles), which essentially makes it difficult in the understanding of the processes that occur under the interaction of laser with skin. According to Rogov et al. 2014 the main constituent of the skin is water (~70 %) and that is why, in a first approximation, the human skin can be considered as water-like tissue media.

Laser-induced breakdown in water-like tissue media has attracted wide attention for its significance in the fundamental research on laser-matter interaction and as a baseline model for studying ablation of skin tissues (Fanjul-Vélez et al. 2014). The prime mechanism of the optical ablation we are dealing with in the present study is plasma induced ablation, and hence the description of the process requires the consideration of multiphoton ionization and ionization by light absorption. All these effects can be included in the rate equation that describes the free electron density as a function of time (Hoseinimotlag et al. 2014):

$$\frac{d\rho}{dt} = \left.\frac{d\rho}{dt}\right|_{mpi} + \left.\frac{d\rho}{dt}\right|_{ch} + W_{casc}\,\rho - W_{diff}\,\rho - W_{rec}\,\rho^2, \qquad (1)$$

where the first three terms describe the evolution of the electron density generated by the combined effect of multiphoton, chromophore (the contribution through light absorption by chromophores in the skin tissue), and cascade ionization, respectively. The multiphoton rate, $(d\rho/dt)|_{mpi}$, is obtained by Eq. (29) of (Kennedy 1995), while for the cascade rate, W_{casc}, we used Eq. (16) of (Kennedy 1995). The second $(d\rho/dt)|_{ch}$, i.e. the free-electron density due to chromophore ionization is estimated as in (Fang and Hu 2004). As indicated by the minus sign, the loss of high-energy electrons due to diffusion and recombination proceeds at the rates, W_{diff} and W_{rec}. The values of loss rates can be found in (Kennedy 1995).

Assuming a Gaussian profile for the temporal distribution of the laser pulse, a Runge–Kutta method with adaptive step size is used to numerically solve Eq. 1 for the time-varying electron density. As suggested by (Fang and Hu, 2004) in our analysis we assumed that pre-existing free electron density is negligible at the start of the laser pulse, $\rho(0) = 0$.

3. RESULTS AND DISSCUSION

To get a deeper insight into the exact contribution of the physical processes to the electron density growth rate, the free electron density equation (Eq. 1) is numerically solved for two laser wavelengths, particularly 355 nm and 532 nm with a focal spot diameter of 10.6 μm and laser pulse intensity of 0.5×10^{10} W/cm^2. Accordingly, Figs. 1(a) and 1(b) represent the time evolution of electron density, when the laser pulses are centered at $t = 20$ ns. It should be noted that when electron density is greater than a critical value (Vogel et. al 2005): ρ_{cr} [cm^{-3}] $\cong 1.1 \times 10^{21}/\lambda^2[\mu m]$, the plasma become highly reflective, and the incoming laser light leads to growth of the plasma volume rather than the free electron number. Therefore, in our analysis, the critical free-electron density is calculated to be $I \sim 8.7 \times 10^{21}$ cm^{-3} at 355 nm (Fig. 1(a)) and $\sim 3.8 \times 10^{21}$ cm^{-3} at 532 nm (Fig. 1(b)).

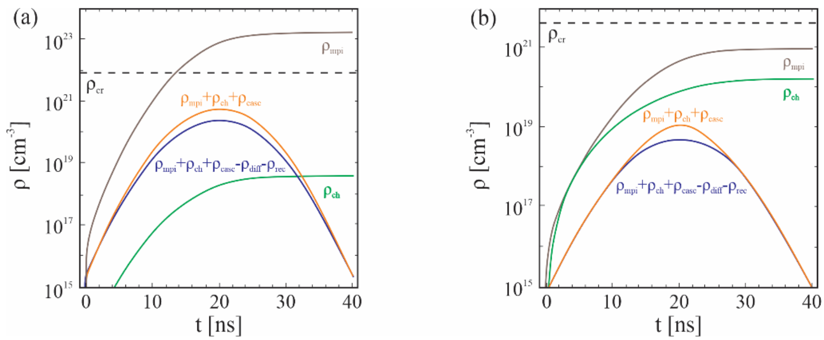

Figure 1: Evolution of the free-electron density for different gain and loss processes at wavelength: (a) 355 nm and (b) 532 nm. The dashed curve is the critical electron density ρ_{cr}.

For skin tissue ablation by nanosecond pulses, the characteristics of the LIB model need to be further elucidated. Acceptance of this model is far from ubiquitous, especially when tissue absorption becomes significant as the light wavelength approaches the ultraviolet region. Given this fact, the effect of the laser wavelength on the free electron density, numerically evaluated via Eq. 1, have been studied. The results are shown in Fig. 2. Calculations were performed for $I \sim 0.35 \times 10^{10}$ W/cm^2 in the wavelength range $200 - 550$ nm. The remaining parameters are the same as those of Fig. 1.

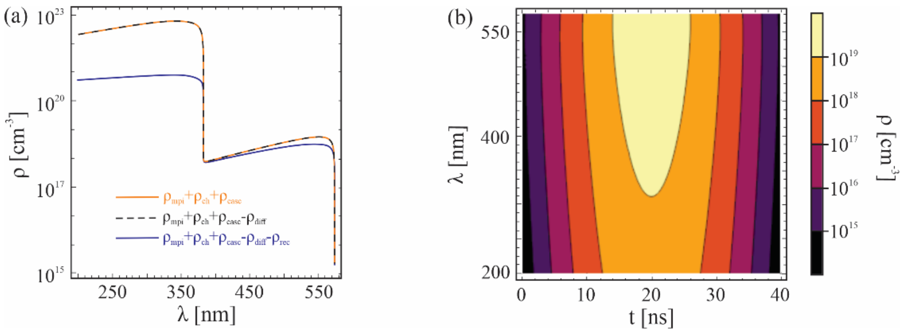

Figure 2: (a) wavelength dependence of electron density for electron creation and loss processes participating in the electron density equation, (b) contour representation of free electron density as a function of time and laser wavelength.

4. CONCLUSION

In summary, we analyzed the time evolution of the free electron density in the absence and presence of each gain and loss process included in the rate equation describing the time evolution of the free electron density in LIB. Beside standard multiphoton and cascade ionization rates, in this paper we incorporated chromophore ionization pathway to explain the skin tissue ablation by nanosecond laser pulses. The presented results verified that in the nanosecond regime, the losses of the free electrons via recombination and diffusion can be neglected (especially when the wavelength exceeds 532 nm) because of the relatively long lifetime and diffusion time of those electrons. Such statement is also confirmed by other researchers (Vogel et. al 2005). We also observed the wavelength dependence of the free electron density and analyzed contribution of each gain and loss mechanisms on this dependence. Our results indicated that shorter wavelengths lead to enhanced electron densities and, hence, the optical breakdown becomes possible at lower laser intensities.

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