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Editorial Open Access

Pharmacogenomics and Pharmacoproteomics Studies of Phosphodiesterase-5 (PDE5) Inhibitors and Paclitaxel Albumin-Stabilized Nanoparticles as Sandwiched Anti-Cancer Nano Drugs between Two DNA/RNA Molecules of Human Cancer Cells

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Editorial

One of the most important goals in the medicine, pharmacology, pharmaceutical, physiological, clinical, biological, medical and medicinal sciences, biochemistry, pharmacogenomics and pharmacoproteomics of anti-cancer Nano drugs such as phosphodiesterase-5 (PDE5) inhibitors and paclitaxel albumin-stabilized nanoparticles is the treatment. The present methods and techniques of Nano-structuring anti-cancer Nano drugs, especially phosphodiesterase-5 (PDE5) inhibitors and paclitaxel albumin-stabilized nanoparticles, are expected to reach their limitations in the next decades. The smallest anti-cancer Nano drugs are of about five nanometers wide and hundreds of millions of them maybe integrated on a single molecule. Below this size the controlled doping becomes more and more difficult. The next important step in the treatment might be done by reducing the anti-cancer Nano drugs such as phosphodiesterase-5 (PDE5) inhibitors and paclitaxel albumin-stabilized nanoparticles to the scale of DNA/RNA molecules of human cancer cells (Figures 1 and 2). This new filed of medicine, pharmacology, pharmaceutical, physiological, clinical, biological, medical and medicinal sciences, biochemistry, pharmacogenomics and pharmacoproteomics is called pharmachemotherapy. Recently, several researchers have measured electron transport in single or small groups of bioorganic molecules such as DNA/RNA of human cancer cells connected to metal such as Cadmium or Ruthenium (Figure 3) [1-29].

Phosphodiesterase-5 (PDE5) inhibitors and paclitaxel albumin-stabilized nanoparticles are two of the molecular systems and anti-cancer Nano drugs that have been studied extensively due to their ability to form a robust Self-Assembled Monolayer (SAM) on DNA/RNA surfaces of human cancer cells (Figure 3) and are useful for synthesizing insulating layers. Recently, tunneling has been identified as the main conduction mechanism for Self–Assembled Monolayers (SAMs) formed in a nanometer scale junction. However, the corresponding biochemical mechanisms governing the electron transport phenomenon in most of this experimental and computational investigation are scarcely mentioned. In this editorial, we report a first-principle study of electron transport in a single molecular conductor consisting of phosphodiesterase-5 (PDE5) inhibitors and paclitaxel albumin-stabilized nanoparticles sandwiched between two DNA/RNA molecules of human cancer cells (Figure 2). We show that the current was increased by increasing the external voltage biases.

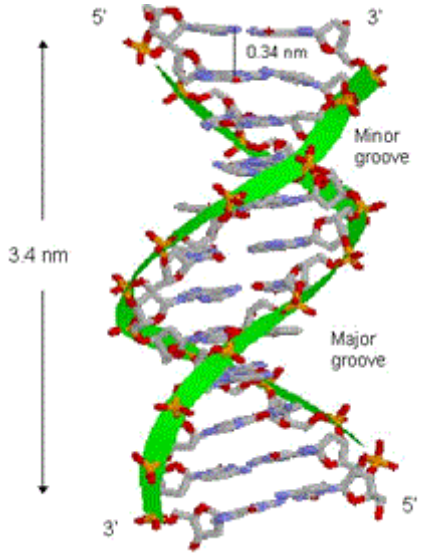

Figure 1: Reducing phosphodiesterase-5 (PDE5) inhibitors to the scale of DNA/RNA molecules of human cancer cells [14,30].

The Projected Density of States (PDOS) and transmission coefficients (T(E)) at various external voltage biases are analyzed and it suggests that the variation of the coupling between the phosphodiesterase-5 (PDE5) inhibitors and paclitaxel albumin-stabilized nanoparticles and the DNA/RNA molecules of human cancer cells with external bias leads to the increase in the current. Therefore, it can be concluded that the most origin of electron transport mechanism in anti-cancer Nano drugs is caused by the characteristics of phosphodiesterase-5 (PDE5) inhibitors and paclitaxel albumin-stabilized nanoparticles and the DNA/RNA molecules of human cancer cells as well as their cooperation, not necessarily only by the inherent properties of certain species of DNA/RNA molecules of human cancer cells themselves.

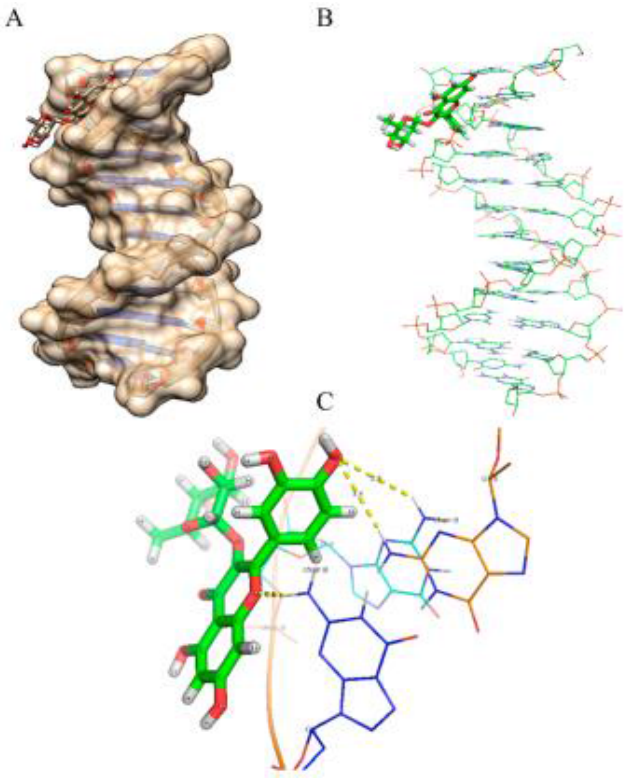

Figure 2: Reducing paclitaxel albumin-stabilized nanoparticles to the scale of DNA/RNA molecules of human cancer cells [14,30].

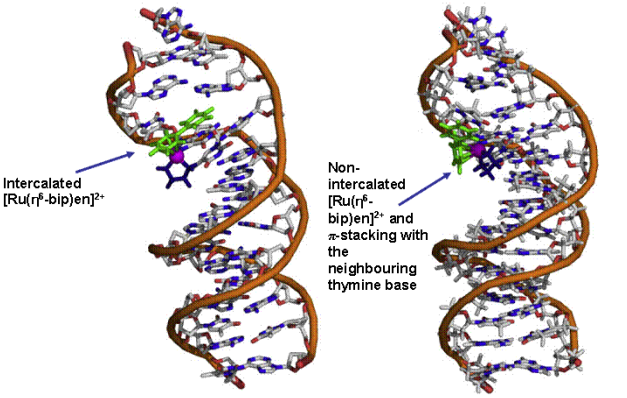

Figure 3: Formation of a robust self–assembled monolayer (SAM) on DNA/RNA surfaces of human cancer cells [14,30].

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