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Comparison of in-office and at-home tooth-whitening products cytotoxicity

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observational planes and making virtual dissections [1, 2].
The aim is demonstrating by means of virtual anatomy how the reticular hypodermic venous system (RHVS) connects the main superficial venous trunks to each other, to the perforating venous system and through them to the deep venous system.
Materials and methods: The scanner used was a 64-detector Phillips multislice tomograph and the program chosen was Phillips' IntelliSpace Portal. To determine the characteristics of RHVS, it was divided the studied population into three groups: 1- without venous pathology, 2- with venous hypertension due to obstruction or compression, 3- in varicose recurrence.
Results: The procedure used allowed to demonstrate that the RHVS produces a true integration of the entire superficial venous system, perforating venous system and through this to the deep venous system as well.
Discussion and conclusions: The MCVT has proven to be a versatile and effective method of observing the entire superficial venous system in detail [3].
The RHVS integrates both truncal venous systems, saphenous vein magna, saphenous vein parva and its main tributaries [4]. It integrates all perforating veins with the reticular venous system. And it links the superficial venous system and the perforator with the deep venous system. This conclude affirming that the entire superficial venous system is a single anatomical unit integrated by the RHVS.

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Comparison of in-office and at-home tooth-whitening products cytotoxicity

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ABSTRACT
Introduction: Bleaching teeth to have a whiter and bright smile is a popular trend and currently one can do it at home, without technical support from dentists. However, whitening products are not innocuous and the European legislation is clear limiting the content in hydrogen peroxide (H_2O_2), present or released, to 6%, still, products containing up to 40% of H_2O_2 are commercially available. In fact, the contact time of live tissues with H_2O_2 agents is usually small, which may restrict side effects, but on the other hand, successively applications as well as at-home careless applications may be harmful. This work aims to verify how whitening products commercialised in Portugal impact on fibroblasts viability.
Materials and methods: We used fibroblasts, the main cells of both pulp and gingivae that contact with whitening gels. Mouse embryo fibroblasts (NIH/3T3) were incubated with serial dilutions of Opalescence PF boost 40%, Opalescent PF 16%, Opalescent PF 10% from Ultradent (USA) and Bbryance 0.095% (France) in culture medium for 1-hour. After that period, the culture medium was removed and cell viability was determined using MTT assay.
Results: Our results showed a huge decrease in fibroblasts viability after exposure to both product types: containing H_2O_2 or carbamide peroxide. To achieve conditions considered non-toxic, i.e. showing a reduction in cell viability $<30\%$ [1], it was necessary to dilute whitening products at least 100- to 62,500-fold, down to 0.0001–0.0004% H_2O_2, as shown in Table 1.
Discussion and conclusions: Although we cannot extrapolate this effect directly to human teeth, because the concentration of H_2O_2 arriving at the pulp depends on diffusion through dentinal tubules, our observations are of great concern in

Table 1. Necessary dilutions to achieved non-toxic conditions (viability >70%).

Utilisation	Product /Whitening agent	Equivalents in H_2O_2 in the product (%)	Product dilution	Predicted [H_2O_2] to achieve 70% viability
In-office	Opalescent PF boost 40%/ hydrogen peroxide	40	1/62,500	0.0001
At-home	Opalescent PF 16% / carbamide peroxide	5.8	1/37,500	0.0001
	Opalescent PF 10% / carbamide peroxide	3.6	>1/1500	0.0004
	BBRYANCE 0.095% / hydrogen peroxide	0.095	1/100	0.0002

particular for gingivae health. Moreover, the whitening products sold for at-home use are as cytotoxic as the in-office product to be applied under the supervision of the dental professional independently of the product type. Since we found that at-home products have similar toxicities, we anticipated that the BBRYANCE gel will induce less severe effects due to his lower H_2O_2 concentration.

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Effect of Vif in doxorubicin treated breast cancer cells

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ABSTRACT

Introduction: Several studies linked DNA cytosine deaminase APOBEC3 to mutational process driving carcinogenesis [1]. However, APOBEC3 expression varies in breast cancer cells [2] and their role in breast cancer treatment remains elusive. The HIV-1 and HIV-2 Vif proteins are APOBEC3 specific inhibitors that recruit the host E3 ubiquitin ligase complex, inducing APOBEC3 ubiquitination and degradation in proteasomes [1]. In this work, our aim is to inhibit APOBEC3 using Vif and determine the sensibility of breast cancer cells to a non-hormonal treatment with doxorubicin.

Materials and methods: Triple negative breast cancer cell line HCC1806 was transduced with lentiviruses containing Vif-1 and Vif-2 genes in fusion with ZsGreen reporter gene producing two different cell lines named as VIF1 and VIF2 cells. Before cells treatment with doxorubicin, the expression of the fluorescent marker and Vif was confirmed by Fluorescent Activated Cell Sorting (FACS-AriaIII) and PCR, respectively. Characterisation of doxorubicin dose-and time-responsive cell viability was performed using MTT assay.

Results: High-titers of Vif-delivering lentiviruses were produced and used to transduce efficiently the HCC1806 cells. More than 99% of sorting population expressed ZsGreen indicating that Vif-1 and Vif-2 genes were integrated in genomic DNA and expressed in VIF1 and VIF2 cell lines. After treatment with doxorubicin for 24 h, all cell lines showed significant decrease of viability when compared with untreated cells, proportional to the concentration of doxorubicin (Figure 1). Comparison between cell viability of HCC1806 (parental) and VIF1 shows no difference in contrast with the behaviour of VIF2 cells that after doxorubicin treatment showed a significant increase in viability.

Discussion and conclusions: The increased viability of doxorubicin treated VIF2 cells correspond to the development of cells resistance to doxorubicin. This resistance of VIF2 cells is probably related to the APOBEC3 inhibition by Vif 2 protein. Our results raise concerns about general use of doxorubicin as breast cancer treatment, especially when APOBEC3 expression is low.

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