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Analysis of the interference of endogenous circadian rhythms on 3'-deoxy- 3'- [18F]Fluorothymidine physiological uptake at the human bone marrow

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Purpose The main purpose of the present study is to determine if the circadian rhythms present in the human bone marrow are likely to influence 3'- deoxy- 3'-[18F] Fluorothymidine (18F-FLT) uptake in the same organ. The 18F-FLT is a Thymidine analogous proliferation agent. The relatively high physiological uptake of this tracer in the bone marrow diminishes the Tumor/Background (T/B) ratio, decreasing the detection accuracy of PET/CT and possibly affecting SUV quantifications. *Methodology* The present study was of level I, descriptive correlational. The methodology was a bibliographic research in the search motors PubMed, Science Direct, among others, in a population of scientific articles having the key words related with the present study. The articles found were analyzed in terms of their global quality and methodologies and exclusion factors, such as publication Impact Factor (IF), were applied to limit the population. *Results* The most relevant finding was the articles describing that the cytosolic enzyme Thymidine Kinase I (TK1) follows a marked circadian variation, with its acrophase (highest concentration) at the bone marrow occurring at 16.00h or 10 HALO (hours after light onsent). TK1 is responsible for intracellular retention of 18F-FLT, similarly to the way hexokinase is responsible for the retention of 3'- deoxy- 3'- [18F] Fluorodeoxyglucose (18F-FDG), and for reduction of mielotoxicity of the oncostatic agents Fluorouridine (FdUdr) and Azido Thymidine (AZT). This circadian variation of TK1 is the rationale behind chronomodulated chemotherapy treatments using the referred oncostatic agents. In these treatments the drug is administered at times of the day when TK1 activity is low, reducing the mielotoxicity and allowing higher doses of these agents to be administered with therefore better survival rates. Using published articles in different fields it was possible to identify the genes and gene mediators responsible for the circadian regulation of TK1. *Conclusions* The

data collected suggests that the physiological uptake of the 18F-FLT in the bone marrow most likely follows a circadian pattern, due to the marked circadian rhythm presented both by TK1 and cell cycle progression of healthy cells. Hence it is possible that differences on the SUV quantifications measured at various times of day do occur. These findings are especially important in cases of treatment assessment scans. The circadian 18F-FLT amplitude of variation at peripheral organs should be quantified in order to aid assessing the error margin of SUV calculations for this tracer.

Keywords: endogenous biological circadian rhythm, chronotherapy, 18F-FLT, Thymidine Kinase I.