INTRODUCTION

Gefitinib is a drug belonging to a class of tyrosine kinase inhibitors (TKIs) which compete with ATP for its binding pocket in mutated or overexpressed EGFR receptors [1]. By inhibiting tyrosine kinase activity, Gefitinib prevents cancer cell proliferation. Previous plasma-based studies have shown evidence of changes in the circulating lipid composition as a result of pharmacological effects of the drug [2]. Gefitinib, N-(3-chloro-4-fluorophenyl)-7-methoxy-3-(4-morpholinopyrimidin-4-yl)-4-amin is a drug approved in 2003 for the treatment of certain breast and non-small cell lung (NSCLC) cancers as well as some other specific cancers under the brand name of Iressa®. Gefitinib acts by interrupting epidermal growth signaling in target cancer cells in the tyrosine kinase domain and is classified as an epidermal growth factor receptor (EGFR) inhibitor. Gefitinib is well absorbed in mammalian systems with good bioavailability, with peak plasma concentrations observed 3.7 h following dosing with a mean oral bioavailability of 60%. Gefitinib undergoes extensive biotransformation in preclinical species and humans (e.g., resulting in a large number of drug metabolites). As a result, Gefitinib is the lipidomic pathway, and has been shown to cause liver damage, the investigation of the lipidomic effect of Gefitinib in the liver was performed [2].

LIPIDOMIC AND DESI IMAGING STUDY OF LIVER LIPOID WITH DOSED WITH A TYROSINE KINASE INHIBITING DRUG

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REFERENCES