3 B E J P H F O N J D T   B O E   3 B E J P Q S P U F P N J D T

Mehdi Djekidel*

Yale University School of Medicine, USA

Recent whole-genome sequencing e orts have expanded on the traditional histologically-driven subtypes to further stratify cases according to the presence/absence of speci c somatic mutations in oncogenes integral to cancer initiation, progression and metastasis. e development of 'targeted therapeutics' intended to speci cally short-circuit regulatory pathways has led to the successful development of e cacious compounds, but only in patients that harbor the targeted mutation. Tissue con rmation is required for targeted therapy prescription. In this era of molecularly based therapeutics, it may be valuable if somatic driver mutations could be correlated to imaging parameters in order to uncover a molecular pro le-imaging signature. Radiogenomics/radioproteomics or radiology imaging based phenotypes–patterns–could be used to predict the molecular subtype of cancers. Functional, morphological and molecular imaging techniques are routinely used to visualize, characterize, and measure biological processes at the cellular, subcellular, and molecular levels in living subjects. Developing a noninvasive means for identifying the molecular subtype of cancer cases that bypasses or compliments the need for tissue acquisition and assay and that can be performed in the clinic at the time of diagnosis would have the potential to be transformational in the accessibility of personalized cancer medicine.

In daily clinical practice tissue con rmation of driver mutations is required for targeted therapy prescription. Consequently, patients with contraindicated, failed or indeterminate biopsies do not receive potentially bene cial targeted therapies. Non-invasive driver mutation correlation methods could:

• Bring tissue characterization from the invasive minimally invasive realm into the clinic and on the other hand add an additional layer of imaging information (besides standard staging) into the portfolio of clinical data necessary to guide therapeutics and prognosis. is will allow us to better stratify patient treatments and ultimately improve survival. Achieving patient speci c personalized medicine by integrating multiple features provided from imaging, pathology and clinical biomarkers; as well as developing complementary synergistic systems between clinical, biochemical, imaging and cellular/subcellular (molecular/gene) biomarkers is essential.

e goal would not be to replace tissue characterization but to complement it in cases where:

• Patients are not candidates for sampling of their tumors because of poor overall performance status, signi cant co-morbidities or the presence of de novo disease or recurrences in anatomic locations that cannot be or are high risk to be accessed–vascular proximity.

• A biopsy sample is not diagnostic and or does not yield su cient viable tumor cells for mutation detection, for e.g. if that biopsy sampled necrotic, brotic or in ammatory regions of the tumor.

• A sampling bias may yield false n@ tnDuN =no~ nuaõ `ðo tonimally

of significantly related factors on CT Cancer 64: 179-186.

25. Djekidel M, Lacy J (2011) Discordant response to therapy assessed by F18 FDG PET in a stage IV gastroesophageal cancer. Can FDG PET detect clone resistance? J Nucl Med 52: 1055

26. Chen N-Q, Prasad M, Delbeke W (2010) Characterization of tumor-induced superior vena cava syndrome with F-18 FDG PET/CT. A case report of an unusual great veins intimal sarcoma. J Nucl Med 51: 1023.

27. Ersahin s6s6s6s6s6s6s6s6s6ss6 u6s6s7W6-116sd by F16l c 6(r)-15(e)-16()-5n5