**Detection of CFTR mutations using a 4-MATTM microarray technology**

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In order to attain higher overall detection rates of CFTR gene mutations, there is a tendency in several countries to move to screening. This implies more typing of multi-ethnic mutations. Most typing technologies are limited in the number of parameters that can be tested. In order to overcome this problem, we are using an innovative microarray technology platform, the 4-MATTM system, that can contain up to 400 probes, for the development of a CFTR genotyping assay.

The 4-MATTM system is an automated multiparameter testing approach that applies in-licensed microarray technology for diagnostic assays. The technology platform is based on the binding of oligonucleotide probes or proteins, that are immobilized on a porous membrane, with labeled test molecules such as DNA amplicons (eg, for genetic testing) or proteins (eg, for antibody detection). The membrane is incorporated in the wells of a disposable unit known as a chip; each chip contains four wells, with each well holding a microarray. When binding occurs between the test sample and a probe or protein, the resulting fluorescence is recorded. All incubation and reading steps are fully automated and are performed by using the thermostatically controlled 4-MATTM Instrument.

The supporting 4-MATTM Master software provides real-time reading, interprets the results and generates a report providing the final typing or reactivity result.

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**Nasal potential difference measurements in Turkish CF patients**

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**Introduction:** Because of the defective cystic fibrosis transmembrane regulator protein (CFTR), abnormal airway epithelial sodium and chloride transport is the characteristic of cystic fibrosis (CF). In vivo measurement of nasal potential difference (NPD) can define this ion transport abnormalities which are characteristic for CF; so it has been advocated as a diagnostic tool for classical and atypical CF patients.

**Aim:** To assess the difference of NPD measurements between CF and non-CF subjects.

**Method:** NPD measurement was applied to 23 CF and 37 non-CF subjects. Basal NPD, maximum NPD with ringer lactate solution (max NPD), response to amiloride (amiloride), chloride free solution (δCL) and isoproterenol (δiso) were measured.

**Results:** Mean values of basal NPD was −37.27 and −24.34; max NPD was −49.37 and −26.72; NPD with amiloride was −28.61 and −18.67; NPD with chloride free solution (OCL) was −29.65 and −27.68; NPD with isoproterenol was −29.44 and −34.63 millivolts (mV) in CF and non-CF groups respectively. Using T-Test, we found significant difference between CF and non-CF subjects on basal NPD, max NPD and measurement with amiloride; but measurement with OCL and isoproterenol were not different. Also, mean values of amiloride was −20.75 and −8.04; δCL was 1.03 and 9.00; δiso was −0.2 and 6.9; δamiloride was 0.83 and 15.96 mV in CF and non-CF groups respectively and there was a significant difference between two groups.

**Conclusion:** NPD measurement is a reliable diagnostic tool for CF which shows CFTR dysfunction easily in vivo.