

**환경독성물질 모니터링을 위한 생체지표 개발 및 응용**  
**Development and application of biomarkers for monitoring of**  
**environmental toxicants in humans**

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Biological monitoring has been performed for occupational and environmental health. Compared to external monitoring, the biological monitoring means measuring internal dose with biospecimens such as urine, blood, etc, in order to assess toxicant-contaminated levels. Thus, development of sensitive exposure biomarkers, e.g. specific metabolites of parental toxicants, is necessary for proper biological monitoring.

On the other hand, the results of biological monitoring are not coincident with those of external monitoring. There are several reasons why the results of external and biological monitoring are different from each other. Among the reasons, the main reason may be individual variations in lifestyle and susceptibility to the toxicants. Therefore, these kinds of individual variations are recently emphasized. Particularly, genetic variations in metabolic enzymes, which are involved in metabolism of toxicants, have been focused.

In a case of polycyclic aromatic hydrocarbons (PAHs) including benzo(a)pyrene a notorious carcinogens, biological monitoring of them has

been performed for several decades. 1-Hydroxypyrene (1-OHP: fig.1), a main metabolite of pyrene, has been routinely used as an exposure biomarker for the biological monitoring of PAHs. However, it reflects food born- as well as air born-PAHs. Thus, we need more sensitive exposure biomarker for air route specific PAHs in order to control air born PAHs. For this purpose, 1-and 2-naphthol, main metabolites of naphthalene, have been developed and applied in my study (ref.1). I also considered susceptibility markers, e.g. genetic polymorphisms in cytochrome P450(CYP) 2E1 and glutathione S-transferases (GST)M1, which were involved in metabolism of PAHs.

Tobacco smoking is a notorious exposure source of PAHs. Cotinine (fig.2), a metabolite of nicotine in tobacco, is a well known exposure biomarker for tobacco smoking. However, smokers who consumed the same volume of tobacco showed different levels of urinary cotinine. I studied susceptibility markers, e.g. genetic polymorphisms in CYP 2A6, and CYP2E1, which were involved in metabolism of nicotine (2).

In addition, endocrine disrupting chemicals(EDCs) have been emphasized for not only occupational health but also public health. Among EDCs, bisphenol A(BPA, fig.3), a substitutional chemical for glass, has been emphasized due to use of huge volume in world wide. I used urinary BPA as exposure biomarker for environmental BPA and monitored its levels in ordinal Koreans who were not occupationally exposed to BPA (3). Using results of biological monitoring, I finally, tried to estimate its environmental exposure level (fig. 4).

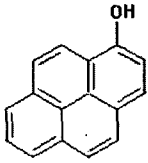


Fig.1 Structure of 1-OHP.

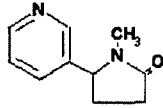


Fig.2 Structure of cotinine

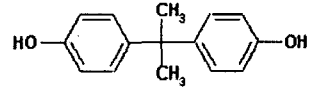


Fig.3 Structure of BPA.

## 일일 노출량 추정

Conjugated BPA in urine (ng/ml) ----- M

0.97 µg/L

Total urinary excretion vol. per day ----- V

0.650 L

Conjugated BPAs excretion dose ----- T<sub>M</sub>

$$T_M = M \times V$$

BPAs exposed dose (ng/kg body weight/day) ----- D

$$D = (T_M \times 1^*) / 30 \text{ kg / day}$$

\*BPA노출량 전부가 거의 뇨의 포함체로 배설된다는 Volkel 등(2002)의 연구결과를 근거

➔ 우리나라 어린이의 **Bisphenol A** 노출량 : **0.021 µg / kg body weight / day** 로 추정

Fig. 4 Estimation of environmental bisphenol A exposure with biological monitoring(4)

### References

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