

# Editorial

## Diet, obesity and cancer

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In the current issue of *J Mol Biochem*, Dachs *et al.* (2015) investigate the links between diet, cancer and obesity by analyzing the effects of high-fat diet on cancer in BALB/c mice, an animal model known to be resistant to diet-induced obesity. The latter is a growing public health problem, whose prevalence has greatly increased and reached epidemic proportions (WHO 2015). Similarly, cancer is one of the main causes of morbidity and mortality worldwide, with over 8 million deaths recorded only during 2012 (IARC 2014) while new cases are expected to dramatically increase over the next few years (WHO 2015b).

In the study published in this issue, Dachs *et al.* (2015) implanted colon adenocarcinoma cells in the flank and monitored tumor growth in mice after being fed a high or normal fat diet. Mice fed the former were found to be 10% heavier compared to the ones fed normal diet. However, such a western style, high fat diet did not have any effect on tumor growth *per se*; i.e. no difference in tumor growth rate or cell proliferation was observed. This is in contrast to two previous studies (Kim *et al.* 2011, Park *et al.* 2012) who have demonstrated an increase in colon and breast cancer growth and metastases in BALB/c mice fed a high fat diet. Even though there are some differences among the study designs, the different outcomes may emphasize the fact that aggravation of tumor growth by increased fat intake may not be universal but other host factors could come into play.

In this regard, Dachs *et al.* (2015) report that within the high-fat diet group, some mice became obese while others remained slim and tumor growth was significantly enhanced within the former subgroup. In other words, tumor growth was aggravated in mice unable to resist obesity. This suggests that high fat diet *per se* does not enhance tumor growth but there are other inherent factors that play a role.

The authors also used protein arrays and the

resulting adipokine profiles generated were shown to be affected by the animals' ability to resist obesity. Most of the adipokines were increased in mice resistant to obesity. But TIMP-1 and CRP were found to be increased in the obese high-fat diet subgroup, suggesting an important role for these factors that needs to be explored further.

### References

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