migration studies clearly show that men with the same genetic background raised in different environments present the risk of the disease associated with their country of residency. Prostate cancer is a good candidate for studies on primary prevention due to several specific features like high prevalence, long latency, hormonal dependency, serum markers for monitoring (prostate specific antigen) and histological precursor lesions (prostatic intraepithelial neoplasia). Nutritional factors that may influence the disease include total energy intake (as reflected by body mass index), dietary fat, cooked meat, micronutrients and vitamins (carotenoids, retinoids, vitamins C, D, and E), fruit and vegetable intake, minerals (calcium, selenium), and phytoestrogens (isoflavonoids, flavonoids, lignans). Most studies reported to date are case-control analysis. The selenium and vitamin E cancer prevention trial (SELECT), however, is a population-based, prospective, randomized clinical trial to examine the effect of selenium and vitamin E alone or in combination on prostate cancer risk reduction. Final results are to be expected in a few years. Until then, lifestyle changes should be recommended to men at risk for developing clinical prostate cancer.

S12 Chemoprevention of prostate cancer – the role of 5 alpha reductase inhibitors

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To date, the Prostate Cancer Prevention trial (PCPT) is the only reported phase III randomized clinical trial to evaluate the role of 5-[alpha] reductase inhibitors in the prevention and treatment of prostate cancer. The original PCPT data revealed that finasteride reduced the risk of prostate cancer by approximately 25% in comparison with placebo. However, patients who received finasteride had a greater incidence of high-grade tumors, which prohibited acceptance of finasteride as a chemopreventive agent by most urologists. Another major concern about the PCPT trial was prevention by finasteride of biologically inconsequential tumors that may otherwise remain undetected during a man's lifetime. Recent updates of the PCPT findings confirmed that finasteride reduces the risk of clinically significant prostate cancer, including high-grade tumors, primarily due to its effects on improving the performance characteristics of prostate-specific antigen and prostate biopsy. There was no increase in high-grade prostate cancer. Rather, there was improved detection of high-grade prostate cancer due to decreased prostate volume. It has been shown that sexual dysfunction with finasteride is not clinically significant and should minimally influence the decision to treat with finasteride. The decision of whether or not to use finasteride in individual cases requires an informed decision that balances the potential benefits of finasteride against the known potential side-effects of prolonged treatment. Patients must weigh the established benefits of a 25–30% reduction in prostate cancer, decreased urinary symptoms, and decreased complications of an enlarged prostate against the established side-effects, which include clinically insignificant reduced sexual function and expense of medication. The results of the REDUCE trial evaluating the role of Dutasteride for chemoprevention of prostate cancer have not been published to date.

S13

PSA screening in early detection of prostate cancer

F.H. Schröder*. Rotterdam, The Netherlands Abstract not available at time of printing.

Keynote Lecture: WHO's Cancer Prevention Policies, Partnerships and Actions

S14

WHO's cancer prevention policies, partnerships and actions

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The known cancer risks such as tobacco, infections, carcinogens which explain more than one third of the global cancer burden are determined by social, cultural and economic factors. There is sufficient knowledge about how to change the level of cancer risk in the population. Examples for effective interventions are vaccination against HBV and HPV, as well as tobacco control as included in the WHO Framework Convention on Tobacco Control, promotion of a healthy lifestyle and avoiding of harmful use of alcohol. There is increasing awareness among WHO Member States to develop national programmes of cancer prevention and control based on evidence based strategies developed by WHO. The medical community including professional organization in cancer play a crucial role in supporting national efforts in implementing prevention strategies. Oncologists in their clinical practice can contribute substantially to cancer prevention by being involved in tobacco cessation, dietary counselling and alcohol control. According to WHO guidance, prevention is part of a comprehensive approach to control cancer which offers the greatest public health potential and most cost-effective long-term method of cancer control.

Session 5. HRT, Lifestyle and Breast Cancer Prevention

S15

Hormone replacement therapy (HRT) and breast cancer risk

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It is clear that administration of estrogen-progestin (E-P) and estrogen alone (E) HRT is sufficient to stimulate the growth of breast tumours in women since withdrawal of HRT results in reduction of proliferation of primary tumours and withdrawal responses in metastatic tumours. E-P, E and tibolone are associated with increased local and distant relapse when given post-operatively. The only large randomised trial (WHI) of E-P or E versus placebo has produced expected and paradoxical results. E-P increases breast cancer risk as previously shown in many observational studies. Risk is increased, particularly in women previously given HRT and known to be compliant. Conversely E either has no effect or reduces breast cancer incidence consistent with some but not all observational studies. Two observational studies report a decrease or at least no increase in risk when E-P or E are given after oophorectomy in young women with BRCA1/2 mutations. Early oophorectomy increases deaths from cardiovascular disease in particular. Thus, HRT may be indicated in young women but, in view of increased cardiovascular disease and breast cancer risk, may be contraindicated in older women particularly taking E-P. Recently progestin treatment has been shown to expand putative tumour stem cell numbers in in-vitro assays by a paracrine effect and this may be one explanation of the apparent differential effects of E-P and E on the breast and breast tumours.