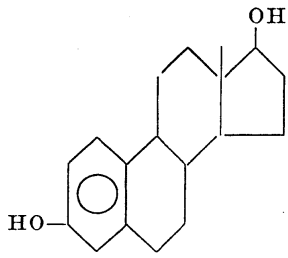
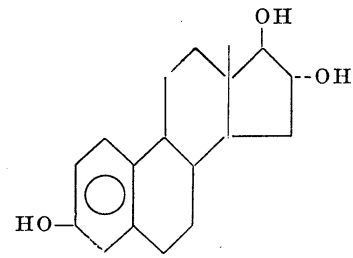
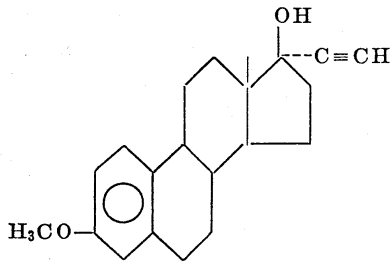
Natural estrogens



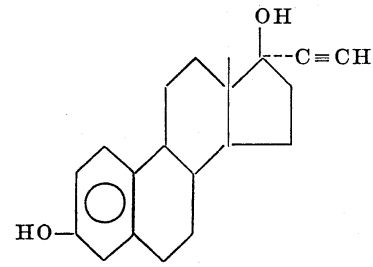


Esthiol

Synthetic estrogens



Mestranol



Ethynylestradiol

In contrast, however, clinical data gleaned from observation and research o thousands upon thousands of women point to no apparent relationship betwee contraceptive therapy and cancer, circulatory system blockages (thromboer bolic diseases), thryoid gland abnormalities, and damage to the ovaries, uteru or Fallopian tubes. This enormous outpouring of safety data led a special FD panel on oral contraceptives last August to declare the pills "not unsafe" fo human use (C&EN, Aug. 15, 1966, page 19). But it didn't close the door on th possibility of such effects on some particularly susceptible women.

Pharmacologists assert that the oral contraceptives occupy a new niche i drug use. Their presence, in many cases for years, affects the sex endocrinol ogy of healthy women during the prime of their reproductive lives. Not onl do they substitute for woman's natural hormonal secretions but, in suppressin ovulation, they also jam the production of her gonadotropic (fertility regulat ing) hormones: follicle stimulating hormone (FSH) and luteinizing hormon (LH), both glycoproteins. Also, they alter carbohydrate metabolism. An chances are that they affect the pattern of cholesterol metabolism, since tha sterol plays such a central and critical role in the synthesis of all body ster oids. In short, the ramifications of oral contraceptive use are immensely wide spread.

The problem with studying the pill is that little enough is known abou normal steroid metabolism per se. Progestin function, for example, seems t differ from dose to dose, from animal to animal, and even from ethnic grou to ethnic group, says Dr. Gabriel Bialy of the Worcester Foundation fo Experimental Biology. Moreover, the effects on target tissue aren't clearl mapped out.

Effects of the synthetic estrogens—mestranol and ethynylestradiol—are a little less fuzzy. These do indeed suppress ovulation, probably via the hypothalamus, by inhibiting production of a protein that triggers secretion of gonadotropin in the pituitary. And they're considerably more powerful than the progestins (see dosages on table).

With this background—the question of subtle dangers and the purely academic fascination—specifically what headway are scientists making toward unraveling this complex biochemistry? The story might well begin at the University of Chicago's Ben May Laboratory for Cancer Research, in the laboratory of Dr. Ellwood V. Jensen.

Organic chemist Jensen was the first to demonstrate the existence of a substance he calls estrogen receptor in the cells of certain target tissue. He remembers his surprise at the swiftness and intensity with which the receptor