DERIVATIVES OF THE ENDODERMAL GERM LAYER
The gastrointestinal tract is the main organ system derived from the endodermal germ layer.

This germ layer covers the ventral surface of the embryo.

With development the embryonic disc begins to bulge into the amniotic cavity and to fold cephalocaudally and Lateral folds also form and move ventrally to assist in body wall closure.

As a result of cephalocaudal folding, a continuously larger portion of the endodermal germ layer is incorporated into the body of the embryo to form the gut tube.

The tube is divided into three regions:

**FOREGUT**

**MIDGUT**

**HINDGUT**
At its cephalic end, **the foregut** is temporarily bounded by an **ectodermal-endodermal (no mesoderm)** membrane called the **OROPHARYNGEAL membrane**.

This membrane separates the stomadeum, (the primitive oral cavity derived from ectoderm), from the pharynx, (a part of the foregut derived from endoderm).

In the fourth week, the oropharyngeal membrane ruptures, establishing an open connection between **the oral cavity** and the primitive gut.

The hindgut also terminates temporarily at an ectodermal-endodermal membrane, **THE CLOACAL membrane**.

This membrane separates the upper part of the anal canal (derived from endoderm), from the lower part called (the proctoderm) that is formed by an invaginating pit lined by ectoderm.

The membrane breaks down in the seventh week to create the opening for the **anus**.
No mesoderm

Buccopharyngeal membrane

Cloacal membrane

WHY?
As a result of folding from the head, tail, and two lateral body wall folds

The ventral body wall of the embryo is closed except for a small part in the umbilical region where the yolk sac duct and connecting stalk are attached.
ENDODERM GIVES RISE TO:

• The epithelial lining of the respiratory tract
• The parenchyma of the thyroid, parathyroids, liver, and pancreas
• The reticular stroma of the tonsils and thymus
• The epithelial lining of the urinary bladder and urethra
• The epithelial lining of the tympanic cavity and auditory tube
PLACENTA

Two component

Fetal
is derived from the trophoblast and extraembryonic mesoderm

CHORION FRONDOSUM

Maternal
The maternal component is derived from the Uterine endometrium

DECIDUA BASALIS
Decidua

- Decidua: (is the structure that will separate)
- Decidua: (is the endometrium after implantation)

Parts:

- Decidua basalis: under the implantation site
- Decidua capsularis: between the implantation site and the uterine lumen
- Decidua parietalis: remaining endometrium
Chorionic plate: extraembryonic mesoderm + trophoblast
1- Chorion laeve: adjacent to *Decidua capsularis*

2- Chorion frondosum: adjacent to *decidua basalis*
At the eighth day of development

The trophoblast differentiates into two layers:

1. *an inner* layer the **CYTOTROPHOBLAST**
2. *an outer* zone the **SYNCYTIOTROPHOBLAST**
At the trophoblast vacuoles appear in the syncytium.

These vacuoles fuse and form large lacunae, and this phase of trophoblast development is known as the LACUNAR STAGE.
Days 11 and 12

At the trophoblast

- The **syncytiotrophoblast** start to penetrate deeper into the stroma and eroding the **maternal capillaries** known as sinusoids.

- The syncytial lacunae become continuous with the sinusoids, and maternal blood enters the **lacunar system**

Thus establishing the **uteroplacental circulation**
Day 13

Cells of the cytotrophoblast proliferate locally and penetrate into the syncytiotrophoblast, forming cellular columns surrounded by syncytium.

Cellular columns with the syncytial covering are known as PRIMARY VILLI.
By the beginning of the third week, the trophoblast is characterized by primary villi that consist of a cytotrophoblastic core covered by an syncytiotrophoblastic layer.

During further development, mesodermal cells penetrate the core of primary villi and grow toward the decidua. The newly formed structure is known as a secondary villus.

By the end of the third week, mesodermal cells in the core of the villus begin to differentiate into blood cells and small blood vessels forming the villous capillary system. The villus is now known as a tertiary villus or definitive placental villus.
Capillaries in tertiary villi make contact with capillaries developing in the mesoderm of the chorionic plate and in the connecting stalk.

These vessels, in turn, establish contact with the intraembryonic circulatory system, connecting the placenta and the embryo.
Maternal blood is delivered to the placenta by spiral arteries in the uterus.

During the following months, numerous small extensions grow out from existing stem villi and extend as free villi into the surrounding lacunar or intervillous spaces.
Initially, these newly formed free villi are primitive but by the beginning of the fourth month, cytотrophoblastic cells and some connective tissue cells disappear.

**The syncytium and endothelial wall of the blood vessels are the only layers that separate the maternal and fetal circulations.**
The placental membrane, which separates maternal and fetal blood, is initially composed of four layers:
(1) the endothelial lining of fetal vessels
(2) the connective tissue in the villus core
(3) the cytotrophoblastic layer
(4) the syncytium

The placental membrane thins because the endothelial lining of the vessels comes in intimate contact with the syncytial membrane, greatly increasing the rate of exchange.

Some times called the placental barrier, the placental membrane is not a true barrier, as many substances pass through it freely.
Cytotrophoblastic cells in the villi penetrate progressively into the overlying syncytium until they reach the maternal endometrium. Here they establish contact with similar extensions of neighboring villous stems forming a thin outer cytotrophoblast shell.

This shell gradually surrounds the trophoblast entirely and attaches the chorionic sac firmly to the maternal endometrial tissue.

Villi that extend from the chorionic plate to the decidua basalis (decidual plate: the part of the endometrium where the placenta will form) are called Stem or anchoring villi. Those that branch from the sides of stem villi are free (terminal) villi, through which exchange of nutrients and other factors will occur.
By the beginning of the fourth month, the placenta has two components:

1. A fetal portion, formed by the chorion frondosum
2. A maternal portion, formed by the decidua basalis

In the junctional zone, trophoblast and decidual cells intermingle.
By this time, most cytotrophoblast cells have degenerated.

Between the chorionic and decidual plates are the intervillous spaces, which are filled with maternal blood. They are derived from lacunae in the syncytiotrophoblast and are lined with syncytium of fetal origin.

The villous trees grow into the intervillous blood lakes.

During the fourth and fifth months, the decidua forms a number of decidual septa, which project into intervillous spaces but do not reach the chorionic plate.

These septa have a core of maternal tissue, but their surface is covered by a layer of syncytial cells, so that at all times, a syncytial layer separates maternal blood in intervillous lakes from fetal tissue of the villi.

As a result of this septum formation, the placenta is divided into a number of compartments, or Cotyledons.
At full term, the placenta is discoid with a diameter of 15 to 25 cm, is approximately 3 cm thick, and weighs about 500 to 600 g. Approximately 30 minutes after birth of the child, it is expelled from the uterine cavity as the afterbirth.

When the placenta is viewed from the maternal side, 15 to 20 slightly bulging areas, the cotyledons, covered by a thin layer of decidua basalis, are clearly recognizable.

Grooves between the cotyledons are formed by decidual septa. The fetal surface of the placenta is covered entirely by the chorionic plate. A number of large arteries and veins, the chorionic vessels, converge toward the umbilical cord. The chorion, in turn, is covered by the amnion.
FUNCTION OF THE PLACENTA
Main functions of the placenta are

(a) *exchange of metabolic and gaseous* products between maternal and fetal bloodstreams

(b) Transmission of Maternal Antibodies

Immunological competence begins to develop late in the first trimester, by which time the fetus makes all of the components of *complement*. Immunoglobulins consist almost entirely of *maternal* immunoglobulin G (IgG) that begins to be transported from mother to fetus at approximately 14 weeks. In this manner, the fetus gains passive immunity against various infectious diseases. Newborns begin to produce their own IgG, but adult levels are not attained until the age of 3 years.
1-During the first two months of pregnancy, the syncytiotrophoblast produces human chorionic gonadotropin (hCG), which maintains the corpus luteum. This hormone is excreted by the mother in the urine, and in the early stages of gestation, its presence is used as an indicator of pregnancy.

2-Estrogenic hormones, predominantly estriol, until just before the end of pregnancy, when a maximum level is reached. These high levels of estrogens stimulate uterine growth and development of the mammary glands.

3-By the end of the fourth month the placenta produces progesterone in sufficient amounts to maintain pregnancy if the corpus luteum is removed or fails to function properly.

4-Somatotropin (formerly placental lactogen). It is a growth hormone-like substance that gives the fetus priority on maternal blood glucose and makes the mother somewhat diabetogenic. It also promotes breast development for milk production.

Note: all hormones are synthesized in the syncytiotrophoblast.
(d) The Placental as a Barrier
Most maternal hormones do not cross the placenta. The hormones that do cross, such as thyroxine, do so only at a slow rate.

Although the placental barrier is frequently considered to act as a protective mechanism against damaging factors, many viruses, such as rubella, cytomegalovirus, Coxsackie, variola, varicella, measles, and poliomyelitis virus, traverse the placenta without difficulty. Once in the fetus, some viruses cause infections, which may result in cell death and birth defects.
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<td>Cytomegalovirus</td>
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<td>Herpes simplex virus</td>
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<td>Valproic acid</td>
<td>Neural tube defects, heart, craniofacial, and limb anomalies</td>
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</tbody>
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TABLE 7.1 Teratogens Associated With Human Malformations
Trimethadione: Cleft palate, heart defects, urogenital and skeletal abnormalities
Lithium: Heart malformations
Amphetamines: Cleft lip and palate, heart defects
Warfarin: Chondrodysplasia, microcephaly
ACE inhibitors: Growth retardation, fetal death
Cocaine: Growth retardation, microcephaly, behavioral abnormalities, gastroschisis
Alcohol: Fetal alcohol syndrome, short palpebral fissures, maxillary hypoplasia, heart defects, mental retardation
Isotretinoin (vitamin A): Vitamin A embryopathy: small, abnormally shaped ears, mandibular hypoplasia, cleft palate, heart defects
Industrial solvents: Low birth weight, craniofacial and neural tube defects
Organic mercury: Neurological symptoms similar to those of cerebral palsy
Lead: Growth retardation, neurological disorders
Hormones
Androgenic agents (ethisterone, norethisterone): Masculinization of female genitalia: fused labia, clitoral hypertrophy
Diethylstilbestrol (DES): Malformation of the uterus, uterine tubes, and upper vagina; vaginal cancer; malformed testes
Maternal diabetes: Variety of malformations; heart and neural tube defects most common