



# About Testicular Cancer

## Overview and Types

If you have been diagnosed with testicular cancer or are worried about it, you likely have a lot of questions. Learning some basics is a good place to start.

- [What Is Testicular Cancer?](#)

## Research and Statistics

See the latest estimates for new cases of testicular cancer and deaths in the US and what research is currently being done.

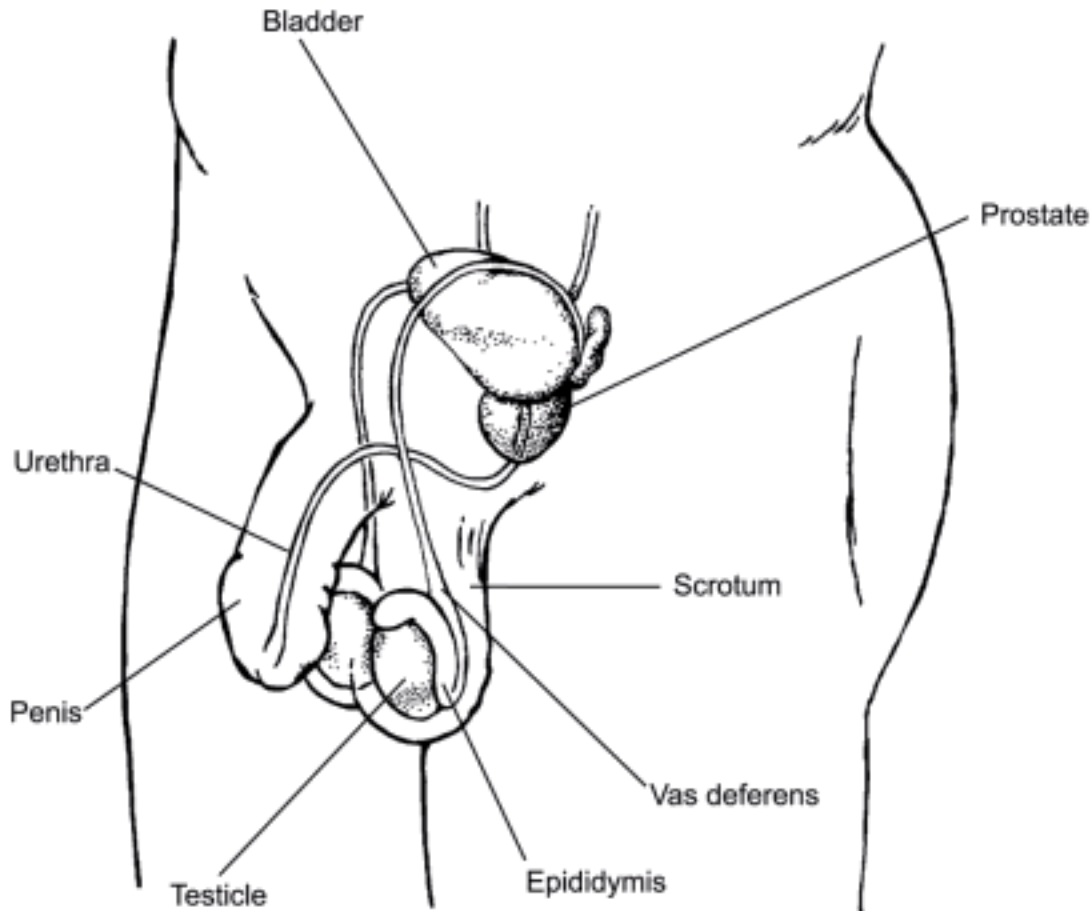
- [Key Statistics for Testicular Cancer](#)
- [What's New in Testicular Cancer Research and Treatment?](#)

# What Is Testicular Cancer?

Cancer starts when cells in the body begin to grow out of control. Cells in nearly any part of the body can become cancer, and can spread to other areas of the body. To learn more about how cancers start and spread, see [What Is Cancer?](#)

Cancer that starts in the testicles is called testicular cancer. To understand this cancer, it helps to know about the normal structure and function of the testicles.

Testicles (also called the *testes*; a single testicle is called a *testis*) are part of the male reproductive system. These 2 organs are each normally a little smaller than a golf ball in adult males and are contained within a sac of skin called the *scrotum*. The scrotum hangs beneath the base of the penis.



Testicles have 2 main functions:

- They make male hormones (androgens) such as testosterone.
- They make sperm, the male cells needed to fertilize a female egg cell to start a pregnancy.

Sperm cells are made in long, thread-like tubes inside the testicles called *seminiferous tubules*. They are then stored in a small coiled tube behind each testicle called the epididymis, where they mature.

During ejaculation, sperm cells are carried from the epididymis through the vas deferens to seminal vesicles, where they mix with fluids made by the vesicles, prostate gland, and other glands to form semen. This fluid then enters the urethra, the tube in the center of the penis through which both urine and semen leave the body.

The testicles are made up of several types of cells, each of which can develop into one or more types of cancer. It is important to distinguish these types of cancers from one

another because they differ in how they are treated and in their prognosis (outlook).

## Germ cell tumors

More than 90% of cancers of the testicle develop in special cells known as *germ cells*. These are the cells that make sperm. The 2 main types of germ cell tumors (GCTs) in men are:

- Seminomas
- Non-seminomas, which are made up of embryonal carcinoma, yolk sac carcinoma, choriocarcinoma, and/or teratoma

Doctors can tell what type of testicular cancer you have by looking at the cells under a microscope.

These 2 types occur about equally. Many testicular cancers contain both seminoma and non-seminoma cells. These *mixed germ cell tumors* are treated as non-seminomas because they grow and spread like non-seminomas.

### Seminomas

Seminomas tend to grow and spread more slowly than non-seminomas. The 2 main subtypes of these tumors are classical (or typical) seminomas and spermatocytic seminomas. Doctors can tell them apart by how they look under the microscope.

**Classical seminoma:** More than 95% of seminomas are classical. These usually occur in men between 25 and 45.

**Spermatocytic seminoma:** This rare type of seminoma tends to occur in older men. The average age of men diagnosed with spermatocytic seminoma is about 65. Spermatocytic tumors tend to grow more slowly and are less likely to spread to other parts of the body than classical seminomas.

Some seminomas can increase blood levels of a protein called *human chorionic gonadotropin* (HCG). HCG can be detected by a simple blood test and is considered a tumor marker for certain types of testicular cancer. It can be used for diagnosis and to check how the patient is responding to treatment.

### Non-seminomas

These types of germ cell tumors usually occur in men between their late teens and early 30s. The 4 main types of non-seminoma tumors are:

- Embryonal carcinoma
- Yolk sac carcinoma
- Choriocarcinoma
- Teratoma

Most tumors are a mix of different types (sometimes with a seminoma component as well), but this doesn't change the general approach to treatment of most non-seminoma cancers.

**Embryonal carcinoma:** This type of non-seminoma is present to some degree in about 40% of testicular tumors, but pure embryonal carcinomas occur only 3% to 4% of the time. When seen under a microscope, these tumors can look like tissues of very early embryos. This type of non-seminoma tends to grow rapidly and spread outside the testicle.

Embryonal carcinoma can increase blood levels of a tumor marker protein called *alpha-fetoprotein* (AFP), as well as *human chorionic gonadotropin* (HCG).

**Yolk sac carcinoma:** These tumors are so named because their cells look like the yolk sac of an early human embryo. Other names for this cancer include yolk sac tumor, endodermal sinus tumor, infantile embryonal carcinoma, or orchidoblastoma.

This is the most common form of testicular cancer in children (especially in infants), but pure yolk sac carcinomas (tumors that do not have other types of non-seminoma cells) are rare in adults. When they occur in children, these tumors usually are treated successfully. But they are of more concern when they occur in adults, especially if they are pure. Yolk sac carcinomas respond very well to chemotherapy, even if they have spread.

This type of tumor almost always increases blood levels of AFP (alpha-fetoprotein).

**Choriocarcinoma:** This is a very rare and aggressive type of testicular cancer in adults. Pure choriocarcinoma is likely to spread rapidly to distant organs of the body, including the lungs, bones, and brain. More often, choriocarcinoma cells are present with other types of non-seminoma cells in a mixed germ cell tumor. These mixed tumors tend to have a somewhat better outlook than pure choriocarcinomas, although the presence of choriocarcinoma is always a worrisome finding.

This type of tumor increases blood levels of HCG (human chorionic gonadotropin).

**Teratoma:** Teratomas are germ cell tumors with areas that, under a microscope, look like each of the 3 layers of a developing embryo: the endoderm (innermost layer), mesoderm (middle layer), and ectoderm (outer layer).

Pure teratomas of the testicles are rare and do not increase AFP (alpha-fetoprotein) or HCG (human chorionic gonadotropin) levels. More often, teratomas are seen as parts of mixed germ cell tumors.

There are 3 main types of teratomas:

- **Mature teratomas** are tumors formed by cells similar to cells of adult tissues. They rarely spread to nearby tissues and distant parts of the body. They can usually be cured with surgery, but some come back (recur) after treatment.
- **Immature teratomas** are less well-developed cancers with cells that look like those of an early embryo. This type is more likely than a mature teratoma to grow into (invade) surrounding tissues, to spread (metastasize) outside the testicle, and to come back (recur) years after treatment.
- **Teratomas with somatic type malignancy** are very rare cancers. These cancers have some areas that look like mature teratomas but have other areas where the cells have become a type of cancer that normally develops outside the testicle (such as a sarcoma, adenocarcinoma, or even leukemia).

## **Carcinoma in situ of the testicle**

Testicular germ cell cancers can begin as a non-invasive form of the disease called *carcinoma in situ* (CIS) or *intratubular germ cell neoplasia*. In testicular CIS, the cells look abnormal under the microscope, but they have not yet spread outside the walls of the seminiferous tubules (where sperm cells are formed). Carcinoma in situ doesn't always progress to invasive cancer.

It is hard to find CIS before it does become an invasive cancer because it generally does not cause symptoms and often does not form a lump that you or the doctor can feel. The only way to diagnose testicular CIS is to have a biopsy (a procedure that removes a tissue sample and looks at it under a microscope). Some cases are found incidentally (by accident) when a testicular biopsy is done for another reason, such as infertility.

Experts don't agree about the best treatment for CIS. Since CIS doesn't always become an invasive cancer, many doctors in the United States consider observation (watchful waiting) to be the best treatment option.

When CIS of the testicle becomes invasive, its cells are no longer just in the seminiferous tubules but have grown into other structures of the testicle. These cancer cells can then spread either to the lymph nodes (small, bean-shaped collections of white blood cells) through lymphatic channels (fluid-filled vessels that connect the lymph nodes), or through the blood to other parts of the body.

## **Stromal tumors**

Tumors can also develop in the supportive and hormone-producing tissues, or stroma, of the testicles. These tumors are known as gonadal stromal tumors. They make up less than 5% of adult testicular tumors but up to 20% of childhood testicular tumors. The 2 main types are Leydig cell tumors and Sertoli cell tumors.

### **Leydig cell tumors**

These tumors develop from the Leydig cells in the testicle that normally make male sex hormones (androgens like testosterone). Leydig cell tumors can develop in both adults and children. These tumors often make androgens (male hormones) but sometimes produce estrogens (female sex hormones).

Most Leydig cell tumors are benign. They usually do not spread beyond the testicle and are cured with surgery. But a small portion of Leydig cell tumors spread to other parts of the body and tend to have a poor outlook because they usually do not respond well to chemotherapy or radiation therapy.

### **Sertoli cell tumors**

These tumors develop from normal Sertoli cells, which support and nourish the sperm-making germ cells. Like the Leydig cell tumors, these tumors are usually benign. But if they spread, they usually don't respond well to chemotherapy and radiation therapy.

## **Secondary testicular cancers**

Cancers that start in another organ and then spread to the testicle are called secondary testicular cancers. These are not true testicular cancers – they are named and treated based on where they started.

[Lymphoma](#) is the most common secondary testicular cancer. Testicular lymphoma occurs more often than primary testicular tumors in men older than 50. The outlook

depends on the type and stage of lymphoma. The usual treatment is surgical removal, followed by radiation and/or chemotherapy.

In boys with acute [leukemia](#), the leukemia cells can sometimes form a tumor in the testicle. Along with chemotherapy to treat the leukemia, this might require treatment with radiation or surgery to remove the testicle.

Cancers of the prostate, lung, skin (melanoma), kidney, and other organs also can spread to the testicles. The prognosis for these cancers tends to be poor because these cancers have usually spread widely to other organs as well. Treatment depends on the specific type of cancer.

- [References](#)

[See all references for Testicular Cancer](#)

Last Medical Review: January 20, 2015 Last Revised: February 12, 2016

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## Key Statistics for Testicular Cancer

The American Cancer Society's estimates for testicular cancer in the United States for 2018 are:

- About 9,310 new cases of testicular cancer diagnosed
- About 400 deaths from testicular cancer

The incidence rate of testicular cancer has been increasing in the United States and many other countries for several decades. The increase is mostly in seminomas. Experts have not been able to find reasons for this increase. Lately, the rate of increase has slowed.

Testicular cancer is not common; about 1 of every 250 males will develop testicular cancer at some point during their lifetime.

The average age at the time of diagnosis of testicular cancer is about 33. This is largely a disease of young and middle-aged men, but about 6% of cases occur in children and teens, and about 8% occur in men over the age of 55.

Because testicular cancer usually can be treated successfully, a man's lifetime risk of dying from this cancer is very low: about 1 in 5,000. If you would like to know more about survival statistics, see [Testicular cancer survival rates](#).

Visit the American Cancer Society's [Cancer Statistics Center](#) for more key statistics.

- [References](#)

[See all references for Testicular Cancer](#)

American Cancer Society. *Cancer Facts & Figures 2018*. Atlanta, Ga: American Cancer Society; 2018.

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Last Medical Review: January 20, 2015 Last Revised: January 4, 2018

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## What's New in Testicular Cancer Research and Treatment?

Important research into testicular cancer is being done in many university hospitals, medical centers, and other institutions around the world. Each year, scientists find out more about what causes the disease, how to prevent it, and how to improve treatment.

### Genetics

In recent years, researchers have found that inherited variations in certain genes, such



as *KITLG*, *SPRY4*, *DMRT1*, *BAK1*, *TERT*, and *ATF7IP*, appear to increase the risk of testicular cancer. These findings may help identify men at higher risk, but they need to be studied more.

Scientists are also studying changes in the genes of testicular cancer cells to learn more about the causes of this disease. Their hope is that improved understanding will lead to even more effective treatment. Certain gene mutations found in the testicular cancer cells have been linked to resistance to chemotherapy and predict poor outcomes. These findings may help individualize treatment and help find new drugs to treat testicular cancer that can target these gene mutations. A better understanding of the genetic changes will also help doctors decide which patients need further treatment and which can be safely treated with surgery alone.

## Treatment

Clinical trials have refined doctors' approaches to treating these cancers. For example, studies have found factors that help predict which patients have a particularly good prognosis and may not need lymph node surgery or radiation therapy. Studies also have found unfavorable prognostic factors that suggest certain patients may benefit from more intense treatment.

A large amount of work is being done to try to limit the long-term toxicities of treatment while maintaining the high cure rate. Doctors want to be able to predict better whose cancer is more likely to recur and then base the amount of therapy on this, thereby not under- or over-treating anyone. For example, one study reported good results by individualizing treatment in men with metastatic cancer based on the decline of tumor marker (AFP and HCG) levels after chemo, giving more intense treatment to those with a slower decline.

New drugs and new drug combinations are being tested for patients with recurrent cancer. Chemo combinations are being refined to see if eliminating certain drugs, replacing them with others, or lowering doses can reduce side effects for some men without reducing the effectiveness of treatment. And high-dose chemotherapy followed by a [stem cell transplant](#) is being studied for men who have tumors with a poor prognosis.

- [References](#)

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