About Pancreatic Cancer

Overview and Types

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

- What Is Pancreatic Cancer?

Research and Statistics

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

- Key Statistics for Pancreatic Cancer
- What’s New in Pancreatic Cancer Research?

What Is Pancreatic Cancer?

Pancreatic cancer is a type of cancer that starts in the pancreas. (Cancer starts when cells in the body begin to grow out of control. To learn more about how cancers start and spread, see What Is Cancer?1)

Pancreatic adenocarcinoma is the most common type of pancreatic cancer. Pancreatic neuroendocrine tumors (NETs) are a less common type and are discussed in Pancreatic Neuroendocrine Tumors2.
Where pancreatic cancer starts

The pancreas

The pancreas is an organ that sits behind the stomach. It's shaped a bit like a fish with a wide head, a tapering body, and a narrow, pointed tail. In adults it's about 6 inches (15 centimeters) long but less than 2 inches (5 centimeters) wide.

- The head of the pancreas is on the right side of the abdomen (belly), behind where the stomach meets the duodenum (the first part of the small intestine).
- The body of the pancreas is behind the stomach.
- The tail of the pancreas is on the left side of the abdomen next to the spleen.

The most common type of pancreatic cancer, adenocarcinoma of the pancreas, starts when exocrine cells in the pancreas start to grow out of control. Most of the pancreas is made up of exocrine cells which form the exocrine glands and ducts. The exocrine glands make pancreatic enzymes that are released into the intestines to help you digest foods (especially fats). The enzymes are released into tiny tubes called ducts which
eventually empty into the pancreatic duct. The pancreatic duct merges with the common bile duct (the duct that carries bile from the liver), and empties into the duodenum (the first part of the small intestine) at the ampulla of Vater.

**Endocrine cells** make up a smaller percentage of the cells in the pancreas. These cells make important hormones like insulin and glucagon (which help control blood sugar levels), and release them directly into the blood. Pancreatic neuroendocrine tumors start in the endocrine cells. See [Pancreatic Neuroendocrine Tumor](#) for more about this type.

If you are diagnosed with pancreatic cancer, it’s very important to know if it’s an endocrine cancer (see [Pancreatic Neuroendocrine Tumor](#)) or exocrine cancer (discussed here). They have distinct risk factors and causes, have different signs and symptoms, are diagnosed with different tests, are treated in different ways, and have different outlooks.

### Types of pancreatic cancer

Exocrine cancers are by far the most common type of pancreas cancer. If you are told you have pancreatic cancer, it's most likely an exocrine pancreatic cancer.

**Pancreatic adenocarcinoma:** About 95% of cancers of the exocrine pancreas are adenocarcinomas. These cancers usually start in the ducts of the pancreas. Less often, they develop from the cells that make the pancreatic enzymes, in which case they are called acinar cell carcinomas.

**Less common types of exocrine cancer:** Other, less common exocrine cancers include adenosquamous carcinomas, squamous cell carcinomas, signet ring cell carcinomas, undifferentiated carcinomas, and undifferentiated carcinomas with giant cells.

**Ampullary cancer (carcinoma of the ampulla of Vater):** This cancer starts in the ampulla of Vater, which is where the bile duct and pancreatic duct come together and empty into the small intestine. Ampullary cancers aren’t technically pancreatic cancers, but they are included here because they are treated much the same.

Ampullary cancers often block the bile duct while they’re still small and have not spread far. This blockage causes bile to build up in the body, which leads to yellowing of the skin and eyes (jaundice). Because of this, these cancers are usually found earlier than most pancreatic cancers, and they usually have a better prognosis (outlook).

### Benign and precancerous growths in the pancreas
Some growths in the pancreas are simply benign (not cancer), while others might become cancer over time if left untreated (known as precancers). Because people are getting imaging tests such as CT scans more often than in the past (for a number of reasons), these types of pancreatic growths are now being found more often.

**Serous cystic neoplasms (SCNs)** (also known as serous cystadenomas) are tumors that have sacs (cysts) filled with fluid. SCNs are almost always benign, and most don’t need to be treated unless they grow large or cause symptoms.

**Mucinous cystic neoplasms (MCNs)** (also known as mucinous cystadenomas) are slow-growing tumors that have cysts filled with a jelly-like substance called mucin. These tumors almost always occur in women. While they are not cancer, some of them can progress to cancer over time if not treated, so these tumors are typically removed with surgery.

**Intraductal papillary mucinous neoplasms (IPMNs)** are benign tumors that grow in the pancreatic ducts. Like MCNs, these tumors make mucin, and over time they sometimes become cancer if not treated. Some IPMNs can just be followed closely over time, but some might need to be removed with surgery if they have certain features, such as if they are in the main pancreatic duct.

**Solid pseudopapillary neoplasms (SPNs)** are rare, slow-growing tumors that almost always develop in young women. Even though these tumors tend to grow slowly, they can sometimes spread to other parts of the body, so they are best treated with surgery. The outlook for people with these tumors is usually very good.

**Hyperlinks**


**References**

Key Statistics for Pancreatic Cancer

How common is pancreatic cancer?

The American Cancer Society’s estimates for pancreatic cancer in the United States for 2019 are:

- About 56,770 people (29,940 men and 26,830 women) will be diagnosed with pancreatic cancer.
- About 45,750 people (23,800 men and 21,950 women) will die of pancreatic cancer.

Pancreatic cancer accounts for about 3% of all cancers in the US and about 7% of all cancer deaths.

It is slightly more common in men than in women.

Lifetime risk of pancreatic cancer

The average lifetime risk of pancreatic cancer is about 1 in 64. But each person’s chances of getting this cancer can be affected by certain risk factors.¹

For statistics related to survival, see Pancreatic Cancer Survival Rates by Stage.²

Visit our Cancer Statistics Center³ for more key statistics.

Hyperlinks
3. [https://cancerstatisticscenter.cancer.org](https://cancerstatisticscenter.cancer.org)

References


Lifetime Risk (Percent) of Being Diagnosed with Cancer by Site and Race/Ethnicity: Both Sexes, 18 SEER Areas, 2013-2015 (Table 1.15)


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What’s New in Pancreatic Cancer Research?

Research into the causes, diagnosis, and treatment of pancreatic cancer is under way in many medical centers throughout the world.

Genetics and early detection

Scientists are learning more about some of the gene changes in pancreas cells that cause them to become cancer. *Inherited changes* in genes such as *BRCA2*, *p16*, and
the genes responsible for Lynch syndrome can increase a person’s risk of developing pancreatic cancer.

Researchers are now looking at how these and other genes may be altered in pancreatic cancers that are not inherited. Pancreatic cancer actually develops over many years in a series of steps known as pancreatic intraepithelial neoplasia or PanIN. In the early steps, such as PanIN 1, there are changes in a small number of genes, and the duct cells of the pancreas do not look very abnormal. In later steps such as PanIN 2 and PanIN 3, there are changes in several genes and the duct cells look more abnormal.

Researchers are using this information to develop tests for detecting acquired (not inherited) gene changes in pancreatic pre-cancerous conditions. One of the most common DNA changes in these conditions affects the \textit{KRAS} oncogene, which affects regulation of cell growth. New diagnostic tests are often able to recognize this change in samples of pancreatic juice collected during an \textit{ERCP} (endoscopic retrograde cholangiopancreatography).

For now, imaging tests like endoscopic ultrasound (EUS), ERCP, and genetic tests for changes in certain genes (such as \textit{KRAS}) are options for people with a strong family history of pancreatic cancer. But these tests are not recommended for widespread testing of people at average risk who do not have any symptoms.

Other tests are looking to see if groups of proteins found in the blood might be used to find pancreatic cancer early, when it is likely to be easier to treat. Some early results with this approach have been promising, but more research is needed to confirm its usefulness.

**Treatment**

A lot of research is focused on finding better treatments for pancreatic cancer. Improving surgery and radiation therapy are major goals, as is determining the best combination of treatments for people with certain stages of cancer.

**Surgery** to remove pancreatic cancer (most often a Whipple procedure) is a long and complex operation that can be hard both for the surgeon and the patient. It often requires a long hospital stay, at least in part because of the long incision (cut) made in the belly.
A newer approach now used at some major medical centers is to do the operation laparoscopically. For this approach, the surgeon makes several small incisions in the belly instead of one large one. Long, thin surgical tools and a tiny video camera are then inserted through these cuts to do the operation. One advantage of this surgery is that people often recover from it more quickly. But this is still a difficult operation. Surgeons are looking to see how it compares to the standard operation and which patients might be helped the most by it.

Radiation therapy

Some studies are looking at different ways to give radiation to treat pancreatic cancer. These include intraoperative radiation therapy (in which a single large dose of radiation is given to the area of the cancer in the operating room at the time of surgery) and proton beam radiation (which uses a special type of radiation that might do less damage to nearby normal cells).

Chemotherapy

Many clinical trials are testing new combinations of chemotherapy drugs for pancreatic cancer. Many studies are seeing if combining gemcitabine with other drugs can help people live longer. Other newer chemo drugs are also being tested, as are combinations of chemo drugs with newer types of drugs.

Targeted therapies

Targeted drugs work differently from standard chemo drugs in that they attack only specific targets on cancer cells (or nearby cells). Targeted therapies may prove to be useful along with, or instead of, current treatments. In general, they seem to have different side effects than traditional chemo drugs. Looking for new targets to attack is an active area of cancer research.

Growth factor inhibitors: Many types of cancer cells, including pancreatic cancer cells, have certain proteins on their surface that help them grow. These proteins are called growth factor receptors. One example is epidermal growth factor receptor (EGFR). Several drugs that target EGFR are now being studied. One, known as erlotinib (Tarceva), is already approved for use along with gemcitabine.

Anti-angiogenesis factors: All cancers depend on new blood vessels to nourish their growth. To block the growth of these vessels and thereby starve the tumor, scientists have developed anti-angiogenesis drugs. These are being studied in clinical trials for patients with pancreatic cancer.
Immune therapy

**Immune therapies**\(^{10}\) attempt to boost a person’s immune system or give them ready-made components of an immune system to attack cancer cells. Some studies of these treatments have shown promising results.

**Monoclonal antibodies**: One form of immune therapy uses injections of man-made **monoclonal antibodies**\(^{11}\). These immune system proteins are made to home in on a specific molecule, such as carcinoembryonic antigen (CEA), which is sometimes found on the surface of pancreatic cancer cells. Toxins or radioactive atoms can be attached to these antibodies, which bring them directly to the tumor cells. The hope is that they will destroy cancer cells while leaving normal cells alone. For use in pancreatic cancer, these types of treatments are available only in clinical trials at this time.

**Cancer vaccines**: Several types of **vaccines**\(^{12}\) for boosting the body’s immune response to pancreatic cancer cells are being tested in clinical trials. Unlike vaccines against infections like measles or mumps, these vaccines are designed to help treat, not prevent, pancreatic cancer. One possible advantage of these types of treatments is that they tend to have very limited side effects. At this time, vaccines are available only in clinical trials.

**Drugs that target immune system checkpoints**: The immune system normally keeps itself from attacking other normal cells in the body by using “checkpoints” – proteins on immune cells that need to be activated (or inactivated) to start an immune response. Cancer cells sometimes find ways to use these checkpoints to avoid being attacked by the immune system. Newer drugs that target these checkpoints have shown a lot of promise in treating some types of cancer. Some of these are now being studied for use in pancreatic cancer.

**Individualization of therapy**

Some drugs seem to work better if certain types of mutations can be found in the patient’s tumor. For example, erlotinib may work better in patients whose tumors have a particular change in the *EGFR* gene. This concept is an area of intense study. There might also be some gene alterations that affect how well gemcitabine will work in a particular patient. Identifying markers that can predict how well a drug will work before it is given is an important area of research in many types of cancer.

**Hyperlinks**

4. www.cancer.org/treatment/understanding-your-diagnosis/staging.html

References


Mellby LD, Nyberg AP, Johansen JS, Wingren C, Nordestgaard BG, Bojesen SE, et


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**Written by**


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