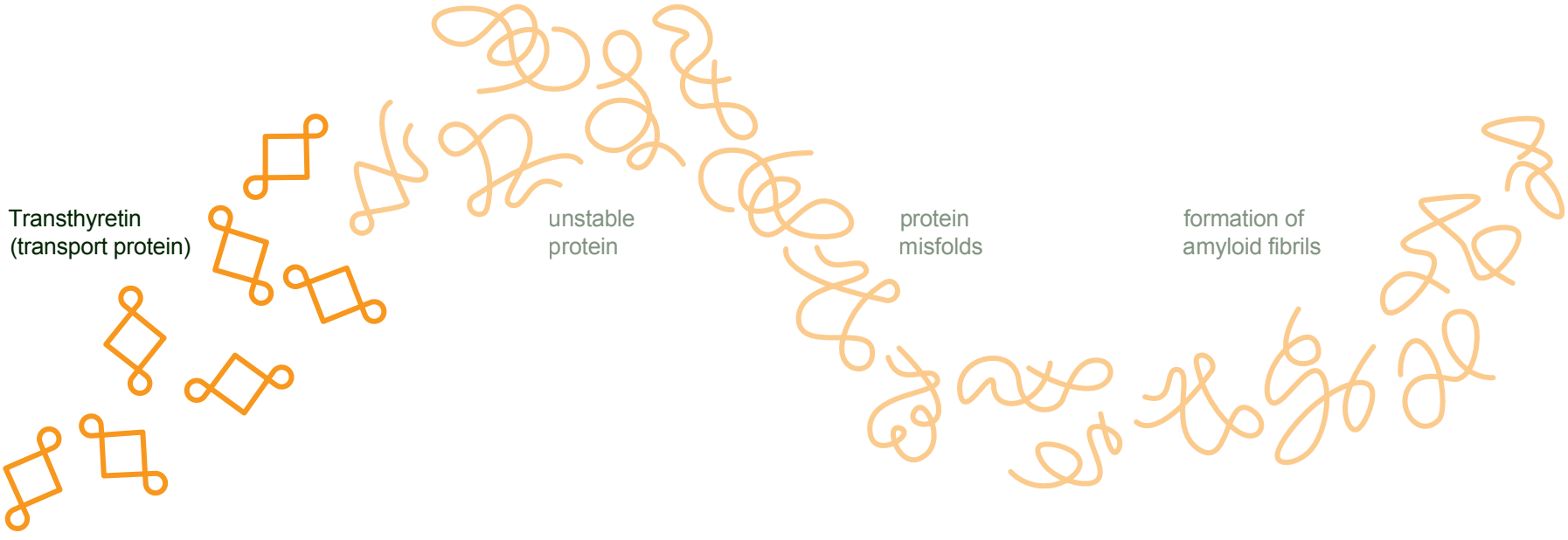
here to learn.

Transthyretin Amyloid Cardiomyopathy (ATTR-CM):
Getting to the Heart of the Matter

The Facts

ATTR-CM is a rare, progressive disease caused when **transthyretin**, a transport protein that naturally circulates in the blood, becomes unstable and misfolds.[1,2]

The misfolded protein can build up in the heart as **amyloid fibrils**, which causes the heart muscle to become stiff, eventually resulting in heart failure.[1,2]



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ATTR-CM can either be hereditary, also known as variant, which is **caused by a mutation** in the transthyretin gene, **or with no mutation and associated with aging,** known as the wild-type form (ATTRwt).[3]

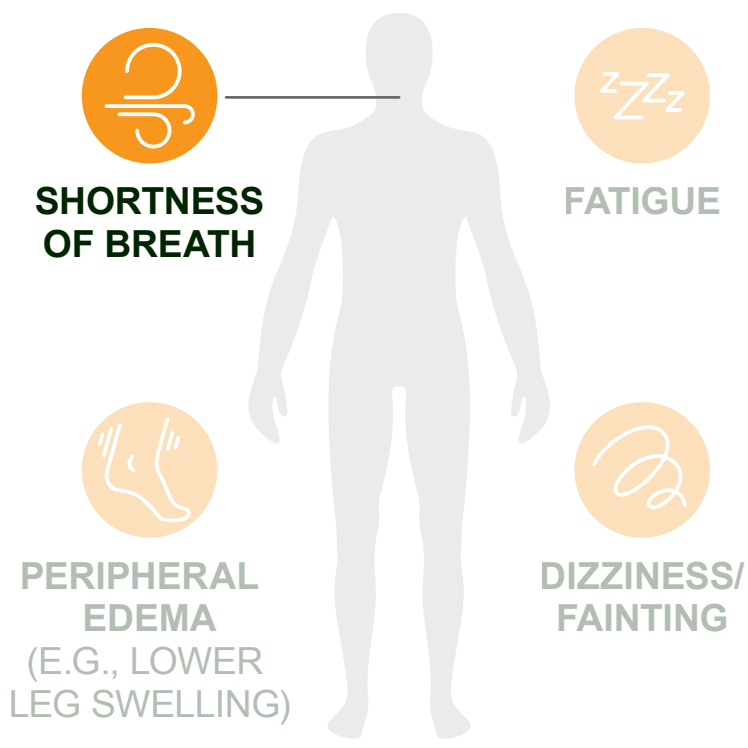
Once diagnosed, the average life expectancy for people with ATTR-CM is approximately 2 to 6 years.[4,5]

A Difficult Road to Diagnosis

It is believed that **less than 1% of people** with ATTR-CM are diagnosed.[6]

Awareness of the disease is low, and symptoms of ATTR-CM are similar to those of heart failure.[7]

Symptoms May Include:[1]



People with ATTR-CM **may not receive a definitive diagnosis**, and if they do, typically remain undiagnosed for many years until the disease has significantly progressed.[8]

How is ATTR-CM diagnosed?

Once ATTR-CM is suspected, based on the signs and symptoms presented, a **number of tests** can be used to help diagnose the disease or identify people at risk.

These include:[9,10]

- **ECHO, ECG, OR CARDIAC MRI**
- **PYP SCINTIGRAPHY** (DIAGNOSTIC TEST TO CONFIRM PRESENCE OF AMYLOID IN THE HEART)
- **GENETIC TEST**
- **CARDIAC BIOPSY**

Treatment Challenges

Currently, there are no approved pharmacologic treatment options for the treatment of ATTR-CM.

Management options include symptom management and in select cases, heart and/or liver transplants.[2]

Education, awareness, and treatment options are critical to improving the diagnosis and care of people affected by ATTR-CM.

1. Ruberg FL, Berk JL. Transthyretin (TTR) cardiac amyloidosis. *Circulation.* 2012;126(10):1286-1300. **2.** Rapezzi C, Quarta CC, Riva L, et al. Transthyretin-related amyloidoses and the heart: a clinical overview. *Nat Rev Cardiol.* 2010;7:398-408. **3.** Siddiqi OK, Ruberg FL. Cardiac amyloidosis: an update on pathophysiology, diagnosis and treatment. *Trends Cardiovasc Med.* 2017;1050-1738. **4.** Ruberg FL, Maurer MS, Judge DP, et al. Prospective evaluation of the morbidity and mortality of wild-type and V122I mutant transthyretin amyloid cardiomyopathy: The Transthyretin Amyloidosis Cardiac Study (TRACS). *Am Heart J.* 2012(164)2:222-228. **5.** Rapezzi C, Merlini G, Quarta CC, et al. Systemic cardiac amyloidosis: disease profiles and clinical courses of the 3 main types. *Circulation.* 2009;120:1203-1212. **6.** 2018 Internal Analysis, Data on File Pfizer Inc. **7.** Rapezzi C, Lorenzini M, Longhi S, et al. Cardiac amyloidosis: the great pretender. *Heart Fail Rev.* 2015;20(2):117-124. **8.** Bhogal S, Ladia V, Sitwala P, et al. Cardiac Amyloidosis: an updated review with emphasis on diagnosis and future directions. *Curr Probl Cardiol.* 2018;43:10-34. **9.** Ando Y, Coelho T, Berk JL, et al. Guideline of transthyretin-related hereditary amyloidosis for clinicians. *Orphanet J Rare Dis.* 2013;8:31. **10.** Gillmore JD, Maurer MS, Falk RH, et al. Non-biopsy diagnosis of cardiac transthyretin amyloidosis. *Circulation.* 2016;133(24):2404-2412.

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