THE ARISING

Strength for the Life Ahead

MUSCLE & STRENGTH



NOVEMBER 2026

Strength for the Life Ahead

Muscle, nourishment, and the work of staying capable

The Arising · Muscle · November 2026

A bag lifted onto a counter. A door held open. The ability to rise, carry, steady and join in. Muscle is part of the machinery of an ordinary day, and ordinary days are where its value becomes visible.

The question is broader than how much muscle a person has. What can they do with it? What is changing? What support would help them preserve or regain the activities they value? A photograph cannot settle those questions. Neither can a single reading from a scale.

This issue brings together four kinds of evidence: a nutrition review, a laboratory study of muscle maintenance, a trial with a real-world mobility outcome, and European experts' shared guidance on sarcopenia, a muscle disorder involving low strength. They illuminate different parts of the same subject. They do not add up to one universal diet, supplement or training prescription.

The research below explores nutrition, strength and cellular maintenance, including why a growth pathway cannot be treated as a simple switch to turn up or down.

This is general education, not an individualized diagnosis or treatment plan. New or unexplained weakness, loss of function or unintended weight loss deserves appropriate clinical assessment rather than an assumption that aging explains everything.

1. Size, strength and performance answer different questions

Muscle quantity asks how much tissue is present. Strength asks about the force a person can produce. Physical performance asks how they complete a task. These ideas are related, but one should not silently substitute for another.

The European expert group known as EWGSOP2 recommends looking first at muscle strength when checking for sarcopenia. Finding too little muscle, or poor muscle quality, helps confirm it. Difficulty with physical tasks helps show how severe it is. It also emphasizes that sarcopenia can occur before old age. This is a clinical framework, not a diagnosis that readers should assign themselves from a mirror or a home device. [4]

The distinction changes how a study should be read. If a treatment increases a measure of lean tissue, ask whether strength or performance was measured too. If a person walks farther, ask what else the program involved. The observation may be worthwhile without proving that muscle growth alone caused the result.

It also changes an everyday conversation. “I have lost muscle” and “I can no longer carry the same groceries” may point toward related concerns, but they are not identical pieces of information. The second statement describes a task and a change that can be explored. The next useful step is to understand the circumstances, not to turn either statement into a diagnosis without assessment.

A small glossary

Sarcopenia: a muscle disorder involving loss of strength and other muscle characteristics, assessed through a clinical framework. Different research definitions can affect which people are included in a study. [4]

Resistance training: activity in which muscles work against resistance. The term does not specify a single load, movement, schedule or level of supervision.

Proteostasis: the balance of making, maintaining and clearing proteins. It is a way of describing cellular upkeep, not a consumer score. [2]

Autophagy: a cellular process involved in breaking down and recycling components. A change in a laboratory marker does not by itself show better function in a person. [2]

2. Nutrition evidence without a magic number

The nutrition review brought together 248 studies: 236 reports of original research and 12 reviews that summarized or combined earlier studies. The original research included 100 randomized trials, in which people were assigned by chance to different treatments. Rather than calculate one overall size of benefit from all 248 studies, the authors looked at the pattern of results and how much confidence to place in them. [1]

One result is especially easy to misread: 84% of the randomized trials testing a single nutrient reported results pointing toward benefit. That counts studies, not people. It is not the percentage of people who benefited, not an 84% improvement in strength, and not a measure of how large the typical benefit was. [1]

A small improvement in one outcome and a larger improvement in another can both contribute to a favorable direction. The count therefore needs to stay attached to its method. The review itself warns against treating these proportions as intervention success rates. That warning belongs beside the attractive number, not buried in a footnote.

The review found the strongest support for getting enough protein alongside strength training, and for correcting low vitamin D in older adults who were deficient. The discussion includes protein amounts around at least 1.0-1.2 grams per kilogram per day. These are research and guideline-context figures for

older adults, not a universal instruction to readers. Needs, restrictions, overall intake and clinical circumstances differ. [1]

The useful conversation is about adequacy and fit: what a person is actually eating, whether the pattern meets their needs, what makes eating difficult, and how nutritional support connects with activity. A supplement label alone does not answer those questions. Correcting a deficiency is also a different proposition from increasing intake in someone who is not deficient.

The authors had moderate confidence in the evidence for Mediterranean-style eating, but much of it came from observing people rather than assigning diets. People’s other habits may help explain the findings. Approaches aimed at changing the gut microbiome, the community of microbes in the digestive tract, had much less reliable evidence. The authors considered these an area for further research, not something ready to prescribe for muscle health. [1]

3. The maintenance workshop inside muscle

Muscle tissue is continually maintained. Proteins are produced, damaged material is cleared, and signaling systems help coordinate the work. The DEAF1 study examines one part of that machinery in flies, mice and cells grown in a laboratory. It does not test a muscle-preserving medicine or an exercise prescription in people. [2]

A useful analogy is a workshop with both construction and maintenance crews. More construction is not always the solution if damaged material accumulates or the work is poorly coordinated. The analogy describes a question about balance; it is not a literal diagram of the cell.

Three names help tell the story. DEAF1 helps control how cells use genetic instructions. mTOR is part of a cell-signaling system called mTORC1 that helps coordinate growth and maintenance. FOXO proteins also help control gene activity.

In these experiments, greater DEAF1 activity increased the production of mTOR and was linked to disrupted protein upkeep. Exercise-related changes involved FOXO activity and reduced activity of the Deaf1 gene. By changing these components experimentally, the researchers could test how they affected one another rather than simply notice that they changed together. These findings concern the studied models, not a proven treatment for people. [2]

This helps explain why the phrase “turn on growth” is too simple. The paper discusses mTORC1's roles in protein production and cellular recycling, and distinguishes brief responses from activity that stays too high. Its findings do not establish that all mTOR activity is harmful or that lowering it continuously would improve a person's health. [2]

An important complication

The authors also discuss apparently differing results elsewhere in the animal literature: their findings support a protective role for FOXO activation in particular aging-muscle conditions, while another study they cite reported better function when FOXO activity was reduced in aged mice. They suggest that age, experimental methods and the extent or duration of the intervention may matter. The DEAF1 authors themselves raise this difference. Their discussion does not settle which explanation is correct. [2]

That caveat is useful. What happens in the body often depends on which tissue is involved, when something happens, the tissue's starting condition and how it is treated. A pathway can be important without having a single direction that is always good. A promising mechanism becomes more informative when the conditions of its success and failure are visible.

Nothing here supports self-directed use of pathway-modifying medicines, gene interventions or research compounds.

4. Keep a human outcome in view

The LIFE trial offers a different kind of evidence. It assigned 1,635 adults aged 70 to 89 by chance to a structured activity program or health education. They were not physically active and had physical limitations, but could still walk 400 metres when the study began. The activity program included aerobic, resistance and flexibility work. [3]

The main question was whether people lost the ability to walk 400 metres. Over an average 2.6 years, this happened to about 30 in 100 people in the activity group and about 36 in 100 in the education group (30.1% and 35.5%). That makes the possible benefit easier to picture: more people retained a useful everyday ability.

A calculation that also accounts for when people lost mobility favored activity: hazard ratio 0.82, where 1 means no difference. Its 95% confidence interval, 0.69 to 0.98, shows the statistical uncertainty around the estimate under the study's assumptions. The range points toward benefit, though its size remains uncertain. The trial tested the whole program, so it cannot tell us that strength training, protein or one cellular process alone caused the difference. [3]

Serious medical problems were reported in about 49 in 100 people in the activity group and 46 in 100 in the education group (49.4% and 45.7%). The difference was uncertain: risk ratio 1.08, with a 95% confidence interval of 0.98 to 1.20. This range includes 1, the value representing no difference, so the result does not clearly establish a difference between groups. The study does not establish that activity caused these problems, but it does not prove the program risk-free either. A supported trial also cannot show that an unsupervised program is suitable for everyone. [3]

Why include this study in a muscle issue? Because it returns the discussion to an ability people recognize. A change inside a cell, a scan of body tissue and a change in walking ability tell us different things. A convincing account of muscle health keeps asking whether the biological changes eventually help people do something that matters to them.

The companion Fitness issue examines LIFE in more detail. Here it supplies an anchor: preserving function is a worthwhile outcome in its own right, and the support built into a program is part of what was tested.

5. What a reader can do with this information

A useful starting point is an observation rather than a target: an activity that has become easier, harder or less reliable. Note what changed, when it changed and what else was happening. The aim is to make a conversation clearer, not to diagnose a muscle disorder at home.

A fictional example: someone notices that carrying a familiar shopping bag has become difficult after an illness. A scale reading alone would not explain the change. A more useful conversation could include recovery, intake, symptoms, medication changes, daily activity and whether a functional assessment would help. This example illustrates questions, not a treatment pathway or a real patient's history.

Another reader is already exercising and wonders whether a new supplement adds value. The evidence-reading questions are practical: was it tested in people like them, what did the comparison group receive, did the study measure strength or just a marker, and what harms or burdens were recorded? A favorable mechanism is a reason to investigate, not an answer to all four questions.

For a clinician, dietitian or rehabilitation professional, possible questions include:

• Which changes in strength or function deserve assessment?

• Are nutritional needs being met, and are there relevant restrictions?

• What kind of activity and support fits the person's present capacity?

• Which measurements would show meaningful progress?

• What would prompt the plan to be reconsidered?

There is no requirement to do everything at once. A clear question can be useful before a complex plan exists. The larger purpose is to connect nourishment, activity, assessment and support to a life the person values.

6. A closer look at the research

The nutrition review brings together human studies. The DEAF1 study examines mechanisms in experimental models. They answer different questions about muscle; one does not prove the other.

Protein and Resistance Training Have the Strongest Support for Preserving Muscle

A large review examined nutrition and muscle health in adults aged 60 and older.

Maintaining muscle is part of maintaining independence. A systematic review in The Journal of Nutrition brings together evidence on nutritional approaches to sarcopenia, the age-related loss of muscle mass, strength and physical performance. Its strongest support goes to familiar foundations: protein-centered nutrition paired with resistance training, and correcting vitamin D deficiency when present.

Dharmansyah and colleagues included 248 studies concerning adults aged 60 and older. The collection included 236 reports of original research and 12 reviews summarizing earlier studies. The original research included 100 trials assigning treatments by chance, alongside studies that observed people and other designs.

A synthesis, not one giant experiment

The authors did not combine all the studies into one estimate of benefit. They examined whether results pointed toward benefit or harm and judged how trustworthy the evidence was. That distinction matters when a large study count appears in a headline.

For example, the review reported a favorable direction of effect in 84% of single-nutrient randomized trials. This is not an 84% chance of success for an individual, and it does not tell us the average size of benefit. Studies can differ in population, outcome and magnitude while pointing in the same direction.

Where the support was strongest

The authors had moderate to high confidence in approaches combining adequate protein intake with strength training. They discuss protein intakes around at least 1.0-1.2 grams per kilogram of body weight per day and approaches enriched with leucine, one of the building blocks of protein. Those are research and guideline-context figures, not a personal prescription; needs and restrictions differ with clinical circumstances.

Correcting low vitamin D in older adults was also strongly supported within the review's framework. That is different from assuming that more vitamin D is better for everyone.

Mediterranean-style dietary patterns came next, with moderate certainty and evidence that was largely observational. Such associations support further study but do not establish that the diet alone prevented muscle loss.

Can changing gut microbes help muscle?

Approaches aimed at the gut microbiome were interesting but much less certain. Results tended to be favorable yet varied substantially, and the authors rated the evidence as low to very low certainty. They described microbiome targeting as a research priority rather than current prescriptive practice.

Studies did not always define sarcopenia the same way or report the same results. That makes confident comparisons harder and limits firm clinical recommendations. A large literature can still leave important questions unresolved.

A useful order of attention

The practical priority is to pay attention to the better-supported foundations while newer ideas are tested. The goal is not only a scan showing more lean tissue, but strength and function that carry into daily life.

Future trials should use clearer, more consistent definitions and measure outcomes people recognize: mobility, capability and independence. The research is most useful when the measurements remain connected to those purposes.

Primary research links appear in the source list below. Study publication: October 21, 2026. [1]

How Exercise Affects Muscle Maintenance in Experimental Models

A study in flies, mice, and cultured cells investigated DEAF1, FOXO, and mTORC1.

Muscle is a place of continuous activity even when a person is resting. Proteins are made, damaged components are cleared, and signaling pathways balance growth with maintenance. A study in laboratory models follows one part of that balance through the DEAF1, FOXO and mTORC1 system described above.

This 2025 study concerns experimental models, not a human exercise trial.

The pathway under study

In the aging-muscle models, DEAF1 helped drive activity in the mTORC1 system. Greater DEAF1 activity led cells to make more of the genetic message used to produce mTOR. It was also linked to disrupted protein upkeep and signs of cellular senescence, lasting changes in cell behavior associated with aging or damage.

In the studied models, exercise increased FOXO activity, which reduced Deaf1 activity. Blocking FOXO or increasing Deaf1 interfered with the exercise-related benefit. The experiments help connect a familiar activity with a specific cellular process.

The work involved flies, mice and cultured cells. It did not determine how much exercise an individual should do, which human training program is optimal or whether a particular person would respond to manipulating the pathway.

What not to conclude

The phrase “reverse muscle aging” in a scientific title can travel much farther than the experiment. Better function in experimental animals does not show that an entire human body has become younger.

Nor does the study establish a birthday after which exercise becomes ineffective. It did not randomize humans to start training at different ages. Turning a mechanistic finding into “it may be too late” advice would answer a question the investigators did not test.

Experiments involving pathway-modifying compounds also do not make those medicines appropriate for self-directed muscle preservation. Clinical benefits, harms, dose and population have to be evaluated separately.

A useful direction for the next experiment

The next question is whether this same system helps explain why people differ in how they maintain muscle and respond to exercise. If it does, researchers could test treatments aimed at it, measuring whether people function better rather than only whether a laboratory marker changes.

The study offers a deeper view of what happens inside muscle during movement. Understanding that process can guide research, but it does not yet tell a person which treatment or exercise plan to choose.

The larger healthspan purpose remains ordinary and important: getting up, climbing a path, carrying something, joining in. Understanding the machinery matters most when it eventually helps preserve those possibilities.

Sources and evidence

Exercise suppresses DEAF1 to normalize mTORC1 activity and reverse muscle aging December 2, 2025. Preclinical flies, mice and cultured cells. [2]

Sources and further reading

[1] Dhika Dharmansyah, Tirta Adikusuma Suparto, Sehabudin Salasa, et al. Protein-Based Strategies and Mediterranean-Style Diets Show the Most Consistent Benefits for Sarcopenia in Older Adults. The Journal of nutrition. 2026 Oct 21:101896. doi:10.1016/j.tjnut.2026.101896. PMID:42906854. Read the primary publication record.

[2] Sze Mun Choy, Kah Yong Goh, Wen Xing Lee, et al. Exercise suppresses DEAF1 to normalize mTORC1 activity and reverse muscle aging. Proceedings of the National Academy of Sciences of the United States of America. 2025 Dec 02;122(48):e2508893122. doi:10.1073/pnas.2508893122. PMID:41284871. Read the primary publication record.

[3] Marco Pahor, Jack M Guralnik, Walter T Ambrosius, et al. Effect of structured physical activity on prevention of major mobility disability in older adults: the LIFE study randomized clinical trial. JAMA. 2014 Jun 18;311(23):2387-96. doi:10.1001/jama.2014.5616. PMID:24866862. Read the primary publication record.

[4] Alfonso J Cruz-Jentoft, Gülistan Bahat, Jürgen Bauer, et al. Sarcopenia: revised European consensus on definition and diagnosis. Age and ageing. 2019 Jan 01;48(1):16-31. doi:10.1093/ageing/afy169. PMID:30312372. Read the primary publication record.

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