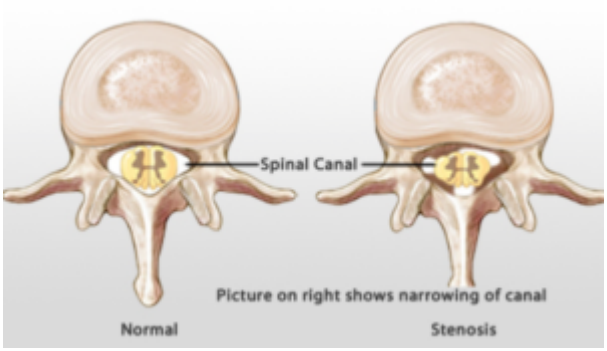
# Lumbar spinal stenosis risk factors




Risk factors for the disease include some comorbidities such as obesity or smoking, daily habits such as an active lifestyle, but also genetic factors that are not completely elucidated yet [1].

Lumbar spinal stenosis (LSS) is frequently observed in obese patients and the elderly especially due to the aging of the spine.

Increased Spinal inclination angle (SIA) and Body Mass Index BMI might be the most relevant risk factors for LSS [2].

DM and low ankle-brachial index values (ABI)s are significantly associated with sLSS in patients with moderate radiographic stenosis. Neither factor is associated with sLSS in patients with severe stenosis. Notably, the effects of intrinsic factors on symptomology may be masked when anatomic stenosis is severe [3].

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Kitab et al., performed a re-analysis of data from their previously reported prospective MRI-based study, stratifying data from the 709 cases into 3 age categories of equal size (instead of the original < 60 vs ≥ 60 years). Relative lumbar spinal canal dimensions, as well as radiological degenerative variables from L1 to S1, were analyzed across age groups in a multivariate mode. The total degenerative scale score (TDSS) for each lumbar segment from L1 to S1 was calculated for each patient. The relationships between age and qualitative stenosis grades, TDSS, disc degeneration, and facet degeneration were analyzed using Pearson's product-moment correlation and multiple regression.

Multivariate analysis of TDSS and spinal canal dimensions revealed highly significant differences across the 3 age groups at L2-3 and L3-4 and a weaker, but still significant, association with changes at L5-S1. Age helped to explain only 9.6% and 12.2% of the variance in TDSS at L1-2 and L2-3, respectively, with a moderate positive correlation, and 7.8%, 1.2%, and 1.9% of the variance in TDSS at L3-4, L4-5, and L5-S1, respectively, with weak positive correlation. Age explained 24%, 26%, and 18.4% of the variance in lumbar intervertebral disc (LID) degeneration at L1-2, L2-3, and L3-4, respectively, while it explained only 6.2% and 7.2% of the variance of LID degeneration at L4-5 and L5-S1, respectively. Age explained only 2.5%, 4.0%, 1.2%, 0.8%, and 0.8% of the variance in facet degeneration at L1-2, L2-3, L3-4, L4-5, and L5-S1, respectively.

Age at presentation correlated weakly with degeneration variables and spinal canal morphometries in LSS segments. Age correlated with upper lumbar segment (L1-4) degeneration more than with lower segment (L4-S1) degeneration. The actual chronological age of the patients did not significantly

correlate with the extent of degenerative pathology of the lumbar spinal stenosis segments. These study results lend support for a developmental contribution to LSS [4].

# References

1)
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