Figure 8: Adult wing phenotypes. A: w\textsuperscript{1118} wing. Longitudinal vein 2 (L2) – blue. Longitudinal vein 3 (L3) – orange. Longitudinal vein 4 (L4) – pink. Longitudinal vein 5 (L5) – red. B: CG11148 wing with L4 gap. C: Df(4)G wing with strong L4 gap. D: Df(4)G wing with L4 gap (pink arrow) and problems with wing hairs (yellow arrow). E: Df(4)38 wing with L4 and L5 gap. F: Wing crumpled by defects in wing formation.

Knowing that the wing vein gap is due to the lack of CG11148, we pursued further research on the gene itself. Previous experiments have shown that a difference in temperature can affect the expression and severity of a gene mutation. Flies were normally stored at room temperature, about 22°C. Flies were placed in new containers at 17°C, room temperature, and 25°C and allowed to mate until larva was present. After the adults were removed so only flies bred in the correct temperature were used for measurements. The flies stored at 22°C and 25°C maintained the normal expression of a wing vein gap with about 70% of all flies observed expressing the mutation. However,
flies kept at 17°C showed no evidence of a wing vein gap. About 10% of the flies had minor planar polarity problems with the wing hairs, but the wings still looked functional. The flies at 17°C have slower development because of the lower temperature, so the longer time seems to allow the wings to develop normally.

**Discussion**

Both CG7224 and CG11148 show clear expression in the mesoderm of the developing embryo. In situ studies on both lines show that in late stages of embryonic formation, there is evidence of each gene in the visceral mesoderm as well as in the somatic mesoderm.

Previous studies implied that CG7224 plays a role in Parkinson’s disease models in *Drosophila*.2,3 The in situ staining showed presence of CG7224 in both the brain and muscle tissues, the two areas of the body Parkinson’s disease affects. Antibody staining with fas III showed breaks in the visceral mesoderm. Visceral mesoderm protects the organs in the body. In fruit flies, it is also important for proper salivary gland migration.9 CG7224 mutants showed defects in the salivary glands, possibly due to the gaps in the visceral mesoderm found in fas III staining. Perhaps using flies that model Parkinson’s, we can stain for CG7224 and see how the embryos develop. We can also use a green fluorescence protein (GFP) marker for fas III or another stain for visceral mesoderm and watch to see the development of mesoderm in the live embryos. This would allow us to visually understand when the problems arise in the mesodermal cells.

The in situ stain also highlighted the developing brain. Further studies on neuronal development and CG7224 can help to clarify if the gene plays a role in neuronal problems found in Parkinson’s. CG7224 was also found in the ring gland of *Drosophila*. The ring gland makes up part of the fruit fly immune system. Research linked CG7224 to the immune system in a study that exposed flies to *Candida albicans*, a fungal pathogen, and discovered an upregulation in the amount of the gene in the presence of the pathogen.10 Further research on CG7224 could be to investigate the role of CG7224 in an immune response to a fly pathogen and if it is different between wild type flies and CG7224 mutants. Investigating these possibilities will help clarify the role of CG7224 in mesoderm development as well as in the brain and ring gland.

From the in situ staining on CG11148, the data shows that there is expression throughout the embryo, especially in the visceral and somatic mesoderm. Fasiclin III staining showed some clumping in the somatic muscles. Fork head staining showed problems with the migration of the salivary glands that may be attributed to the problems with the mesoderm formation.9 We can again use a GFP marker for fas III in CG11148 embryos to see when the muscles are clumping and how the salivary glands are migrating.

The lethality study on CG11148 showed 25-30% death during embryogenesis, which is confirmed by the fork head stain that showed 20% of embryos had deformations in the salivary glands. This backs up the lethality findings and shows that CG11148 mutants are not completely viable.

Other work on the CG11148 line will be to investigate the adult phenotype. It is clear that the gap in the L4 vein is caused somehow by CG11148. The wing veins follow a known pattern. In these flies, the pattern for the correct formation of the wing veins is
not completed. Two pathways that are involved in wing vein patterning are *Notch* and *egrf*. These pathways are found not only in flies but are conserved through evolution and are critical in human embryonic development. While we only visually see a problem with the L4 wing vein, the gap could signal a bigger problem in overall pattern formation of the adult fly. Using a fly lacking *Notch* or *egrf* would help us understand how *CG11148* interacts with the two pathways.

Another approach to finding out how the gap in the wing vein is important in development is to look at the larva. The adult wing in flies is derived from the wing disc, one of many imaginal discs found in the larva that develop into the adult body of the fly. Staining of the imaginal discs with the in situ probe for *CG11148* as well as other probes that are responsible for the development of wing veins will hopefully clarify the importance of *CG11148* in wing vein development.

An interesting study may also be to cross the *CG11148* line and the *CG7224* line to see how the mesodermal tissue would develop with flies lacking both the genes. The overall effort will be to understand how *CG11148* and *CG7224* interact in the fly and the role they play in the formation of the embryo and adult fly.

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