S19). For longer connections there was no clear relationship. Future work should investigate, in greater detail, the underpinnings of the decrease in geometric constraints.

The aim of this study was not to model the growth and development of the human connectome. Doing so would have required a more complicated model that included more system-specific detail. Instead, our models were designed to reduce a network's description length. Naïvely, we can reconstruct a network exactly from a list of its nodes and edges. However, such a precise reconstruction may not be necessary or even desirable. Oftentimes we are more interested in a network's high-level properties (e.g. modularity, degree distribution, etc.), than the exact configuration of its connections. In such a case, a mechanism that generates synthetic networks with the approximately the same set of properties represents a much more economical (compressed) description of the network. Our models are in line with this approach, seeking a parsimonious description of the human connectome, wherein its overt complexity gets compressed into a model's wiring rule and parameters. This type of compressed description can be used toward any number of ends, including investigation of differences in individual participants. For instance, we found that some participants' connectomes were compressible (low energy) while others were not (high energy). An important question, moving forward, is whether these differences become meaningful when examining individual differences or comparing clinical and control populations, or whether they can be related to some behavioral measures across both individual and group levels.

There are a number of methodological considerations that should be discussed. First, the class of dual-term models left the definition of $K(u, v)$ up to the user. For practical reasons, we explored only twelve such rules. Even with this limited exploration, we found a great deal of stratification in terms