Feature	Burgi	Butts	Godfrey	Hennig
Neuron model	Hodgkin-Huxley conductances	Integrate and fire	Abstract	Morris-Lecar conductances
Architecture	One RGC layer	One SAC layer connecting to one RGC layer	One SAC layer	One SAC layer
Initiation mechanism	External simulation of 2–3 boundary RGCs	SACs have fixed probability of firing when non-refractory	A sufficient density of SACs becomes active	A sufficient density of SACs becomes active
Propagation mechanism	RGCs release K^+ which diffuses, increasing excitability of neighbouring RGCs	SACs can spontaneously depolarize and are laterally connected; also connected to RGCs in readout layer	SACs can spontaneously depolarize and are laterally connected	SACs can spontaneously depolarize and are laterally connected
Refractory mechanism	AHP in RGCs	Fixed refractory period	Relative refractory period dependent on the duration of previous activation with identical sAHP	Relative refractory period dependent on the duration of previous activation with identical sAHP
Model output	Membrane voltage of all neurons	Position and times when RGCs are above threshold	Activity levels of SACs	Membrane voltage of all neurons
Comparison to experimental data	Qualitative	Calcium-imaging results published in same paper	Wide-range of calcium imaging data	Quantitative comparisons of MEA recordings presented in same paper

Table 1: Summary of the models of retinal wave generation.