RA Motivations Solutions:

Excerpt #1
Q1.1) retinyl palmitate

Q1.2) central nervous system, face, cardiovascular (ventricular septal defect, aortic arch defect), limbs, urogenital, respiratory, and gastrointestinal

Q1.3) 1953, the vitamin A metabolic pathway is complex, requiring many years of study to decipher its different effects

Q1.4) Any agent that can disturb the development of an embryo or fetus. Teratogens may cause a birth defect in the child. Or a teratogen may halt the pregnancy outright. The classes of teratogens include radiation, maternal infections, chemicals, and drugs.

Q1.5) The contradiction is that while one study reported the birth defects occur at >40,000 IU, another study stated they occur at >10,000 IU. Both cannot be correct at the same time. It is important to settle this contradiction, because we need to know the true limit of retinoic acid consumption in order to design appropriate therapies.

Excerpt #2
Q2.1) #1 providing mother with vitamin A supplements during pregnancy
#2 providing the newborn and infants with vitamin A supplements

Q2.2) Vitamin A treatment of newborn infants was most effective when given up till 1 month. Range of mortality reduction was between 16-64%. (large range probably due to differences in demographics and locations)

Q2.3) diarrhea, measles, acute respiratory infections (acute meaning “severe in effect, intense”)

Q2.4) Vitamin A effectively mitigates illnesses, but is less helpful with illness prevention. This would further suggest that vitamin A is activated in response to an illness, versus working to continually heighten the immune system.

Q2.5) The authors collected human data from pregnant mothers and their newborns in vitamin A deficient populations of South India. Data included incidence of common illnesses, and the associated survival rate of children with observed illnesses. Advantages: data is in humans and very closely related to the actual problem trying to solve (vitamin A deficient deaths) Disadvantage: the cellular mechanisms of how defects form (and how they can be prevented) can not be determined from this human data. Animals provide a great test system for us to study these cellular and molecular details of vitamin A related defect development.

Excerpt #3
Q3.1) 14 different types – thyroid hormone receptors, vitamin D receptors, peroxisome proliferators-activated receptors (PPAR), rar, rxr

Q3.2) cardiovascular, lung, kidney, skeletal, eye defects

Q3.3) retinaldehyde dehydrogenase (RALDH)

Q3.4) Retinoic acid binds to rxr and rar receptor which are attached to the DNA

Q3.5) The fact that vitamin A deficiency symptoms only appear in multiple KO animals possibly suggests that there exists some redundancy of function between the different vitamin A metabolites.