```
3.11, 3.12, 3.13, 3.20.
   3.11. a. For the model \log \mu = \alpha + \beta x, where x = 1 for treatment B and
x=0 for treatment A. Then
   \log \mu_B = \alpha + \beta, and \log \mu_A = \alpha, it is obvious
   \beta = \log \mu_B - \log \mu_A = \log(\mu_B/\mu_A),
   and e^{\beta} = \mu_B/\mu_A.
   b. Fitting the model, we get \log(\hat{\mu}) = 1.6094 + 0.5878x.
   \exp(\hat{\beta}) = \exp(0.5878) = 1.80. The estimated mean number of defects with
treatment B is 80% higher than that with treatment A.
glm(formula = defects ~ trt, family = poisson, data = wafer)
Deviance Residuals:
                1Q
                      Median
                                     3Q
                                               Max
-1.5280 -0.7622 -0.1699
                                 0.6938
                                           1.5399
Coefficients:
              Estimate Std. Error z value Pr(>|z|)
(Intercept)
                1.6094
                             0.1414 11.380 < 2e-16 ***
trtB
                0.5878
                             0.1764
                                      3.332 0.000861 ***
Signif. codes: 0 '*** 0.001 '** 0.01 '* 0.05 '.' 0.1 ' 1
(Dispersion parameter for poisson family taken to be 1)
    Null deviance: 27.857 on 19
                                        degrees of freedom
Residual deviance: 16.268
                               on 18
                                        degrees of freedom
AIC: 94.35
Number of Fisher Scoring iterations: 4
   c. To test H_0: \beta = 0 vs H_a: \beta \neq 0, the Wald test statistic is z = 3.332
with p-value = 0.0009. Reject H_0. The mean number of defects are significantly
different in the two treatment groups.
   d. A 95% CI for log(\mu_B/\mu_A) (witch is just \beta) is:
   0.5878 \pm 1.960.1764 = (0.2421, 0.9335) and
   A 95% CI for (\mu_B/\mu_A) is
   (\exp(0.2421), \exp(0.9335)) = (1.27, 2.54)
   The estimated mean number of defects in Treatment B is between 1.27 to
2.54 times the estimated mean number of defects in Treatment A.
   3.12. Adding coating as a predictor, we get
glm(formula = defects ~ trt + coating, family = poisson, data = wafer)
```

Deviance Residuals:

```
Min 1Q Median 3Q Max
-1.2952 -0.6785 -0.2688 0.6776 1.6307
```

## Coefficients:

Estimate Std. Error z value Pr(>|z|)
(Intercept) 1.7177 0.1602 10.719 < 2e-16 \*\*\*
trtB 0.5878 0.1764 3.332 0.000861 \*\*\*
coating1 -0.2296 0.1701 -1.349 0.177246

---

Signif. codes: 0 '\*\*\* 0.001 '\*\* 0.01 '\* 0.05 '.' 0.1 ' 1

(Dispersion parameter for poisson family taken to be 1)

Null deviance: 27.857 on 19 degrees of freedom Residual deviance: 14.435 on 17 degrees of freedom

AIC: 94.517

Number of Fisher Scoring iterations: 4

The conditional effects of treatment is:

 $\exp(0.5878) = 1.80$ . Controlling for coating, the estimated mean number of defects in B is 80% higher than in A.

 $\exp(-0.2296) = 0.79$ . Controlling for treatment, the estimated mean number of defects is 21% lower with thick coating than with thin coating. But this effect is not significant.

3.13.

a. Change the weight unit to kg (this can be done by dividing the weight column by 1000), and fit the model we get

# Coefficients:

(Dispersion parameter for poisson family taken to be 1)

Null deviance: 632.79 on 172 degrees of freedom Residual deviance: 560.87 on 171 degrees of freedom AIC: 920.16

The fitted equation is  $\log(\hat{\mu}) = -0.4284 + 0.5893x$ 

b.  $\exp(\log(\hat{\mu})) = \exp(-0.4284 + 0.5893 * 2.44) = 2.74.$ 

c.  $\exp(0.5893 \pm 1.96 * 0.065) = \exp(0.4619, 0.7167) = (1.59, 2.05).$ 

The estimated mean number of satellites increase between 59% and 105% for each additional kg increase in weight.

- d. From the output, the z test statistic is 9.064 with p-value close to 0. The effect of weight is significant.
- e. The LR test can be performed by comparing the null deviance and residual deviance. The difference is 632.79 560.87 = 71.92, d.f. = 1,

```
p-value = P(\chi^2 > 71.92) \approx 0.
3.20.
```

	age	personyears	smoking	deaths	deathrate
1	35-44	18793	no	2	0.1064226
2	35-44	52407	yes	32	0.6106055
3	45-54	10673	no	12	1.1243324
4	45-54	43248	yes	104	2.4047355
5	55-64	5710	no	28	4.9036778
6	55-64	28612	yes	206	7.1997763
7	65-74	2585	no	28	10.8317215
8	65-74	12663	yes	186	14.6884624
9	75-84	1462	no	31	21.2038304
10	75-84	5317	ves	102	19.1837502

a. The death rate per 1000 person-years are given in the death rate column above. We can see the death rate increases with age, for both smokers and nonsmokers.

b.

```
glm(formula = deaths ~ age + smoking, family = poisson, data = doctor,
    offset = log(personyears))
```

## Coefficients:

```
Estimate Std. Error z value Pr(>|z|)
(Intercept)
            -7.9194
                         0.1918 -41.298 < 2e-16 ***
age45-54
              1.4840
                         0.1951
                                  7.606 2.82e-14 ***
age55-64
              2.6275
                         0.1837
                                 14.301
                                         < 2e-16 ***
age65-74
              3.3505
                         0.1848
                                 18.131
                                         < 2e-16 ***
age75-84
              3.7001
                                 19.250
                                         < 2e-16 ***
                         0.1922
smokingyes
              0.3545
                         0.1074
                                  3.302 0.00096 ***
```

(Dispersion parameter for poisson family taken to be 1)

```
Null deviance: 935.091 on 9 degrees of freedom
Residual deviance: 12.134 on 4 degrees of freedom
AIC: 79.202
```

b. The main effects only model assumes constant ratio of nonsmokers to smokers death rates over levels of age. It also assumes the age effect is the same

over levels of smoking status. Based on a), the model might not be proper as the effect of smoking becomes less pronounced at older age groups.

c. Based on a, we can see the death rate strictly increases with age, therefore we can treat age as a quantitative variable. This model can be specified as  $\log(\mu/t) = \alpha + \beta_1 age + \beta_2 smoking + \beta_3 (smoking * age)$ .

```
Now for smokers, \log(\mu/t) = \alpha + \beta_2 + (\beta_1 + \beta_3)age,
```

for nonsmokers,  $\log(\mu/t) = \alpha + \beta_1 age$ .

So for either smokers or nonsmokers, the log of the rate changes linearly with age.

d. Assign scores 1, 2, 3, 4, 5 to the age groups, we get

#### Call:

```
glm(formula = deaths ~ age2 * smoking, family = poisson, data = doctor,
    offset = log(personyears))
```

## Deviance Residuals:

```
Min 1Q Median 3Q Max
-3.8784 -2.1219 -0.2482 1.7184 3.5269
```

#### Coefficients:

(Dispersion parameter for poisson family taken to be 1)

```
Null deviance: 935.091 on 9 degrees of freedom
Residual deviance: 59.895 on 6 degrees of freedom
```

AIC: 122.96

Number of Fisher Scoring iterations: 4

By comparing the deviance of the two models, the model in b) seems more appropriate. This model also has a smaller AIC value. The model in d does not fit well perhaps due to the fact the log of rate does not change linearly with age. In fact, adding an  $age^2$  terms can significantly improve the fit of the model.